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ABSTRACT

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

Jakob Madsen
Monash University
900 Dandenong Rd.
Caulfield East
3145 Victoria, Australia
jakob.madsen@monash.edu

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 uncover 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. Instead their regressions reveal that health innovations lead to faster population growth and, therefore, that improved 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 the channel issue 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 show that these effects are quantitatively small. By exploiting the exogenous variation in working age mortality across countries, Lorentzen *at al*. (2008) show that lower working-age mortality leads to higher growth. Using microeconomic estimates for earnings outcomes as a result of improved health, Weil (2007) argues that the contribution to growth from improved health is quite modest. However, a potentially important channel through which health influences 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 during the first half of the 20th century.

This paper argues that health is potentially influential for knowledge and human capital production, the two core drivers of technological progress in endogenous growth models, where health is influenced by disease as well as nutrition as detailed below. The knowledge and human

capital production channels of transmission have not been considered in the empirical literature and cannot be captured by conventional health indicators such as height and life expectancy, because they cannot capture the multi-dimensional aspects of health, the interaction between health and other variables, and, particularly, the time–lag between the health shock and the time at which productivity is affected. The schooling channel illustrates this point. The human capital of the 64-year age cohort, for instance, was partly formed when the cohort started school 58 years earlier. Thus, what is needed is to account for the interaction between health and schooling at the time at which the working age population did their schooling; an effect that cannot be captured by conventional summary measures of health, not even if sufficiently long lags are allowed for. A much more complicated algorithm is required to capture this effect.

Why is health potentially important for learning and knowledge production? A direct effect of health on learning is that malnourished and sick children are often absent from school and are even less likely than healthy children to be enrolled in school (Jamison *et al*. 2006; Ch. 58; Mayer-Foulkus, 2005; Currie *et al*., 2010). More importantly, illness can severely weaken the learning capacity of students due to reduced concentration in the classroom, cognitive impairment, stigma, and coping skills problems (Holding and Snow, 2001; Alderman *et al*., 2005; Tucker, 2006; Mayer-Foulkus, 2005; and Bloom and Canning, 2009). Chronic poor health can adversely affects ideas production because it impairs creativity, entrepreneurship and lateral thinking (Howitt, 2005). 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 leads to xenophobia and ethnocentrism (Schaller and Murray, 2008). As a result, high disease societies tend to value collectivism while penalizing individualism; consequently, being less innovative than individualist societies and tend to encourage rote learning (Fincher *et al*., 2008).

This paper makes three contributions to the literature. First, it incorporates health into learning capacity of school children while allowing for its interaction with schooling. From this measure a health-adjusted measure of educational attainment for the working age population is constructed and used in productivity growth regressions. Real food prices and, in some first-round regressions, infant mortality are used as instruments for age dependent mortality rates. Second,

health among the working age population is incorporated into an ideas production function. Third, whether school enrollment is influenced by health is examined. To enable the analysis a unique annual dataset is constructed for 21 OECD countries over the past two centuries. The long data enables one to trace cohort health effects on schooling that affect productivity with a considerable time-lag for countries that have a long history of collecting reasonable good quality data. Furthermore, the OECD countries went through an epidemiologic and nutritional transition during the late 19th century and the first half of the 20th century; a transition that can only be captured by long historical data.

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). Empirical estimates are undertaken in Section 4 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

The qualitative effects of health on growth through the channels advocated in this paper can be most easily illustrated by a condensed version of the Schumpeterian growth 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 the quantity of 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), depends on 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):

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

$$g = \mu\rho\psi F(k,s)(1-a) \tag{5}$$

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 growth through the parameters ε, ρ, ϕ, λ, and μ. The quantity of schooling, ε, comprises the proportion of an age cohort that is enrolled in school (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 (2000) in which, there is a one-to-one relationship between life expectancy and the optimal number of school 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 to sickness (Jamison *et al*. 2006, Ch. 58; and Mayer-Foulkus, 2005). Furthermore, Chen (1989) finds a strong

positive relationship between absenteeism and infection among pupils in rural China. Finally, Currie *et al*. (2010) find that health at birth, in early childhood and during late teens has a significant effect on educational attainment. Absenteeism has been a potentially large problem for the sample used here, e.g., in the 19th century the attendance rates in Sweden were on average below 50% (Ljungberg and Nelson, 2009). Note that attendance rates are even lower than this in the poorest countries in the world today (United Nations, 2008). Thus, 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 equilibrium research intensity, ρ, is a positive function of health because the pay-off from innovations is higher the more easily the workforce is to adapt to innovations (Aghion and Howitt, 1998, Ch. 6) and because healthy workers are better at dealing with stress and adapting to a new and innovative environment (Howitt, 2005). The skill-adjusted death rate, ϕ, affects growth to the extent that skilled individuals die before reaching retirement age.

