DOES EARLY-LIFE HEALTH ENHANCE GROWTH? EVIDENCE FROM SPAIN

Área Temática: *11. Sector Público, financiación autonómica y local*

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Abstract

In light of recent literature, this paper analyses the hypothesis of causal effect of early-life health on economic growth for the Spanish regions over the period 1980-2007. We offer a unified framework that encompasses the growth effects of both infant mortality and the channels through which growth is affected. We provide empirical evidence that higher infant mortality has a direct negative impact on per capita income growth. Also, that a greater risk of early-life death is associated with losses on accumulation of both physical and human capital, and fertility gains, which in turn more even reduces growth. Overall, our findings do support the importance to broaden the concept of human capital by including health.

Keywords: Growth, Infant Mortality, Investment, Human Capital, Fertility.
JEL Classifications: I10; J10; O10.

1 Introduction

Since Grossman (1972), there has been a huge literature on health as a form of capital stock in complex ways. So, at present the role of health capital in economic growth is one of the best-known relations in international development (Bloom and Canning 2000). This correlation has been traditionally seen as a causal link in only one direction: wealth allows better access to food, investments in health care or education (Pritchett and Summers 1996). In this regard, Preston (1976) emphasized that economic growth is the most important determinant of life expectancy. However, in recent years a sizeable body of research has addressed the reverse causation, i.e. healthy populations increase labour productivity and per capita income. The World Health Organization Commission on Macroeconomics and Health (2001) indicated that there are several ways that disease impedes economic well-being and development. In this context, economic literature has developed models where health is incorporated in traditional growth ones (Howit 2005; Van Zon and Muysken 2005; Weil 2007 or Aghion et al. 2011).

The aim of this paper is to analyse the empirical relationship between early-life health and economic growth across Spanish regions over the period of 1980 to 2007. According to OECD Health Statistics (2014), the index of per capita income for Spain rose from 74.1 in 1980 to 83.7 in 2007 (based on PPS EU-15=100). Meanwhile, the index for infant mortality rate fell from 100.2 to 97.5. It is worth noting that the negative correlation between those variables could be modified in recent years because of the Great Recession started in 2008 (Svensson and Krüger 2012). Thus, the rates of income and mortality in 2012 were 77.7 and 96.3, respectively. These data widely justify addressing the relationship between early-life health and economic growth. Moreover, there is little research on the health-related growth in countries of southern Europe, let alone on regions which exhibit less extraneous variation than those.

This paper contributes to the recent literature concerning health capital as a determinant of economic growth. There are at least two causal links running from health to economic growth: one direct, the other indirect. The former is related to the idea that healthier populations tend to have higher labour productivity and per capita income.

The latter states that healthier people who live longer have stronger incentives to invest in developing skills, generate more investment in physical capital, and trigger subsequent declines in fertility. All these mechanisms can lead to increase per capita income (Bloom and Canning 2000).

Several features distinguish this study from its predecessors. Initially, as far as we are concerned it is among the first to disentangle the health-related growth for the Spanish regions, when using infant mortality as a proxy for health status. Qualitatively, choosing infant mortality rather than life expectancy for health does not seem to make a big difference. As said by Annoni and Dijkstra (2013), health is one of the basic pillars of competitiveness that is captured through six indicators. Infant mortality correlates to 5% with road fatalities, healthy life expectancy, cancer disease death rate and heart disease, and to 10% with suicide. In doing so, we transmit a distinction on previous contributions. Then, it supposes a different point of view from Rivera and Currais (2004) that analysed how the composition of public health spending affects productivity. Also, it departs from Oliva-Moreno (2012) that studied labour productivity losses associated with illnesses.

Besides, this paper uses an innovative method for testing of on hypotheses derived from economic theory viz., whether health improves growth. This approach was introduced by Lorentzen, McMillan and Wacziarg (2008, hence LMW) to focus on channels through growth is affected by health, i.e. investment, education and fertility. It encompasses both direct and indirect effects of health on growth. Hence, we first estimate direct effects of infant mortality on per capita income growth through both linear and dynamic panel models. We then use a structural system to also capture likely indirect effects. There is a growing consensus in the literature on indirect large effects that improving health has on accelerating economic growth.

