HEALTH AND INCOME:

THEORY AND EVIDENCE FOR OECD COUNTRIES

Alberto BUCCI
(*University of Milan, Department of Economics, Management, and Quantitative Methods*)

Lorenzo CARBONARI
(*University of Rome ñTor Vergataò, Department of Economics and Finance*)

Giovanni TROVATO
(*University of Rome ñTor Vergataò, Department of Economics and Finance*)

Abstract

In this chapter we examine whether the Solow growth model is consistent with the international variation in the standard of living once investments in education and health are explicitly and simultaneously taken into account. Using the sample of OECD countries, we provide evidence that the level of per capita income is positively affected by the populationôs health level, here proxied by the life expectancy at birth. Public expenditure on health affects indirectly the level of per capita income through its positive effect on life expectancy. Using a Finite Mixture approach, we also show that richer countries are those in which the impact of unobserved factors is stronger in the determination of the level of per capita income.

1. INTRODUCTION: MOTIVATION AND BACKGROUND

This chapter takes Mankiw-Romer-Weil seriously. In their famous 1992ôs paper, they demonstrated that an *augmented* Solow (1956) model that includes accumulation of human as well as physical capital provides a better understanding of the international differences in income per capita. In more detail, their *augmented* Solow model reveals that differences in saving, education and population change well explain cross-country differences in living standards. However, the Mankiw *et al.* (1992)ôs story totally neglects the role of health, so in their framework the following two questions still remain open: Across the world, what is the role of health in determining a countryôs degree of economic development (*i.e.*, its level of real *per capita* income)? And, what is healthôs role in explaining cross-country differences in economic development? In this chapter we fill this gap in their analysis.

Our starting point here is, indeed, to formally recognize that human capital can appear either in the form of education/schooling or in the form of health (see Weil, 2014). Given this, our objective is to quantitatively assess the relative contribution of the health-variable (in addition to, and separately from, the education-variable) to explaining the international differences in *per capita* income, in the sample of OECD countries.

To test the *augmented* Solow model, we include proxies for human-capital accumulation and populationôs health as additional explanatory variables in our regressions. In order to deal with the potential endogeneity between income and health, we develop a parsimonious statistical approach to model different sources of heterogeneity. In particular, we assume that heterogeneity sources can be simply modeled by introducing a latent effect to each country experience, allowing for a posterior classification of countries based on the latent variable values.

According to the report that followed the second consultation of the World Health Organizationôs Commission on Macroeconomics and Health held in Geneva in 2003 (WHO, 2003):

> *é The last few years have seen the emergence of an international consensus in global public health: socio-economic development can be achieved only by rigorously promoting the implementation of pro-poor policies within a viable development strategy, financed through a significant increase in health investments. We have witnessed important achievements, including heightened attention among policy-makers to the health of the poor and recent increases in assistance for health. But more determination and resources are needed to meet the real health needs of the poor. Failure to act promptly and decisively will result in countless additional deaths and illness from preventable causes, trapping individuals and families in poverty and hindering economic growth and development.*

At the heart of such a declaration is the belief that sizeable gains, in terms of economic growth and development, can still be reaped from health improvements. Bloom *et al.* (2014), for example, estimate that, over an 18-year period, five categories of non-communicable diseases can together reduce labor supply and capital accumulation in China and India in such a way to cause an overall 34 trillion dollarsô worth damage for lost output. Our goal in this paper is to quantitatively assess the role that health differences can play in explaining income differences across countries, with a special focus on OECD ones, where results seem to be more ambiguous. Nonetheless, before doing this, we review the main theoretical, as well as empirical, literature that has analyzed the multifaceted relation between health and income.[1]

Apparently, the answer to the question of whether a better health is able to support higher productivity and standard of living seems trivial: Of course, yes! After all, healthy people lose less time from work due to ill-health and, when working, are more productive (both physically and mentally). In a word, better health contributes without any doubt to increase directly labor market participation and workersô productivity (Strauss and Thomas, 1998; Bloom and Canning, 2000; Schultz, 2002). At the same time, better health appear to have also a number of other indirect positive effects on the level (and growth rate) of per capita income through, for example, a change in the individualsô incentives to invest in human and physical capital, to save, and eventually through a change in the population growth rate, as well.

However, four major difficulties do immediately arise when one tries to assess the whole existing work in this area. The first is the measurement issue. Health is measured differently across various studies (measures employed in microeconomic analyses are generally diverse from those

used in macroeconomic models). Moreover, while some works focus on the influence of specific diseases on income per capita,[2] others instead look at the economic effects of more ñaggregativeò measures of health (such as life expectancy, or survival, say).

The second issue has to do with causality: the correlation between health and income can result either from income causing health, or from health causing income, or from some other factor(s) causing both, or else from some combination of these three channels all together.

The third issue concerns the existence of healthôs **ópartial-equilibriumô** effects (*i.e.*, those arising from holding all other factors fixed), as opposed to its **ógeneral equilibriumô** effects (*i.e.*, those stemming from a framework where other factors that respond to improved health are explicitly and jointly considered in the analysis). In other words, while some studies focus only on the proximate (or direct) impact of health, others attempt to capture also its indirect economic effects.

Last but not least, the final issue is related to the composition of the sample (developing *vs.* developed, or pre-demographic-transition *vs.* post-demographic-transition countries), with regard to which the consequences of health improvements are evaluated and that in empirical studies play a crucial role in determining the sign of the possible correlation between health and income.

In the last few years there has been an upsurge in the empirical and theoretical work on the nexus between health and income. In this section we briefly review such advancements, with a special eye on the four major difficulties briefly outlined above.

THE MEASUREMENT OF HEALTH: Research examining the link between health and economic outcomes generally employs two types of health measures: health inputs and health outcomes (Weil, 2007, pp. 1268-1269). Health inputs are all those physical factors that may influence an individualôs health. These include, but are not limited to, nutrition at various points in life (*in utero,* in childhood, in adulthood), exposure to various forms of pathogens, availability of medical care, etc. Health outcomes are all those personal characteristics that are determined either by an individualôs health inputs or her/his genetic endowments (life expectancy, survival rate,

height, the ability to work harder and produce more than others are all possible examples of health outcomes). In turn, health outcomes can be either non-observable ï a notable example is what Weil (2007 and 2014) defines as *"human capital in the form of health"*, that measures how health affects one's ability to produce output[3] ï , or observable (in this case health outcomes are called "health indicators").

An extensive literature examines the effects of health, defined and measured in several ways, on *individual* (as opposed to national or aggregate) economic outcomes. Examples of this approach include Fogel (1997), Behrman and Rosenzweig (2004), Black *et al.* (2007), Almond (2006), Bleakley (2007), and Miguel and Kremer (2004), among many others. Fogel (1997) studies the effects of better nutrition on output per worker and labor supply over an interval of 200 years. Behrman and Rosenzweig (2004) use variations in birth weight among identical twins to identify the effect of fetal nutrition on education and wages among adults. Their main estimate regresses the gap in the dependent variable (height, log wages, or schooling) between a pair of twins on the gap in fetal growth. They find that a one-unit difference in fetal growth (measured in ounces per week of gestation) leads to a difference of 0.657 years of schooling, 3.76 centimeters of adult height, and 0.190 gap in log wages. Black *et al.* (2007) perform a similar analysis using data on Norwegian men. For the full sample, their within-twin-pair estimate of the effect of log birthweight on log earnings is 0.24. Almond (2006) shows that individuals exposed to Spanish influenza in utero had lower education attainment and higher rates of disability than surrounding cohorts. Bleakley (2007) and Miguel and Kremer (2004) find that treatment with deworming drugs increases school attendance. Overall, microeconomic studies are mainly aimed at assessing the economic consequences of health inputs (or morbidity-measures) at an individual level. On the contrary, in macroeconomic models population health is usually taken to be life expectancy, or some other mortality-measures (as opposed to the morbidity ones employed at the individual, or

microeconomic, level).[4] It is therefore quite difficult to compare studies that use such different notions of health.