Learning efficiency in school, λ, is related to health through concentration in the classroom, absence from school, cognitive processing, and the stigma associated with illness. Students who suffer from chronic or frequent malnutrition and parasitic and infectious diseases tend to have difficulty concentrating and focusing in the classroom, perform poorly in cognitive tests, have low comprehension and judgmental capabilities, have poor fine motor skills, delayed psychomotor development, and have poor scholastic achievements (Grantham-McGregor, 1995; Watkins and Pollitt 1997; Scrimshaw, 1998; Dickson *et al.*, 2000; Holding and Snow, 2001; Alderman *et al*., 2005; Tucker, 2006; Mayer-Foulkus, 2005; and Bloom and Canning, 2009).

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). 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).

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, as discussed in the Introduction, highly infectious environments render societies more collectivist and, consequently, less innovative and less critical to established wisdom. Individualistic cultures, by contrast, value initiative, independent thinking (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 than 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 productivity growth is affected by school enrollments and the 65+ cohort's exit from the labor market. 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. Health is incorporated into human capital as follows.

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*:

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

where h is the health-adjusted human capital among the working age 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 (skill per worker in

manufacturing; see Eq. (4)), 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. Finally, GER^a is, for expositional simplicity, assumed to be independent of health; however, since GERs, as shown in Section 4.5.4, are dependent of health, the health effects on $\dot{h}_t^a$ will be underestimated.

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*:

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

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 the mortality rate is probably the best available proxy for measuring the general health status, it is not a perfect proxy of age-dependent morbidity, which is the variable relevant to learning capacity, fitness and cognitive development. Diseases that can significantly impair learning capacity, such as helminth (all kind of parasitic worms), and iron and iodine deficiency, are not always fatal and only affect age-dependent mortalities indirectly. However, mortality is the only age-dependent morbidity indicator that is available annually over the past two centuries and iron and iodine deficiencies, for example, are often associated with protein-energy malnutrition, which may lead to secondary fatal diseases because protein-energy malnutrition impairs the immune system (Fogel, 1994). Furthermore, age-dependent mortality does capture prevalence of 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. Finally, regressing the sum of age dependent mortality rates in the 0-64 age group on compulsory reported infectious diseases per capita in Italy in the period 1888-2009 yield a correlation coefficient of 0.93 and coefficient of infectious diseases is close to one, indicating a very strong and almost proportional

relationship between morbidity and mortality.[1]

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

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

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

$$h(\Phi)_t = \frac{\sum_{i=0}^{49}\left\{Pop_{15+i}\sum_{j=6}^{22}\left[GER_{t-i+j}exp\left(\Phi ln\left(1-m_{t-i+j}^{j}\right)\right)\right]\right\}}{\sum_{a=15}^{64} pop_t^a} \tag{9}$$

where *h* is the stock of human capital, pop^a is the size of the population at the age cohort *a*, *i* is age cohort *i* starting at the age of 15, *j* is the school grade/level, and 22 is the maximum schooling age.

Eq. (9) 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 since the quality-adjusted GERs are multiplied by the population in each age cohort. From Eq. (9) it follows that 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:

[1] The regression result is as follows:

$$\ln m_t^{0-64} = \underset{(20.6)}{1.04} \ln mb_t, \qquad \text{Estimation period: 1888-2009,}$$

where $m^{0\text{-}64}$ is the average age-dependent mortality in the 0-64 age group, *mb* is morbidity and the number in parenthesis is *t*-statistics. Robust standard errors are used and a constant term is included in the regression but is not reported. Morbidity is measured as the sum of the following compulsory reported infectious diseases per capita: measles, scarlet fever, malaria, brucellosis, viral hepatitis, thyroid fever, meningitis, mumps, whooping coughs, tetanus, and chicken pox. Data sources are reported in the data appendix.

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

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 instruments

Guided by Eqs. (3), (5) and (10) the ideas production and the productivity growth model are stochastically specified as follows:

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

$$\Delta \ln y_{it} = \alpha_0 + \alpha_1 \Delta h(\Phi)_{it} + \alpha_6 \Delta \ln Pat_t + \alpha_7 \Delta \ln S_t^f + \alpha_8 \ln(Pat/L)_{it} + CD + e_{2,it} \tag{12}$$

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 expenditure 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. Both (*Pat*/*L*) 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 S^{pat}, the empirical counterpart of *A* in Eq. (3), is allowed to vary freely and in that ideas production is positively related to the interaction between research intensity and the *DTF*. 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 coefficient of A is free to vary and, therefore, allows for the possibility that there are diminishing returns to knowledge stock as assumed in semi-endogenous growth models. The mortality rates among the working age population are assumed to directly influence ideas production independently of research intensity.