Lastly, from an economic policy perspective this paper encourages debates about the implications of government's involvement for the provision of health ─namely the cost and benefits of health care programs, and the sustainability of national health care systems.

According to OECD (2014), Spain's child poverty has risen to 21% against an EU average of 13% in the years of financial crisis and downturn. Besides, health care powers have been decentralized to the regions, since the early eighties until 2002, following an asymmetric process. Definitely, it enlarges the consciousness on the determinants of economic growth, which is crucial to policymakers when designing public policies.

The remainder of this paper is organized as follows. Section 2 shows the related literature on health-related growth. Section 3 describes the model to be estimated, data variables and empirical strategy. Section 4 presents the results. Finally, Section 5 concludes and points out the main policy implications.

2 Literature review

With respect to the empirical evidence on health-related growth, the focus of researchers has shifted from the exploration of direct effects to the indirect ones. To begin with the direct growth-effects of health, Bloom and Canning (2005) observed that a 1% increase in adult survival rates increases labour productivity by about 2.8%. In this regard, Aghion et al. (2011) found that only the reduction in mortality rates below forty generates productivity gains in OECD countries. Meanwhile, Acemoglu and Johnson (2007) studied the effect of life expectancy on economic performance, using a model based on a predicted mortality instrument. Unlike previous papers, they found that there is no evidence that the large increase in life expectancy raised per capita income. In spite of, Bloom et al. (2013) revisited Acemoglu and Johnson (2007) and showed that their main result is mostly driven by a priori exclusion of initial life expectancy. Additionally, Cervellati and Sunde (2011) suggested that life expectancy may have direct effects on economic growth. These appear to be non-monotonic and depend on the level of demographic development. Finally, French (2012) positively tested for some OECD countries that better health improves income while the latter in turn also affects health.

On the other hand, Mayer (2001) studied the long-term impact of health on economic growth in Latin America.

He found that a permanent increment of 0.8-1.5% of annual income is associated with adult and, unexpectedly, old aged health improvements. Moreover, he points out that the channels of causation from health to income are diverse, and some of them may be indirect. In the same line, LMW explored three channels whereby adult mortality may affect growth. They found that a greater risk of death during the prime productive years is associated with higher levels of risky behaviour, higher fertility, and lower investment in physical capital. Instead, Cooray (2013) supported that health capital does not have a robust and significant effect on economic growth, unless through their interactions with health expenditure and education. At this regard, Kumar and Chen (2013) argued that health and education contributed on the growth rate of total factor productivity. They pointed out the importance of including health capital on the policies design, which contributes to technology diffusion.

3 Model and data

In this section we briefly review the conceptual framework, data, and strategy employed in this paper. Different empirical contributions of the literature, focussing on the causality between the level of health and economic growth, provide a motivation for the model discussed below.

3.1 Theoretical framework

There is a large body of research devoted to health economics, which supports the premise that health status is a determinant for economic growth i.e. Howit (2005) or Van Zon and Muysken (2005). The testable hypothesis is that mortality affects economic growth by diminishing incentives for behaviour with short-run costs and long-run payoffs. The main assumptions of the problem to be studied can be illustrated within the framework of Acemoglu and Johnson (2007).

We consider a closed-economy in continuous time *t*, where a unique consumption good (Y) is produced with a constant returns to scale aggregate production function. Generalising, the model structure takes the following form

$$Y_{it} = (A_{it}H_{it})^{\alpha} K_{it}^{1-\alpha} , \qquad (1)$$

where 0<α< 1; *i* and *t* index region and time respectively; *A* is the level of technology; *K* denotes physical capital; and *H* is the aggregate human capital given by $H = h\,N$, where h is human capital per person and *N* is the total population. Substituting into Eq. (1) and taking logs, the level of log per capita income can be written as

$$\log \mathrm{y_{it}} = \alpha \log \mathrm{A_{it}} + \alpha \log \mathrm{h_{it}} - (1-\alpha) \log \mathrm{N_{it}} + (1-\alpha) \log \mathrm{K_{it}} , \qquad (2)$$

where *y* = *Y/N*.