CAUSALITY: While it is now accepted that high levels of population health go hand in hand with high levels of national income, it still remains disputable whether better health is the consequence or the cause of higher income (Adams *et al.*, 2003). The most important piece of evidence of a positive correlation between income and health is represented by the so-called *"Preston Curve"* (after the path-breaking work by Preston, 1975), that shows the link between GDP per capita (on the horizontal axis) and life expectancy at birth (on the vertical axis). One intuitive reason for this positive correlation to exist is that higher income generally allows for better food, shelter, cleaner water and sanitation, and enhanced medical treatments. Moreover, countries that are richer can afford higher expenditures on public health. According to Preston (1975), the increase in life expectancy experienced in a country over time can be decomposed into two different parts: the part due to higher income (this is the movement along the Preston curve), and the shift in the whole curve. Prestonôs calculations show that less than one quarter of the average mortality-improvement observed between 1930 and 1960 was due to movement along the curve, with the remainder due to shifts in the curve. In other words, income gains were not the primary source of health improvements (health interventions can improve individualsô health without the need for prior improvements in their incomes). As far as the causal link between income and health is concerned, Pritchett and Summers (1996), Bloom and Canning (2000), Bloom, Canning, and Sevilla (2003), and Bloom and Fink (2014) all focus on the channel that runs from health to income, while Cutler *et al.* (2006) and Hall and Jones (2007) are examples of how the reverse causal channel (going from income to health) can work. The existence of complementarity between health and education make the causality problem even worse as education exhibits the same two-way causality in its association with income (Becker, 2007).

Two alternative methods are employed by the literature to assess the overall effects of health on income and economic growth.[5] The first aggregates the results of Mincerian wage-regressions of the return on individual health to derive the macroeconomic effects of population health. The second, instead, relies on the estimation of a generalized aggregate production function that decomposes human capital into its components (including health). While most of the studies based on both methods point to a positive effect of health on economic growth, the size of such effect remains, however, subject to debate (it is generally found to be smaller in the first and larger in the second type of studies). Weil (2007) has been among the first to use the available microeconomic estimates to quantitatively evaluate the importance of health at the macroeconomic level. More specifically, he constructs a framework in which estimates of the effect of variation in health-inputs on individual wages can be used to generate estimates of how differences in health, as measured by observable outcomes, contribute in turn to differences in national income. In so doing, Weil (2007) extends the development accounting methodology of Klenow and Rodriguez-Clare (1997) and Hall and Jones (1999) to include also a measure of health. He finds that eliminating health gaps among countries would reduce the variance of log GDP per worker by about 9.9 percent. The conclusion is therefore that while the estimated effect of health on income is positive and economically significant, it is also much smaller than existing estimates derived from cross-country regressions would suggest. Bloom, Canning, and Sevilla (2004, pp. 2-4) report the results of thirteen such studies, which mostly reach similar quantitative results. Their own estimate, which comes from regressing residual productivity (after accounting for physical capital and education) on health measures in a panel of countries observed every 10 years over 1960-1990 is that a one-year increase in life expectancy raises output by 4 percent. In a paper companion to Weil (2007), Shastry and Weil (2003) calibrate a production function model of aggregate output using microeconomic estimates of the return to health. They assume a stable relationship between average height and adult survival rates so that when adult survival rates improve they can infer a rise in population

heights. Using estimates of the effect of height on worker productivity and wages from microeconomic studies they calibrate what health improvements in the form of changes in adult survival rates should mean for aggregate output. They find (Table 3, p. 395) that cross-country gaps in income levels can be explained in part (20.1%) by differential levels of physical capital, in part (21.6%) by differential levels of education, and in part (19%) by differential levels of health. Hence, over half of cross-country differences in income levels can be explained by these three factors, the remainder of the gap (39.3%) being ascribed, instead, to differences in total factor productivity.

In an influential macro-level paper addressing the issue of whether health may or not have an effect on income, Acemoglu and Johnson (2007) use panel data for 47 countries and exploit the drop in mortality from specific infectious diseases, due to the international epidemiological transition, as an instrument for the change in life expectancy. This identification strategy makes use of the fact that the mortality rate from these diseases was exogenous in 1940, because no treatments, medication, or vaccines were available before that time. Starting from 1980, instead, all these diseases can be treated or prevented in all countries, due to medical advances. After regressing per capita income growth on the increase in life expectancy between 1940 and 1980, Acemoglu and Johnson (2007) report a positive but insignificant effect of increased life expectancy on *aggregate GDP*, and a positive and significant effect on population growth. The overall effect on *GDP per capita* is found to be negative (which means that countries that experienced larger exogenous health improvements saw lower gains in income per capita). Acemoglu and Johnson (2007) attribute their findings to the fact that increases in health result mainly in large increases in population. In turn, as it is well known from Solow (1956), the capital dilution effect associated to a faster population growth reduces income per capita at the steady state. Therefore, in the end improved health lowers per capita income. However, the Acemoglu and Johnson (2007)ôs methodology has been challenged as it regresses economic growth against health improvements without including initial health in the

model. As such, the negative correlation between health improvements and economic growth shown in their analysis may simply be the consequence of the fact that countries starting with better health economically grow faster (while experiencing smaller improvements in health) than those starting with lower initial health conditions and that at the same time experiment larger health improvements.[6] To study this possibility, Aghion *et al.* (2011) and Bloom, Canning, and Fink (2014) include initial health in the Acemoglu and Johnson (2007) regressions and find that, indeed, the negative causal effect vanishes.[7] More specifically, Aghion *et al.* (2011) combine the so-called Mankiw-Romer-Weil (1992)ôs approach (whereby output growth is correlated with the rate of improvement in human capital) with the so-called Nelson-Phelps (1966)ôs approach (whereby a higher level of health should spur growth by facilitating technological innovation), and look at the joint effect of health and health accumulation on economic growth. After running cross-country growth regressions over the period 1960-2000, they show that the level and the accumulation of health have significant positive effects on per capita income growth.[8] Finally, they find a weaker relationship between health and growth over the contemporary period in OECD countries. According to them, this is explained by the fact that only gains in life expectancy below 40 years are significantly correlated with per capita income growth.