The productivity growth model, Eq. (12), 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 following the predictions of semi-endogenous growth models. Consistently with the Howitt (2005) model, human capital influences productivity growth through the Δh term. The level of human capital was also included in the initial regressions; however, it was insignificant or negative and, consequently, omitted. This result makes intuitive sense in that educational workers are unlikely to continuously innovate unless they are employed as R&D workers.

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. Labor productivity, as noted above, is measured as real GDP divided by economy-wide employment and annual hours worked. 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, adopted the Western schooling system in the early 19th century.

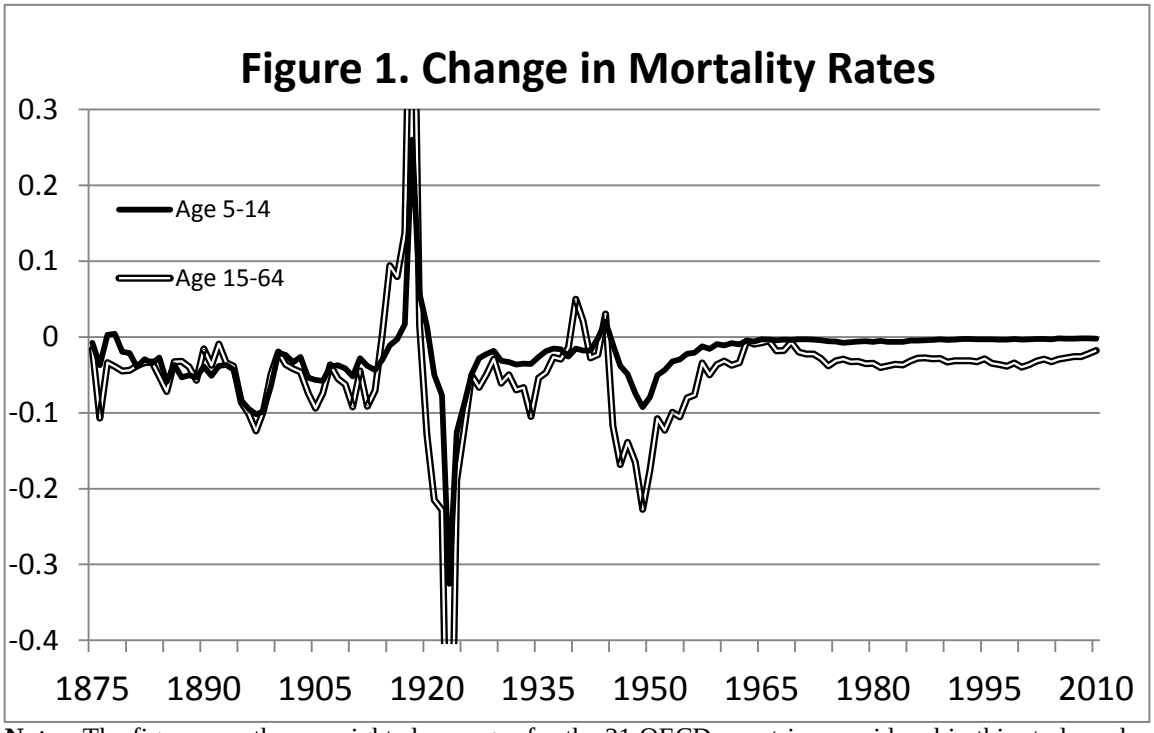
Knowledge spillovers through the channel of imports of intermediate products that contain new technology from country j to country i are computed from the following weighting scheme suggested by Lichtenberg and van Pottelsberghe de la Potterie (1998) and modified by Madsen (2007) to smooth out erratic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

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

where M_{if}^{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).

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, which are required back to 1812 in order to compute the health-adjusted GERs, are available over the whole sample period for quite a few countries, and these in turn are used to backdate the data for other countries that have missing data, as discussed in the Data Appendix.

Figure 1 displays the five-year changes in mortality rates in the 5-14 and 15-64 age cohorts, where the former captures the school-age population while the latter is 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 less than 20% of the mortality growth in 1918, suggesting that the Spanish flu was the dominant cause of the mortality hike in 1918 and, therefore, it is important to incorporate the 1918-mortality spike into the health-adjusted human capital.