DIRECT *VS.* INDIRECT HEALTHôS ECONOMIC EFFECTS: According to Weil (2007, p. 1266), there are

> ***ñé*** *several channels through which health affects the level of output in a country. One channel, which I call the proximate or direct effect of health, is that healthier people****é*** *can work harder and longer and also think more clearly. Beyond this proximate effect of health, there are a number of indirect channels through which health affects output. Improvements in health raise the incentive to acquire schooling since investments in schooling can be amortized over a longer working life. Healthier students also have lower absenteeism and higher cognitive functioning and, thus, receive a better education for a given level of schooling. Improvements in mortality may also lead people to save for retirement, thus raising the levels of investment and physical capital per worker. Physical capital per worker may also rise because the increase in labor input from healthier workers will increase capitalôs marginal product. The effect of better health on population growth is ambiguous. In the short run, higher child survival leads to more rapid population growth. Over longer horizons, however, lower infant and child mortality may lead to a more-than-offsetting decline in fertility, so that the net rate of reproduction falls****é ò***.[9]

Further than the (direct and/or indirect) effects of life expectancy on income, reductions in mortality may also increase individual wellbeing by not only extending the lifetime horizon but also by improving peopleôs quality of life (Becker *et al.*, 2005, and Murphy and Topel, 2006). By looking only at the direct, or proximate, effects of health on income, and thus holding constant the level of physical capital, education, the quality of institutions and so forth, Weil (2007) concludes that the size of the impact is relatively small, and definitely littler than the estimated effect of health on economic growth that is obtained from cross-country regressions. To translate Weil (2007)ôs conclusions in numbers, he finds that a health improvement that raises life expectancy by five years would increase labor productivity by 3.6 percent and output per capita in the steady state by the same amount. In order to have a raw idea of what these figures might imply notice that along the 2010ôs Preston curve an increase in life expectancy of five years would have been associated with a *doubling* of output per capita.

All this said, the positive effects ultimately accruing to economic growth from a better health (no matter how it is measured) through, say, education are undeniable. In this regard, Ben-Porath (1967) was among the first to show that if people live longer then investments in human capital are more likely to pay off because the working life is lengthier. This implies an increase in the return to individualsô (or their childrenôs) human capital investment, hence higher incentives in investing in skills. De la Croix and Licandro (1999), Kalemli-Ozcan *et al.* (2000), and Boucekkine *et al.* (2002 and 2003) build models in which a decline in mortality produces greater investments in individual human capital and, therefore, a rise of economic growth. Chakraborty (2004) reaches similar conclusions in a model where longevity is made endogenous by public health investments. Cervellati and Sunde (2005) and Soares (2005) consider settings in which the mortality decline pushes parents to have (fewer but) better educated children. Bleakley and Lange (2009), and Jayachandran and Lleras-Muney (2009) provide robust and convincing evidence that higher life expectancy increases educational attainments at the individual level.

Concerning the impact of health improvements on economic growth by way of individualsô saving decisions, Blanchard (1985)[10] analyzes the growth effects of an increase in life expectancy in developed economies by replacing the representative agent assumption of the standard neoclassical growth theory with an overlapping-generations structure in which individuals face a constant risk of death. In his framework, an increase in life expectancy raises aggregate savings and therefore, according to the canonical mechanism of the neoclassic growth model, the growth rate of the economy during the transition to the steady state. Hurd *et al.* (1998) find that increased expectation of longevity leads to greater householdôs wealth in the United States. Bloom, Canning, and Graham (2003) find an effect of life expectancy on national savings, using cross-country data. Lee *et al.* (2000) argue that rising life expectancy accounts for the boom in savings in Taiwan since the 1960s. Finally, Zhang and Zhang (2005) construct a three-period overlapping-generations model showing that rising longevity reduces fertility and enhances savings and schooling investment, even though these effects are empirically quite small.

The regression results of Madsen (2016) show that health has been highly influential for economic growth since 1870 for the 21 OECD countries considered not only through human capital investment, but also through ideas-production, the two core drivers of modern technological change and economic growth. A direct (and intuitive) effect of health on human capital is that sick children are often absent from school (Mayer-Foulkes, 2005; Bundy *et al.*, 2006; Currie *et al.*, 2010). More importantly, illness can severely diminish the learning capacity of students because of reduced concentration in the classroom, cognitive impairment, and stigma (Holding and Snow, 2001; Alderman *et al.*, 2005; Mayer-Foulkes, 2005; Bloom and Canning, 2009). Furthermore, chronic poor health can adversely affect ideas production because it impairs creativity and entrepreneurship (Howitt, 2005). Last but not least, recent research shows that societies with high pathogen stress are less innovative, less open to new ideas, and display introversion (Schaller and Murray, 2008; Fincher *et al.*, 2008). According to Madsen (2016)ôs findings, while working-age mortality rates are

highly significant determinants of ideas production, school-age mortality rates (as proxies for morbidity rates) are especially influential on secondary and tertiary school enrolment, suggesting that health affects not only learning but also enrollment rates, and ultimately human capital accumulation. These results are consistent with the empirical estimates of Chakraborty (2004), who shows that initial educational attainment in cross-country growth regressions is insignificant once initial life expectancy is included as an explanatory variable in the regressions, indicating that health affects economic growth through educational attainment.

Another channel through which health improvements may (indirectly) affect economic growth and development is represented by the so-called *"demographic dividend"*.[11] A common feature of every episode of demographic transition (Lee, 2003, and Bloom and Canning, 2009) is that it generally starts with a reduction in mortality rates, while birth rates still remain high. With some delay, fertility also drops. The delay between the initial reduction in mortality and the subsequent decline in fertility induces the typical hump-shaped pattern of population growth, which initially increases (due to lower mortality rates), but eventually slows down, if reduced fertility more than compensates the initial increase in population. As fertility begins to fall, the overall dependency ratio (the sum of the youth and the old-age dependency ratios) may decrease, as well. If this happens, the resulting *"demographic dividend"* releases resources that can be invested in further health, education and infrastructure improvements , so speeding up the transition towards a phase of sustained long-run growth. All this ultimately suggests that the demographic transition plays a central role in determining the sign of the effect of life expectancy on income per capita growth (see Cervellati and Sunde, 2011, and our discussion below).

Ashraf *et al.* (2009) undertake a detailed analysis of the different channels though which health affects output. Hence, they go beyond the static analysis of Weil (2007) in order to uncover also the dynamic effects of health shocks. Their simulation model allows for several channels through which health improvements may have an economic impact, including the effect of better health on human

capital investment, the change in population growth triggered by increased survival rates, and the possible response of fertility to increased child survival. However, as in Weil (2007), the effect that they find is still relatively modest: an increase in life expectancy from forty to sixty years would raise GDP per capita in the long run by only 15 percent, and for the first thirty years after such an increase, output per capita would be lower than if health had not improved at all. Overall, their results imply that causation from health to income does not drive much of the observed cross-country correlation between the two variables.

HEALTHôS EFFECTS ACROSS DIFFERENT SAMPLE-COMPOSITIONS: Weil (2007, p. 1295, and 2005, pp. 153-161) suggests that healthôs positive effect on GDP is strongest across poor countries. For richer countries, instead, the existing empirical evidence is mixed. For instance, while Rivera and Currais (1999a, 1999b, 2003, 2004) find a positive effect of health expenditure growth on productivity growth for OECD countries (or Spanish regions), Knowles and Owen (1995, 1997) as well as McDonald and Roberts (2002), reject the hypothesis that life expectancy is a statistically significant explanatory variable for productivity growth in high-income countries. Bhargava *et al.* (2001) even estimate a negative effect of the adult survival rate on economic growth for the US, France, and Switzerland. Hartwig (2010) looks at health-data gathered for 21 OECD countries, and finds no evidence that either health care expenditure or the rise in life expectancy positively Granger-cause per capita GDP growth. On the contrary, when per capita GDP growth is regressed on its own lags and on lags of per capita health care expenditure growth in a panel Granger-causality testing framework, the coefficients for lagged health care expenditure growth are robustly negative (even though the statistical significance of the negative coefficients is not robust to the choice of the GMM estimator).

Recently, Cervellati and Sunde (2011) have tested the hypothesis that the causal effect of life expectancy on income per capita growth is non-monotonic, and convincingly show that the inception of the demographic transition represents an important turning-point in the analysis of the

sign of such causal relation. In more detail, they document the presence of a strong and robust positive causal effect of life expectancy on income per capita in *post-transitional countries* (in which it is also possible to observe a significantly negative effect of life expectancy on population growth). On the other hand, they notice a negative (although sometimes insignificant) causal effect of life expectancy on income per capita in *pre-transitional countries* (where the effect of life expectancy on population growth is generally positive). In sum, Cervellati and Sunde (2011)[12] show that longevity improvements stimulate economic growth only if a country has already undergone the demographic transition from high to low rates of fertility and mortality. Otherwise, such improvements merely translate into greater population growth. Overall, their results *ñé help reconcile the seemingly contradictory empirical findings in the existing literature. The analysis documents that the mixed results obtained in the literature are not due to the use of different instrumentation strategies. In the presence of non-monotonic effects, the estimates of the causal effect of life expectancy on income growth obtained with a linear estimation framework may be misleading since they crucially depend on the sample composition in terms of pre-transitional and post-transitional countries. The key role of sample composition is documented by weighted estimations on the full sample in a linear framework. The effect of life expectancy on income growth is more positive the larger the weight given to countries that have larger initial life expectancy or lower crude birth rates in 1940. These are the countries which are more likely to be post-transitionalé ò* (Cervellati and Sunde, 2011, p. 131).

After reviewing the main literature that has studied the relation between health and income (level and/or growth), we derive in the next section the *augmented* Solow model while the econometric analysis is developed in Section xx.3, in which we also present the estimates and show how unobserved heterogeneity can help in explaining differences among countries. A discussion of the results with a comparison to the closest papers to ours is provided in Section xx.4. Section xx.5 concludes.

2. THE ENVIRONMENT

Consider an economy where the rates of saving (s), population growth (n) and technological progress (g) are all exogenous. In this economy there are four rival inputs: capital (K), raw labor (L), human capital in the form of education (E), and human capital in the form of health (H). Following Barro (2013, Eq. 2, p. 352), we assume that production at time t takes the following Cobb-Douglas form:

$$Y_t = K_t^{\alpha} E_t^{\beta} H_t^{\gamma} \left(A_t L_t\right)^{1-\alpha-\beta-\gamma}, \qquad 0<\alpha, \beta, \gamma<1, \qquad 0<\alpha+\beta+\gamma<1 \tag{xx.1}$$

Notice that in Eq. (xx.1) we assume that there are decreasing returns to all capital $\left(0<\alpha+\beta+\gamma<1\right)$, and that raw labor, the stock of physical capital, the stock of human capital in the form of education and the stock of human capital in the form of health are considered as four different inputs in the same aggregate production function. This means that output depends not only on *"conventional"* inputs (such as physical capital, raw labor, and human capital in the form of schooling),[13] but also on workers' health. Moreover, Eq. (xx.1) assumes that technological progress (*i.e.*, the growth over time of the level of technology, A_t) is labor-augmenting , and that the contribution to total GDP of raw labor, human capital in the form of education and human capital in the form of health (as reflected, respectively, by the elasticities $\left(1-\alpha-\beta-\gamma\right)$, β, and γ) is potentially dissimilar across each other and different from that of physical capital, as well. For the sake of simplicity, the total of labor input (L) is also assumed to correspond to total population. The dynamics of the size of population and the level of technology are exogenous and obey, respectively to:

$$L_t = L_0 e^{nt} \tag{xx.2}$$

$$A_t = A_0 e^{gt}. \tag{xx.3}$$

The number of effective units of labor is $A_t L_t$, and grows at rate $(n+g)$. Physical capital, human capital in the form of education and human capital in the form of health are three reproducible factor inputs. The economy-wide budget constraint is:

$$Y_t = K_t^{\alpha} E_t^{\beta} H_t^{\gamma} \left(A_t L_t\right)^{1-\alpha-\beta-\gamma} = C_t + I_{E,t} + I_{H,t} + I_{K,t}. \quad \text{(xx.4)}$$

Thus, the same production function applies to physical capital, education, health, and consumption: once produced, one unit of forgone consumption can be transformed costlessly into either one unit of physical capital or one unit of human capital in the form of schooling or one unit of human capital in the form of health.

After defining by:

$$k_t \equiv K_t / A_t L_t, \qquad e_t \equiv E_t / A_t L_t, \qquad h_t \equiv H_t / A_t L_t, \quad \text{(xx.5)}$$

the variables K_t, E_t and H_t per unit of effective labor, it is possible to express the production function in intensive form as:

$$y_t \equiv Y_t / A_t L_t = k_t^{\alpha} e_t^{\beta} h_t^{\gamma}. \quad \text{(xx.6)}$$

Let s_k, s_e and s_h be, respectively, the exogenous fractions of total income invested in physical capital, education and health, with $s \equiv s_k + s_e + s_h$ being the total saving rate of the economy. The evolution of the three capital stocks is determined as follows:

$$\dot{k}_t = s_k y_t - (n+g+\delta) k_t. \quad \text{(xx.7)}$$

$$\dot{e}_t = s_e y_t - (n+g+d) e_t. \quad \text{(xx.8)}$$

$$\dot{h}_t = s_h y_t - (n+g+d) h_t. \quad \text{(xx.9)}$$

We continue to follow Barro (2013, p. 353) in assuming that the exogenous depreciation rate for physical capital ($\delta > 0$) differs from the exogenous depreciation rate for education and health ($d > 0$).

Eqs. (xx.7), (xx.8) and (xx.9) imply that the economy converges to a steady state equilibrium (defined by $\dot{k}_t = \dot{e}_t = \dot{h}_t = 0$) in which:

$$h^* = \left[\frac{s_k^{\alpha} s_e^{\beta} s_h^{1-\alpha-\beta}}{\left(n+g+d\right)^{1-\alpha} \left(n+g+\delta\right)^{\alpha}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}}. \tag{xx.10}$$

$$e^* = \left[\frac{s_k^{\alpha} s_h^{\gamma} s_e^{1-\alpha-\gamma}}{\left(n+g+d\right)^{1-\alpha} \left(n+g+\delta\right)^{\alpha}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}}. \tag{xx.11}$$

$$k^* = \left[\frac{s_e^{\beta} s_h^{\gamma} s_k^{1-\beta-\gamma}}{\left(n+g+d\right)^{\beta+\gamma} \left(n+g+\delta\right)^{1-\beta-\gamma}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}}. \tag{xx.12}$$

Substituting Eqs. (xx.10), (xx.11) and (xx.12) into the production function in intensive form (Eq. xx.6) gives the steady state level of *per capita* income, $\left(Y_t / L_t\right)^*$:

$$\left(\frac{Y_t}{L_t}\right)^* = \left(k^*\right)^{\alpha} \left(e^*\right)^{\beta} \left(h^*\right)^{\gamma} A_t = \left[\frac{s_e^{\beta} s_h^{\gamma} s_k^{\alpha}}{\left(n+g+d\right)^{\beta+\gamma} \left(n+g+\delta\right)^{\alpha}} \right]^{\frac{1}{1-\alpha-\beta-\gamma}} A_0 e^{gt} \tag{xx.13}$$