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

Up until the end of WWII the changes in mortality rates are quite similar 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, which has predominantly affected the working-age cohort (Preston, 1996). Common for both graphs is that the fastest mortality decline occurred before 1960, a time 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 age-dependent mortality rates in the ideas production function as well as for the estimates of $h(\Phi)$ in the productivity growth regressions because 1) mortalities may be endogenous; 2) a third factor may drive mortalities and the economic outcome simultaneously; and 3) the instruments improve identification if they capture morbidity better than mortality. To reduce the potential bias due to endogeneity and measurement errors the generalized least squares (GLS) regressions, in which the covariance matrix is weighted by the cross-country residual correlations,

are complemented with IV-GLS regressions.

The instrument set consists of infant mortality and relative food prices. The quality of these instruments has to be judged against the fact that it is incredibly difficult and labor intensive to find alternative instruments based on annual data that span two centuries for all the 21 countries considered here. 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 to be completely exogenous it nevertheless benefits from being 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 cows' milk; a factor that would not have been that influential for adult mortality. Infant mortality is excluded from the instrument set in some of the regressions.

Relative food prices, P^F/P, are measured as food prices, P^F, divided by the GDP-deflator, P. Food prices are not deflated by consumer prices since, for many countries, consumer prices often consist of food prices only before WWI and even later for some countries. Relative food prices serve as good instruments for mortality because high food prices, before around the 1950s, were major approximate causes of starvation and protein-energy malnutrition. Although unknown to the modern industrialized world today, protein-energy malnutrition and starvation were widespread in the 19th century and up to WWII and immediately thereafter. Since fluctuations in relative food prices were almost entirely determined by the local weather conditions and food shortages due to wars well into the 20th century they can be considered exogenous. Furthermore, the food supply effects of crop failures were not counterbalanced by international food supply as the poor people affected by the crop failures did not have sufficient savings or access to credit to smooth out the food purchase. The Great Irish Famine of 1845, the famine of 1866 in Finland and Northern Sweden, the Greek famine of 1941-1944 and the starvation in the Netherlands in 1944 are examples of how food shortages had the most extreme consequences among the countries considered here.

Furthermore, relative food prices are likely to capture morbidity better than mortalities because protein-energy malnutrition increases the risk of infection and infectious disease, and moderate malnutrition weakens every part of the immune system and is a major risk factor in the onset of active tuberculosis and often led to intestinal infection, helminth, measles and influenza (Schaible and Kaufmann, 2007). Furthermore, stimulation of an immune response by infection

increases the demand for metabolically derived anabolic energy and associated substrates, leading to a synergistic vicious cycle of adverse nutritional status and increased susceptibility to infection (Schaible and Kaufmann, 2007).

4.3 First round IV-regressions

The first-round IV regressions are displayed in Table 1. Country dummies are included in all regressions. Time-dummies are not included in the instrument set because they are not valid instruments but may just capture a common time-profile that may have been caused by a third and unknown factors. However, the principal results are unaltered if time-dummies are included in the regressions. A one-period lag of the dependent variable is included in the regressions to deal with the serial correlation that would otherwise have affected the regressions. The first-round regressions are carried out for working age mortalities (in ideas production) and mortalities for the age groups at which students did their primary, secondary and tertiary education (human capital production).

Infant mortality as well as real food prices are highly significant determinants of working age mortality as shown in the first two columns. The *F*-tests for excluded instruments are 37 (including infant mortality) and 19 (excluding infant mortality). Infant mortality and real food prices remain highly significant for mortalities in the ages in which secondary and tertiary education are undertaken and the *F*-tests of excluded instruments are correspondingly high. The instruments are the least significant for the primary schooling age cohort; however, the *F*-tests for excluded instruments are still in the satisfactory range between 9 and 15. Overall, the first-round regressions indicate that infant mortality as well as real food prices are adequate instruments for age dependent mortalities in terms of explanatory power. Furthermore, the coefficients of the instruments are all positive and therefore, of the expected sign.

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

Dep. Var.	$\Delta \ln m_{it}^{wa}$	$\Delta \ln m_{it}^{w}$	m^P	m^S	m^T	m^P	m^S	m^T
Lagged Dep. Var.	-0.208 (5.47)	-0.206 (5.20)	0.96 (266)	0.95 (201)	0.92 (146)	0.95 (232)	0.87 (114)	0.79 (82.4)
P^F/P	0.044 (7.24)	0.048 (5.20)	0.10 (2.98)	0.06 (4.34)	0.17 (5.53)	0.07 (2.20)	0.07 (5.26)	0.17 (5.85)
m^0	0.002 (7.24)					0.004 (4.58)	0.21 (13.6)	0.57 (16.7)
$F(r, N\text{-}22\text{-}r)$	36.7	19.3	8.88	18.9	30.7	14.9	103	255

Est. period	1875-2009	1875-2009	1812-2009	1819-2009	1824-2009	1812-2009	1819-2009	1824-2009
N	588	588	4356	4202	4092	4356	4202	4092

Notes: the numbers in parentheses are absolute *t*-statistics; m^0 is infant mortality; P^F is food prices; P is the GDP deflator; and F = F-test for excluded instruments, distributed as $F(1, N\text{-}23\text{-}r)$ under the null hypothesis that the coefficient of P^F/P is zero, where r is the number of restrictions (excluded instruments) and N is the number of observations. The independent variables are measured in five-year differences in the regression in the first column in which $\Delta \ln m_{it}^{wa}$ is the dependent variable; annual data are used in the other regressions. Country dummies and constant terms are included all regressions.

4.4 Estimation results: Ideas production

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 rejected because the coefficients of A are highly significant; however, increasing returns to knowledge are highly 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 and significant as predicted by theory. The coefficients of research intensity are all significantly positive as predicted by Schumpeterian theory.

The coefficients of working age mortality rates are highly significant and negative regardless of whether mortality is instrumented and regardless of which instrument set is used. Economically, the decline in working age mortality in the OECD countries over the period from 1875 to 2009, on average, has contributed 0.8% (1.4%) to annual growth in patents using the estimates in the first (second) column. This corresponds to 20.9% (35.3%) of the average growth in patents over the same period. Thus, the mortality decline has, quantitatively been very influential for the growth in patenting. Interestingly, working age mortality is statistically and economically more significant when real food prices only are used as instruments (column 2) than when real food prices as well as infant mortality rates are used as instruments (column 3), indicating that real food prices are sufficient instruments for working age mortalities. Coupled with the finding of scale effects in ideas production, these results imply that mortality as well as research intensity have permanent growth effects. If R&D expenditure is 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. In the extreme case mortality rates may override positive growth effects from R&D and other factors (as captured by the constant term), and productivity growth would become negative. However, technological regress is an unlikely event

and could only occur if a large scale epidemic got to a stage where past innovations were forgotten.

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

Dep. Var.	$\Delta ln\dot{A}_{it}$	$\Delta ln\dot{A}_{it}$	$\Delta ln\dot{A}_{it}$	$\Delta ln\dot{A}_{it}$	$\Delta ln\dot{A}_{it}$	$\Delta ln\dot{A}_{it}$
	1	2	3	4	5	6
Instruments	None	P^F/P	$P^F/P,m^0$	$P^F/P,m^0$	$P^F/P,m^0$	$P^F/P,m^0$
$\Delta ln(R/Y)_{it}$	0.05(3.18)	0.06(3.49)	0.05(2.93)	0.10(7.07)	0.09(6.52)	0.009(4.46)
$\Delta ln\, m_{it}^{wa}$	-0.60(17.7)	-1.01(7.25)	-0.60(6.34)		-1.12(9.02)	
ΔlnA_{it}	1.05(61.5)	1.06(53.1)	1.05(53.3)	1.06(63.5)	1.05(61.2)	1.06(63.5)
$\left(\frac{R\&D}{Y} DTF\right)_t$	1.66(2.14)	2.52(2.65)	2.33(2.56)	2.42(3.11)	3.01(3.88)	2.36(3.08)
$\Delta lnLexp_{it}$				-0.20(1.54)	-0.20(1.57)	
Δm_{it}^0						-0.001(2.18)
DW	2.04	2.07	2.05	2.07	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. 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,... *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,...*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. 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.

Life expectancy at birth, *Lexp*, is included instead of working age mortality in column 4 to gain insight into the sensitivity of the results to health measures. The coefficient of *Lexp* is of the wrong sign and insignificant. It remains insignificant when working age mortality is added to the regression (column 5). 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 simultaneously affecting mortality and ideas production. The coefficient of infant mortality is just marginally statistically and economically insignificant, suggesting that 1) it is specifically the health among the adult population that is essential for ideas production; 2) it is unlikely that working-age mortality is capturing the effects of a third variable that influences ideas production and 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.

4.5 Estimation results: Productivity growth

4.5.1 Results when instruments are not used

The results of estimating Eq. (12) are presented in Table 3. Consider first the regression in the first column in which educational attainment is health unadjusted, that is $\Phi = 0$. The coefficient of Δh is statistically insignificant, thus, education has no direct effect on growth; a result that is consistent with the literature which finds it hard to identify any impact of human capital on growth (see, for overview, Delgado *et al*., 2011). The innovation-based variables, $\Delta \ln S^f$, $\Delta \ln Pat$, and $\ln(Pat/L)$, are all positive and highly significant as predicted by endogenous growth models. The significance of research intensity implies that 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, innovation-driven growth effects 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 increase productivity. This result is consistent with the results in the literature (see, for survey, Keller, 2004).