This equation shows how in the steady state *per capita* income depends on the rates of investment in education, physical capital and health, the rate of population growth, the rate of technical change, and the depreciation rates for schooling/health and physical capital. After taking logs of both sides of Eq. (xx.13), this equation can be transformed into:

$$\begin{aligned} \ln\left(\frac{Y_t}{L_t}\right)^* = \ln A_0 + gt - \left(\frac{\beta+\gamma}{1-\alpha-\beta-\gamma}\right)\ln\left(n+g+d\right) - \left(\frac{\alpha}{1-\alpha-\beta-\gamma}\right)\ln\left(n+g+\delta\right) \\ + \left(\frac{\beta}{1-\alpha-\beta-\gamma}\right)\ln\left(s_e\right) + \left(\frac{\gamma}{1-\alpha-\beta-\gamma}\right)\ln\left(s_h\right) + \left(\frac{\alpha}{1-\alpha-\beta-\gamma}\right)\ln\left(s_k\right) \end{aligned} \tag{xx.14}$$

In order to make more explicit the role of health in determining the steady state level of *per capita* income, from Eq. (xx.10) we first obtain:

$$\left(\frac{\gamma}{1-\alpha-\beta-\gamma}\right)\ln(s_h)=\left(\frac{\gamma}{1-\alpha-\beta}\right)\ln(h^*)-\frac{\alpha\gamma}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(s_k)-$$
$$\frac{\beta\gamma}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(s_e)+\frac{\gamma(1-\alpha)}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(n+g+d)+$$
$$\frac{\gamma\alpha}{(1-\alpha-\beta)(1-\alpha-\beta-\gamma)}\ln(n+g+\delta)$$

Then, after plugging the last expression into Eq. (xx.14), we finally obtain:

$$\ln\left(\frac{Y_t}{L_t}\right)^*=\ln A_0+gt+\left(\frac{\beta}{1-\alpha-\beta}\right)\ln(s_e)+\left(\frac{\alpha}{1-\alpha-\beta}\right)\ln(s_k)$$
$$-\left(\frac{\beta}{1-\alpha-\beta}\right)\ln(n+g+d)-\left(\frac{\alpha}{1-\alpha-\beta}\right)\ln(n+g+\delta)+\left(\frac{\gamma}{1-\alpha-\beta}\right)\ln(h^*) \quad \text{(xx.15)}$$

Eq. (xx.15) yields an expression for the steady state level of *per capita* income as a function of (some of) the same variables already mentioned earlier and, more importantly, of the level of health in the population (h^*). In the next Section we implement empirically the theoretical model presented here.

3. EMPIRICS

As pointed out in the previous section, the effect of health on real GDP is far from univocal. A large body of both theoretical and empirical literature shows a positive impact of health on economic growth, but for rich countries, the existing empirical evidence is mixed (Hartwig, 2010). In this section, we revisit the question whether health capital formation stimulates the growth of real income in advanced countries.

DATA. Our sample consists of 31 high income OECD countries (Australia, Austria, Belgium, Canada, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Iceland, Ireland, Israel, Italy, Japan, Luxembourg, Netherlands, New Zealand, Norway, Poland, Portugal, Republic of Korea, Slovakia, Slovenia, Spain, Sweden, Switzerland, United Kingdom, United States) along the period 1995-2010. The data are from the Penn World Table 8.1 (PWT hereafter)

and the World Bank (WB, hereafter).[14] The variables taken into account are real income, physical capital, population, education, public expenditure on health and life expectancy at birth. We measure the population growth rate as the average rate of growth of the working-age population, where working age is defined as 15 to 65. We measure s_k as the average share of real investment (including government investment) in real GDP, and we use the human capital index provided by PWT and the life expectancy at birth provided by the World Bank as proxies of s_e and s_h, respectively.[15] For simplicity, we assume $d = \delta$, *i.e.* human and physical capital have the same depreciation rate. Summary statistics are provided in Table xx.1, in which all variables are measured in logarithms.

Table xx.1 - Summary statistics

Variable	Source	Obs.	Mean	Std. Dev.	Min	Max
Life expectancy	WB	620	78.53	2.80	67.54	83.59
HC index	PWT	620	3.23	0.32	2.07	3.73
Capital share	PWT	620	0.26	0.05	0.14	0.56
(n +g + d)	PWT	620	0.57	0.01	0.17	0.32
Public expenditure on health (% GDP)	WB	589	6.28	1.44	1.38	10.05
Real *per capita* GDP	PWT	620	34497	12412	9221	95176

ECONOMETRIC STRATEGY. In order to deal with the reverse causation between the level of real *per capita* GDP and country health status (see Weil, 2014; Tamakoshi and Hamori, 2015; and Linden and Ray, 2017), we apply a Bivariate Finite Mixture Model (BFMM, hereafter; see Alfò and Trovato, 2004; Alfò *et al*, 2008; Lu *et al.*, 2016; Ng and Mclachlan, 2014; Yu *et al.*, 2014). The advantage of this approach is that it allows to consider, as source of unobserved heterogeneity, the endogeneity between *per capita* income and health status. Following Linden and Ray (2017), therefore, we assume that real GDP levels and life expectancy are jointly correlated in some time points.

The BFMM allows to deal with a complex phenomenon which can be potentially characterized by non-trivial correlation structure. For instance, omitted covariates may affect *both* real GDP and aggregate health. It is well known that when responses are correlated (in our case, real GDP level and life expectancy), the univariate approach is less efficient than the multivariate one.[16]

Recalling equation (XX.15), to check if the empirical model is affected by endogeneity between $ln(y)_{it}$ and $ln(h)_{it}$,we estimate the following two equations:

$$E(\ln(y)_{it}|\ln(h)_{it}, \epsilon_{it}) = \alpha + \beta_1 \ln(h)_{it} \quad \text{(xx.16)}$$

$$E(\ln(h)_{it}|\ln(y)_{it}, \vartheta_{it}) = \delta + \beta_2 \ln(y)_{it} \quad \text{(xx.17)}$$

In equation (xx.16) we have a reverse regression in which life expectancy is the response and the level of *per capita* GDP is the covariate.

Table xx.2 Real *per capita* GDP and Health

	β	Std. Err.	z	P>|z|	[95% Conf. Interval]	
Real per capita GDP						
Health	1.236	0.019	64.330	0.000	1.199	1.274
Constant	6.468	0.078	82.800	0.000	6.315	6.621
Health						
Real GDP	0.646	0.010	64.960	0.000	0.627	0.666
Constant	-3.648	0.117	-31.120	0.000	-3.878	-3.419

Both the estimated parameters β_1 and β_2 are positive and significant ($\widehat{\beta_1} = 1.236$ with s.e.= 0.019 and $\widehat{\beta_2} = 0.646$ with s.e.= 0.009, see Table xx.2).

In order to consider the process under investigation based on a multivariate joint density, we model *per capita* GDP level and life expectancy y_{1it} and y_{2it} at time $t=1,..,T$ as a bivariate process. Vectors of outcome-specific p_{jt} covariates have been recorded for each country and will be denoted by $\mathbf{x}_{it}$ and $\mathbf{z}_{it}$. We consider the case where covariates differ across outcomes and are, respectively, $\mathbf{x}_{it} = [ln(s_k)_{i,t}, ln(s_e)_{i,t}, ln(n+g+d)_{i,t}, ln(s_h)_{i,(t-1)}]$ and $\mathbf{z}_{it} = [ln(y)_{i,(t-1)}]$.[17] Following the usual notation, for multivariate data, let $\mathbf{y}_i = (\mathbf{y}_{i1}, \mathbf{y}_{i2})$ denote respectively the vector of observed *per capita* level of GDP and measure of health (*i.e.* life expectancy) for the *i-th* country, $i=1,...,n$ in the analyzed time-window.