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

	1	2	3	4	5	6	7	8
Φ	$\Phi = 0$	$\Phi = 465$	$\Phi = 1000$	$\Phi = 0$	$\Phi = 0$	$\Phi = 303$	$\Phi = 320$	$\Phi = 287$
Instruments	None	None	None	None	None	P^F/P	P^F/P	P^F/P, m^0
Δh_t^x				0.104(16.0)				
Δh_t	0.000(0.01)	0.094(14.7)	0.060(10.0)	0.063(6.87)	-0.045(2.05)	0.075(3.64)	0.095(14.3)	0.088(13.2)
$\Delta ln S_t^f$	0.032(12.0)	0.035(17.5)	0.033(12.4)	0.035(17.0)	-0.023(2.12)	-0.014(1.19)	0.035(17.4)	0.035(16.6)
$\Delta ln Pat_t$	0.027(8.59)	0.035(14.8)	0.032(12.4)	0.036(14.6)	0.001(0.08)	0.011(0.83)	0.034(14.3)	0.035(13.3)
$ln(Pat/L)_t$	0.022(11.4)	0.009(6.37)	0.016(8.59)	0.013(7.84)	0.033(3.85)	0.026(3.28)	0.014(7.91)	0.015(7.90)
Differences	5-Year	5-Year	5-Year	5-Year	10-Year	10-Year	5-Year	5-Year
DW	2.00	1.95	1.96	1.89	1.95	1.90	1.95	1.96

Notes: The numbers in parentheses are absolute *t*-statistics and the numbers in squared brackets are *p*-values. $\Delta h_t^x = \Delta h_t(\Phi = 465) - \Delta h_t(\Phi = 0)$. 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).

The regression in the second column shows the results from the simulations that give Φ^{max} = 465, which is the value of Φ that maximizes the statistical significance of Δ*h* when instruments are not used. The coefficient of Δ*h* is highly significant, suggesting that good health is essential for students' learning and accumulation of human capital. Quantitatively, health-adjusted education has been influential for growth. Multiplying the coefficient of Δ*h* with the average value of Δ*h* in the sample and annualizing yields value of 0.00437, implying that health-adjusted educational attainment has contributed 0.437 percentage points to the annual productivity growth for the average country. With an average labor productivity growth rate of 2.22% this means that health-adjusted educational attainment has been responsible for 20% of the growth in the country sample considered here.

The growth effects of health are furthered through research intensity, *Pat*/*L*. Simulations of the model suggest that *Pat*/*L* has contributed 0.38% to productivity growth for the average country over the period 1875-2009 using the base-line regression in column 2 in Table 3. Since working age mortality explains between 21% and 35% of the variance in patents, depending on which of the estimates are used in Table 2; the effect of morbidity on growth through ideas production is around 0.1 percentage points.

Health-adjusted educational attainment is decomposed into the GER^a-term and the exp[Φ ln(1-m^a)]-term (see Eq. (8)) in the regression in column 4, where $h = h(\Phi = 0)$ and $h^x = h(\Phi = 465) - h(\Phi = 0)$. The coefficients of Δh^x and Δ*h* are both statistically highly significant and of the expected signs. This result shows that unadjusted educational attainment is influential for growth when the regressions are conditional on the health-capital during the period in which the labor force did their education. Furthermore, these results reinforce the finding in the second column that health is highly influential for leaning and subsequently for productivity when workers join the labor force. Finally, the coefficient of human capital is still highly significant if Φ is set to 1000 (column 3); however, it is quite a lot less significant than the value of Φ that maximizes the significance of Δ*h*.

4.5.2 IV results

Real food prices as well as infant mortality are used as instruments in the regressions in the last two columns of Table 3. Here, Φ^{max} = 320 when real food prices are used as instruments (column 7) and Φ^{max} = 287 when real food prices as well as infant mortality are used as instruments in (column, 8). The coefficient of human capital, Δ*h*, is statistically highly significant regardless of the instrument

set that is used and the coefficients are close to the un-instrumented regressions. Finally, all the innovation driven variables remain statistically significant.

4.5.3 Estimates in 10-year differences

Columns 5 and 6 of Table 3 present 10-year difference estimates. Note that the coefficients in the 10-year regressions are, statistically, less significant that their counterpart in 5-year differences, which reflects that the number of observations per country is too small in the 10-year estimates for the contemporaneous correlations between the residuals to be allowed for. The coefficient of human capital, Δh, is negative and only marginally significant when unadjusted human capital data are used, $\Phi = 0$ (column 5). However, it becomes statistically and economically quite significant when education is adjusted for age-dependent mortality (column 5). The maximum significance is achieved at $\Phi^{\max} = 303$, which is close to the other regressions in which $\Phi^{\max}$ is in the range of 287-465. Finally, in regards to the innovation variables, international 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.

4.5.4 GERs and mortality

Thus far it has been assumed that health influences the quality of human capital. However, health is also assumed to influence growth through the term ε in the skills production function in the model of Howitt (2005) (see Eq. (2) above). The following model is regressed to test this possibility:

$$\Delta GER_{it}^{X} = \nu_0 + \nu_1 \Delta \ln m_{it}^{X} + \nu_2 \Delta \ln y_{it} + \nu_3 \Delta \Omega_{it}^{Y} + \nu_4 \Delta \ln m_{it}^{wa} + CD + e_{3,it} \qquad (13)$$

where GER^X is GERs in X = primary (P), secondary (S) and tertiary (T) education; Ω^Y is the years of compulsory education at the Y = primary (P) and secondary (S) educational levels; m^X is the mortality rates among students at X = primary, secondary and tertiary levels of education; and Δ is the five-year difference operator. Ω^S is measured as the years of compulsory schooling (YCS) beyond primary schooling, $\Omega^S = \Omega^P - 7$ if $YCS \geq 7$; and 0, otherwise.

Productivity growth, Δlny, and the change working-age mortality are included in the

regression model following the predictions of the model of Bils and Klenow (2000) in which the return to schooling is a positive function of expected productivity growth and life expectancy. Productivity growth may also impact positively on GERs under the assumption that schooling is a normal good. Working age mortality is used instead of life expectancy because the relevant metric is the likelihood of surviving from the time the pupil enrols in school until retirement at the approximate age of 64.

The change in mortalities of students at primary, secondary and tertiary levels of education, m^X, are, again, instrumented using the changes in infant mortality as well as real food prices and a one-period lag of the dependent variable. The *F*-tests for excluded exogenous variables are $F(2, 4332) = 14.2$ (primary schooling), 34.1 (secondary schooling) and 42.7 (tertiary schooling); reinforcing that the instruments are, as far as the correlation is concerned, excellent instruments even when the variables are measured in first differences.

Table 4. Parameter estimates of GERs for secondary education (Eq. (13)).

	1	2	3	4	5	6
Dep. Var.	ΔGER_{it}^{P}	ΔGER_{it}^{S}	ΔGER_{it}^{T}	ΔGER_{it}^{P}	ΔGER_{it}^{S}	ΔGER_{it}^{T}
$\Delta\, m_{it}^{X}$	0.14(1.71)	-0.72(5.35)	-0.12(2.79)	0.14(1.68)	-0.71(4.70)	-0.12(2.06)
$\Delta \ln y_{it}$	0.005(0.86)	0.023(4.03)	0.025(4.73)	0.005(0.86)	0.024(4.11)	0.024(4.61)
$\Delta\, \Omega_{it}^{Y}$	0.008(12.3)	0.007(6.15)		0.008(12.2)	0.007(5.81)	
$\Delta \ln m_{it}^{wa}$				-0.001(0.15)	-0.001(0.26)	0.001(0.16)
DW	1.95	1.93	1.97	1.96	1.93	1.97

Notes. See Notes to Table 2. m^X = mortality rates at the ages at which students attend school, where X = primary, secondary and tertiary education and Ω^Y is the years of compulsory schooling at the Y = primary (*P*) and secondary (*S*) educational levels. Real food prices and infant mortality are used as instruments for mortality rates.

The results of regressing Eq. (13) are presented in Table 4. GER^P depends almost entirely on the years of compulsory schooling, which is highly significant, while the coefficients of growth, working age mortality and primary school age mortality rates are insignificant at conventional significance levels (columns 1 and 4). It is not surprising that the rise in primary education has been driven by compulsory schooling years and less so by other variables; expected earnings get little weight in the schooling decision at the primary school age because the opportunity costs of schooling are close to zero and because primary schooling is not only a matter of earnings potential but a matter of adequately functioning in an increasingly complicated society where literacy was increasingly taken for granted during the course of the 19th century.