We can now write the empirical counterpart of equation (XX.5) as:

$$E(y_i|X_i, Z_i, \vartheta_{it}) = \begin{cases} E(y_1|x_i, \epsilon_{it}) = \alpha_i + \beta_1 \ln(se)_{i,t} + \beta_2 \ln(sk)_{i,t} + \beta_3 \ln(h)_{it} + \beta_4 \ln(ngd)_{it} \\ E(y_2|z_i, \vartheta_{it}) = \delta_i + \ln(y)_{i,t-1} \end{cases}$$

(xx.18)

According to the previous assumptions, we estimate a linear model for the response variables $y_i = (y_{t1}, y_{t2})$, in which some covariates are missing, collinear or describe a non-linear relationship with GDP levels (see *e.g.* Aitkin *et al.*, 2005).

Notice that the intercept terms vary across countries in order to capture country-specific features, *i.e.* $\alpha_i = \ln(A_0 + gt) + u_{1i}$ and $\delta_i = \delta + u_{2i}$ are random intercepts in which u_{1i} and u_{2i} are specific random terms that follow any *a priori* distribution. The set of subjects and the outcome-specific random coefficients appear additively in the linear predictor. This assumption, however, can be relaxed by associating random parameters to some elements of the covariatesô set, generalizing our specification to a random coefficient model. In other terms, the values $u_{ij} \in U$, with $i=1,...,n$, for response $J=1,2$, represent individual-specific features varying over the data set in an unknown way. According to Kiefer and Wolfowitz (1956), they can be treated as drawn from N i.i.d. random variables u_i with a common unknown density function $g(\cdot)$. Given the assumption of conditionally independence and treating the $u_i$ôs as nuisance parameters and integrating them out, the corresponding likelihood function can be rewritten as follows:

$$L(\cdot) = \prod_{i=1}^{n} \int_U f_i(y_i|x_i, z_i, u_i) = \prod_{i=1}^{n} \int_U \left[\prod_j \prod_{t=1}^{T} y_{ijt}, x_{it}, z_{it}, u_{ij} \right] dG(u_i)$$

(xx.19)

where u represents the support for $G(\boldsymbol{u})$, the distribution function of u_i. Model parameters can be estimated through the above marginal likelihood. In this context, the random component represents mean zero deviations from the fixed part, *i.e.* the country-specific latent effects u_i capture the country variability in the dynamic process of the *technological* factor. Various alternative parametric specifications may be proposed for modeling random effect distribution. However, parametric specifications of the mixing distribution can be restrictive and are generally unverifiable.

As proved by Lindsay (1983a; 1983b), the maximum likelihood estimator is concentrated on a support of cardinality at most equal to the number of distinct points in the analyzed sample. Therefore, the integral of the likelihood may be approximated by a sum on a finite number *K* of locations:

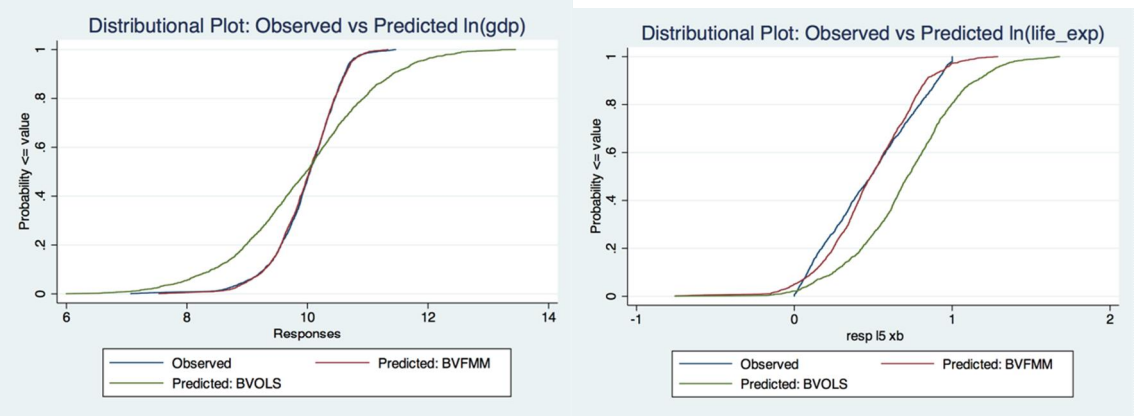
$$L(\cdot) = \prod_{i=1}^{n}\left[\sum_{k=1}^{K} f_i(y_i|x_i, z_i, u_k)\,\pi_k\right] = \prod_{i=1}^{n}\sum_{k=1}^{K} f_{ik}\,\pi_k \qquad \text{(xx.20)}$$

where f_{ik} is the response density distribution for the *k-th* component of the BFMM, where the intercept terms vary across countries in order to capture country-specific features. Finally we assume that unobserved heterogeneity affects outcomes in correlated ways, *i.e.* the latent effects in the two regression equations are correlated.

RESULTS. Table xx.3 reports the estimates for the OLS fixed effects model, for the *restricted* Solow model and for three alternative specifications of the BFMM. The results strongly support our *augmented* version of Solow model, with education and health. Equation xx.15 shows that the *augmented* model predicts that the coefficients of s_k, s_e, s_h and $(n + g + d)$ sum to zero. The (implicit) estimated values for α, β and γ show that this restriction is not rejected by the data.

All three bivariate finite mixture models show a better global fit in comparison with the OLS model. Figure xx.1 overlays the empirical density function for real *per capita* GDP (left panel) and for life expectancy (right panel), obtained via BFMM(1) (red line) and OLS model (green line), to those corresponding to observed data (blue line).

Figure xx.1 - Empirical density functions



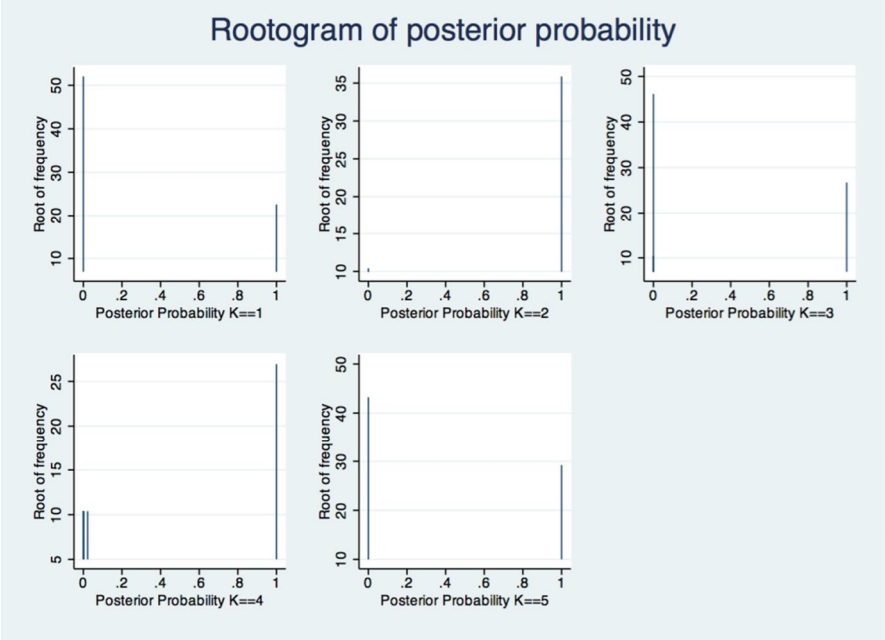
The estimates are all significant: the coefficients on s_k, *i.e.* the capital share, and on the sum of the rates of change in population and in technological progress plus depreciation $(n + g + d)$ are in line with the literature while the elasticity of the *per capita* GDP to life expectancy is 0.399. The bottom half of Table xx.2 reports the estimates for the health equation: life expectancy is positively and significantly associated to an increase in the real *per capita* GDP, with an elasticity of 0.607. A positive impact of the population growth rate (0.407) and the lagged value of life expectancy (0.543) are found in the BFMM(2), in which also the public expenditure on health is slightly positively associated (0.023) to the aggregate level of health of a country. The BFMM(3), which has a richer specification of the equation for life expectancy, presents similar estimates but also an unpleasant positive parameter of the depreciation term ($n+g+d$).

A useful by-product of the BFMM approach is that it allows clustering of countries on the basis of the posterior probabilities estimates $\hat{u}_{ij}$. Notice that each component is characterized by homogeneous values of estimated latent effects, *i.e.* conditionally on the observed covariates, countries belonging to the same cluster show a similar *structure*, at least in the long-run. The latent variables, therefore, may capture the effect of missing covariates, *i.e.* those factors not included in the *augmented* Solow model. Using AIC, CAIC and BIC criteria, BFMM(1) identifies 5 clusters of

countries while 6 clusters are obtained via BFMM(2) and BFMM(3). Clustersô composition does not change significantly moving across different specifications. In the following, for the sake of brevity, we focus on the BFMM(1).[18]

Estimated locations are shown in Table xx.4, while corresponding clusters are reported in Table xx.5. Finally, Figure xx.2 provides the rootogram of the posterior probability, which shows that the mixture components are well separated one from each other, *i.e.* no significant mass can be found in the middle of the unit interval (no overlapping components).

Figure xx.2 - Rootogram



Looking at the first column of Table xx.4, we see that the random terms positively affect the level of the real *per capita* GDP of the countries belonging to clusters 2 (0.055) and 5 (0.158), which are also those with the higher average level of *per capita* GDP, 10.11 and 10.43, respectively. Symmetrically, the same partition emerges when we move to the third column of the Table. In fact, the model suggests that in clusters 2 and 4 can be found some unobserved factors, not directly captured by the model, that are harmful for health, reducing life expectancy. The opposite happens for countries grouped in the remaining clusters.

Table xx.3 - Estimates

	OLS FE	Restricted model	BFMM (1)	BFMM (2)	BFMM (3)
Real per capita GDP					
s_e	0.675***	0.399***	0.177***	0.358***	0.120***
s_k	0.409***	0.318***	0.145***	0.197***	0.128***
n+g+d	-0.556***		-0.076***	-0.252***	0.205***
s_h	0.802***	0.943***	0.399***	0.458***	0.447***
Constant	8.162***	4.955***	3.625***	4.648***	6.499***
$\hat{\alpha}$		0.120***			
$\hat{\beta}$		0.150***			
$\hat{\gamma}$		0.354***			
Life expectancy					
Lagged Real *per capita* GDP			0.607***	0.447***	0.407***
Public exp. on health					0.023**
Population growth				3.400***	
Lagged life expectancy				0.362***	0.543***
Constant			-5.542***	-4.142***	-3.897***
σ^2			0.014	0.011	0.013
σ^2_{GDP}			0.015	0.011	0.016
$\sigma^2_{GDP,\ health}$			0.049	0.027	0.051
$\sigma_{GDP,\ health}$			-0.028	-0.029	-0.029
k			5.000	6	6.000
Observations	620	620	3059	3059	1240

Table xx.4 - BFMM(1), locations and probabilities

Cluster	Real GDP		Life expectancy		Prob.
k	*loc.*	*s.e.*	*loc.*	*s.e.*	
1	-0.276	0.024	0.498	0.041	0.097
2	0.055	0.022	-0.107	0.040	0.322
3	-0.104	0.023	0.187	0.040	0.194
4	-0.008	0.023	0.019	0.040	0.194
5	0.158	0.024	-0.277	0.030	0.195

Note: *k*, number of mixture components selected by penalized criteria; *loc.*, locations; *s.e.*, locationsô standard errors. Prob., prior probability of belonging to that local area. The probabilities are for both equations in the bivariate model.

Table xx.5 ï Clusters

	Country	Real GDP	Life expectancy
Cluster 1			
	Poland	9.25	72.61
	Portugal	9.47	72.72
	Republic of Korea	9.03	69.49
	mean	*9.25*	*71.61*
Cluster 2			
	Australia	10.18	76.16
	Austria	10.04	74.85
	Belgium	10.09	75.18
	Canada	10.22	76.55
	France	10.06	76.03
	Germany	10.06	74.79
	Iceland	10.23	77.75
	New Zealand	9.97	75.40
	Sweden	10.18	77.42
	United Kingdom	10.05	75.49
	mean	*10.11*	*75.96*
Cluster 3			
	Estonia	9.64	71.78
	Greece	9.67	75.35
	Hungary	9.48	70.89
	Japan	9.94	77.52
	Slovakia	9.79	73.77
	Spain	9.74	76.35
	mean	*9.71*	*74.28*
Cluster 4			
	Czech Republic	10.06	75.40
	Finland	10.01	74.69
	Ireland	9.81	74.69
	Israel	9.98	76.34
	Italy	9.95	76.18
	Slovenia	10.07	76.68
	mean	*9.98*	*75.66*
Cluster 5			
	Denmark	10.17	75.34
	Luxembourg	10.63	74.57
	Netherlands	10.17	76.70
	Norway	10.24	76.91
	Switzerland	10.45	77.14
	United States	*10.43*	*74.54*

mean	10.35	75.87

4. DISCUSSION

Our estimates show that, at least for the sample of OECD countries, population's health positively and significantly affects the level of *per capita* income. This finding is consistent with our theoretical model, in which the typical capital "dilution effect", due to the increase in population induced by a better aggregate health, is offset by the increase in productivity arising from healthier workers.

Apparently, this result is in contrast with that of Acemoglu and Johnson (2007). We deem that this discrepancy is due to the following reasons. Differently to them, we have restricted our attention on a sample of high income countries (for which they find not significant estimates) and we have followed an alternative econometric route to deal with the potential endogeneity between life expectancy and *per capita* income.

Our evidence is in line with Cervellati and Sunde (2011); by moving from a radically different perspective, they also find that the effect of life expectancy on income *per capita* is positive for the high income countries (see Table 10 in their Online Appendix). Their argument, however, relies on demographic issues, which we do not directly tackle in our econometric exercise. The inclusion of the population growth rate and/or the lagged value of life expectancy in our equation for aggregate health can be seen as an attempt to include these demographic forces into our model.