Schooling age mortalities are statistically highly significant negative determinants of GER^S

and GER^T (columns 2 and 4), showing that morbidity lowers school enrolment because morbidity lowers the quality and the value of education. Economically, the decline in mortality rates among children and young adults of school age were also influential for the increasing GERs. The decline in m^S over the period from 1875 to 2009, for example, has contributed to a 3.8 percentage point increase on GER^S on average. This number is quite significant taking into account that GER^S was only 3% on average in 1875 and that the mortality decline was predominantly concentrated in the period 1875-1930. For tertiary education the decline in m^T has, on average, contributed to a 1.1 percentage point increase in GERT over the period from 1875 to 2009.

Turning to the other variables in the GER^S and GER^T regressions, the coefficients of productivity growth are positive and significant as expected and years of compulsory schooling is also influential for secondary schooling. Finally, working age mortality rates are insignificant in all the regressions (columns 4-6) suggesting that the number of years at which schooling dividends can be earned is not a key parameter for the educational decision and that broad mortality rates may not capture the effects of morbidity on schooling.

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 growth since 1870 for the 21 OECD countries considered here 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 productivity growth in the 21 OECD

countries would have been 20% higher during the period 1875-2009 had school age mortality rates been at the 2009 level during the entire period. Second, the regressions of knowledge production functions revealed that working age mortalities are highly significant determinants of ideas production. Since research intensity is also a highly significant determinant of productivity growth the health-induced growth effects are increased by approximately 0.1 percentage points through this channel. Third, it was shown that school age mortality rates, as proxies for morbidity rates, were influential for secondary and tertiary school enrolment, suggesting that health not only affects learning but also enrolment rates and, therefore, human capital.

Since productivity growth is predominantly driven by human capital and innovations the results in this paper show that declining mortality, as a proxy for morbidity, has been an important force behind the transition from the post-Malthusian to the modern growth regime in the 21 OECD countries considered here. The results may also give a clue to why some countries have converged to the technology frontier while others have not. The marked mortality decline in the second half of the 19th century and the beginning of the 20th century was, approximately, limited to the countries considered here and, therefore, may help to explain why some countries took off while others stayed behind.

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 why human capital and innovations increased in the original OECD countries since the Industrial Revolution. This paper takes one of the first steps to empirically attempting to shed light on the increase in human capital and innovations during the Industrial Revolution and suggests 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 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 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 Mills (2009) in which the Malthusian mechanism operates only through preventive checks (fertility) and not through the positive checks (mortality). Fourth, the difficulties associated with finding a positive relationship between growth and educational attainment may be related to the finding of this paper that it is health-adjusted human capital as opposed to educational attainment that is essential for growth (see, for a literature review, Delgardo *et al*., 2011).

Data Appendix

TFP, employment, international knowledge spillovers, real GDP, annual hours worked, GERs, population distributed on ages, patents and years of compulsory schooling. Madsen (2007, 2008, 2010, and 2012).

Patent stock. Inventory perpetual method using 15% depression rate applied to patents applied for. The initial patent stock is estimated as the number of patent applications in the period 1870-1874 divided by the depreciation rate (0.15) and the average growth in patent applications over the period from 1870-74 to 2005-2009. See Madsen (2007) for patent data sources.

R&D. The data do not exist for every year for most countries. The data are interpolated as follows: The ratio of R&D and nominal GDP is interpolated geometrically and R&D is recovered by multiplying by the interpolated ratio of R&D and nominal GDP by nominal GDP. The data are backdated using the ratio of R&D and Nominal GDP as follows: The data are spliced with the average R&D/Y-ratio for the countries for which the data are available. All countries 1963-1980: OECD Archive (OECD -DSTI/EAS) Contact: Elena Bernaldo de Quirós- elena.bernaldo@OECD.org. 1981-2009: OECD, Main Science and Technology Indicators, various years. Before 1962 for individual countries. USA. 1921-1952. Terleckyj, Nestor E. (1963) *Research and Development: Its Growth and Composition.* National Industries Conference Board Studies in Business Economics, No. 82. New York: The Board. 1953-2009. National Science Foundation. Japan. 1953-2009. *Historical Statistics of Japan*, Statistics Bureau and Statistical Research and Training Institute, Ministry of Internal Affairs and Communication (http://www.stat.go.jp/english/data/chouki/index.htm). Australia. 1940-1964. Table ES 249-257. Vamplew, W. (1987) *Australians, Historical Statistics*. Fairfax, Syme and Weldon Associates: New South Wales. Netherlands. 1959-1972: Statistics Netherlands, Contact: Ferry Lapré at infoservice@cbs.nl. Germany. 1870-1970. Pfetsch, Frank R. (1985) *Datenhandbuch zur*

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