Our classification shows that for the group of countries with the highest average income, namely Denmark, Luxembourg, Netherlands, Norway, Switzerland and United States, the random component strongly affects the level of *per capita* GDP, while the effect is strongly negative for the group with the lowest average income. This difference can be explained as the consequence of the gap in terms of aggregate efficiency, which is actually due to differences in institutional factors. To

this extent, along with Mankiw *et al.* (1992) we may argue that fiscal, education and innovation policies together with political stability will end up among the ultimate determinants of cross-country differences in income. Richest countries are also characterized by having unobservable factors that are harmful for the level of population's health. The strength of these factors, however, declines when we move from BFMM(1) towards the less parsimonious specifications of the equation for aggregate health, provided by BFMM(2) and BFMM(3).

5. CONCLUDING REMARKS

Following Mankiw *et al*. (1992) we have argued that international differences in income *per capita* are best understood using an *augmented* Solow growth model. In particular, we build a model in which output is produced from capital, raw labor, human capital in the form of education, and human capital in the form of health. One of the predictions of this simple model is that the long-run level of *per capita* real GDP of a country is positively affected by the level of health of its population. We test this prediction by using data from the sample of OECD countries, along the period 1995-2010. As it is standard in this literature, we use life expectancy at birth as a proxy for population health. To deal with the endogeneity problem between health and income we estimate a Bivariate Finite Mixture Model. The empirical analysis corroborates our theoretical finding. Interestingly, our semi-parametric approach allows countries classification. Despite public expenditure on health positively affects *per capita* income, through its effect on life expectancy, cluster membership does not change significantly when this kind of public intervention is included as a covariate in the equation for life expectancy. Our estimates also indicate that the richer is the country, the stronger is the role of unobservable factors in explaining the level of *per capita* income. Interestingly, the richest countries in the sample are also characterized by having unobservable factors that are harmful for the level of population's health.

The Bivariate Finite Mixture approach is able to measure local variation in the observed data.

Therefore, it makes the *augmented* Solow model with human capital accumulation via education and health – conditionally on heterogeneous groups – a useful tool to understand the differences among countries in long-run *per capita* income.

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[1] A more exhaustive survey of recent literature on the relationship between health and economic growth can be found in Weil (2014).

[2] As an example, using GMM-based panel data methods, Suhrcke and Urban (2010) find a causal negative effect of cardiovascular mortality on subsequent economic growth in high-income countries for the time span 1960-2000. Hyclak *et al.* (2016) show that across the OECD countries, most of the correlation between cardiovascular mortality and income per capita arises from variation within the Eastern European countries.

[3] ***ñé*** *We do not observe human capital in the form of health directly, but presumably it is some combination of ability to work hard, cognitive function, and possibly other aspects of health****é ò*** (Weil, 2007, p. 1268).

[4] Hartwig (2010, Table 1, p. 315) reports the results of fourteen studies that analyze the relation between health and macroeconomic growth, using different measures of health.

[5] In this chapter we are interested to the channel that goes from health to income. Leading proponents of the view that there is a large structural effect of health on income are also Sachs (2001) and Fogel (1997). In particular, Fogel (1997), examining the historical evolution of body size and calorie consumption in the United Kingdom, concludes that over the period 1780ï 1980 better nutrition raised labor input per work-aged adult by a factor of 1.96.

[6] Aghion *et al.* (2011) document that growth of life expectancy is strongly negatively correlated with initial life expectancy across countries over both the 1940-1980 and 1960-2000 periods. In other words, there has been a massive process of world-wide convergence in life expectancy in the last few decades (see also Becker *et al.*, 2005). Indeed, it is intuitive that in countries where life expectancy is initially high (due to already well-developed, highly efficient, and well-equipped health-care systems), further improvements in populationôs health conditions can only be achieved at extremely large health-investment costs. Moreover, if such costs are so big to overwhelm the potential economic benefits related to further health improvements, then the ultimate consequence of the amelioration of a population health conditions can definitely be a worsening of the general economic performance (*i.e.*, the GDP growth rate of a country). This is consistent with Bharghava *et al.* (2001)ôs finding that the effect of health on the GDP growth rate is larger in developing countries than in developed countries. For example, they estimate that for the poorest countries a 1% change in the adult survival rate is associated with an approximate 0.05% increase in the economic growth rate. The parameter estimates imply large positive effects of the adult survival rate on economic growth for countries such as Burkina Faso, Burundi, the Central African Republic, India, Ivory Coast, and Nigeria. For highly developed countries, such as USA, France and Switzerland, the estimated effect of adult survival rate on economic growth is, instead, found to be negative.

[7] Consistently with Lorentzen *et al.* (2008) ï who use exogenous variation across countries, such as climatic factors, geographical features, or disease indices, as instruments for differences in life expectancy ï , Aghion *et al.* (2011) find that the initial level of life expectancy has a positive impact on the average rates of investment in physical and human capital, while both the growth and the initial level of life expectancy help reducing fertility. Hence, health variables are an important determinant of economic growth. Doppelhofer *et al.* (2004) use Bayesian averaging models techniques to show that the initial level of life expectancy is also one of the most robust causes of economic growth.

[8] Other empirical results, however, contradict such finding. Controlling for initial health, Hansen (2014) finds no significant effect of the change (or level) of longevity on GDP per capita among U.S. states. Hansen and Lönstrup (2015) show that, when implementing a three-point panel (with international data from 1900, 1940, and 1980) and controlling for initial health and country fixed effects, increased longevity appears to play a negative role on GDP per capita.

[9] Concerning the relation among life expectancy, mortality, fertility, population growth, and ultimately income, papers that find a positive impact of life expectancy on income (abstracting, however, from possibly-related changes in fertility) include de la Croix and Licandro (1999), Kalemli-Ozcan *et al.* (2000), Blackburn and Cipriani (2002), Boucekkine *et al.* (2002 and 2003), Lagerlöf (2003), and Bar and Leukhina (2010b), among many others. Galor and Moav (2002) study the role of survival and natural selection for the so-called fertility transition. Papers in which greater life expectancy causally implies a fall in fertility include, instead, Kalemli-Ozcan (2002 and 2003), Boldrin and Jones (2002), Soares (2005), Strulik (2008), and Bar and Leukhina (2010a). Whether reductions in mortality may cause fertility reductions is still a highly debated issue in the empirical literature.

[10] See also Buiter (1988).

[11] See, among others, Bloom and Williamson, 1998; Bloom, Canning, and Sevilla, 2003; Mason *et al.*, 2016; Bloom, Kuhn, and Prettner, 2017.

[12] See also Cervellati and Sunde (2015).

[13] See, as a notable example, Mankiw *et al.* (1992, Eq. 8, p. 416).

[14] For more information on the PWT see: http://www.rug.nl/ggdc/productivity/pwt/.

[15] A lot of empirical literature uses, as a proxy for health-status, the health expenditure (for example Hartwig, 2010 and Tamagoshi and Hamorri, 2015); others measure it using life expectancy (Linden and Ray, 2017). In our estimates, we use the public expenditure on health and the population growth rate as regressors in the equation for our measure of health, *i.e.* life expectancy.

[16] See Zellner, 1962 and Davidson and Mackinnon, 1993 for a detailed discussion of this topic in the SUR context.

[17] We also run two alternative specifications of the Bivariate Finite Mixture model: BFMM(2), in which $\mathbf{z}_{it} = [ln(y)_{i,(t-1)}, life\ expectancy_{i,t-1}, n_t]$, and BFMM(3) in which $\mathbf{z}_{it} = [ln(y)_{i,(t-1)}, life\ expectancy_{i,t-1}, ln(public\ exp.\ on\ health)_{i,t}]$.

[18] Details on BFMM(2) and BFMM(3) not presented in the chapter are available upon request.