Eq. (2) shows that per capita income is driven positively by technology, human capital and capital stock, and negatively by population. We now briefly discuss each element on the right hand side, assuming a steady state as an approximation. First, we follow the method introduced by Bils and Klenow (2000) that build on the earlier work of Mincer (1974) and the large literature on schooling and wages. The individual human-capital *h* is proportional to exp(l_h), where l_h is the average number of years of educational attainment. Second, we can write the population level *N* by equating the flow of deaths with the flow of total births. While the former depends on the mortality rate, the latter is related to fertility. Lower mortality increases population size since more people survive at each point in time. Also, greater longevity may lead to changes in the decisions of parents to their children (planned fertility) (Kalemli-Ozcan 2008). Third, to arrive to a measure of *K* we assume the stock of capital at any moment would be determined by the Solow model. It can be shown that the effect of changes of capital stock would then appear in terms of investment rate. Fourth, let $A_{it} = A_i$, which is consistent with some baseline differences in technical progress across regions.

Therefore, subtracting the lagged dependent variable from both sides of Eq. (2) yields the reduced-form growth model

$$\Delta \log y_{it} = \eta_i + \log y_{it-1} + log\ imr_{it} + log\ X_{it} + Z_{it} + \varepsilon_{it}, \qquad (3)$$

where η denotes unobserved time-invariant region-specific effects, *X* stands for channel variables (investment, school and fertility), and *Z* is a collection of control variables.

In Eq. (3) the dependent variable is the growth rate of per capita income and the variable of interest is health status, which we proxy for infant mortality rate, *imr*. The lagged log of per capita income captures the hypothesis of convergence.

Next, we turn to specify the channel equations as:

$$log\, X_{it} = \eta_i + \log y_{it-1} + log\, imr_{it} + Z_{it} + \xi_{it} \,. \tag{4}$$

Last, according to LMW we specify a system of structural equations from Eq. (3)–(4), making explicit the causal relations between economic growth, the channels linking it to infant mortality, and the mortality variable as:

$$\Delta \log y_{it} = \eta_i + \log X_{it} + \mu_{it} \tag{5}$$

and

$$\log X_{it} = \eta_i + \log imr_{it} + \upsilon_{it} \,. \tag{6}$$

Then, this structural-form model contains two main equations, i.e.: an income growth specification derived from an augmented Solow model (Eq. (5)), and a channel specification based on the health economics literature (Eq. (6)).

3.2 Data and variables

This paper tested health-related growth with panel data for the Spanish regions. The level of disaggregation corresponds to NUTS2 in the Eurostat nomenclature of territorial units. We exploit databases that are widely used to investigate regional and health economics issues in Spain. In particular, we focus on the regional database BD.MORES which collects data from 1980 to 2007. All monetary data are quoted at 2000 constant prices. Output is measured as gross value added. We exclude the real estate sector, which includes imputed rents of owner-occupied properties, because of its volatility (Tortosa and Peiró 2012). The housing bubble, which burst around 2008, started in the early nineties in Spain. So, much of the sample period was affected by this event. The list of main variables used in our econometric analysis includes investment share, education attainments, and fertility and infant mortality rates. Regarding control variables we use health power. The reason for including this variable is based on the fact that in our country since 1978 a centralized state has turned into a decentralized one, associated with the devolution of power to the regions.

So, the coexistence of several models concerning the degree of health power makes Spain a singular case and it justifies to employ health power as control variable. Table 1 provides details concerning the definitions and sources of the variables, and Table 2 summarizes the descriptive statistics.

('insert table 1 here')

('insert table 2 here')

Over the sample period, the Spanish National Health Service is characterized by two main features. First, universal access to health care for all Spanish citizens, and second, a fast asymmetric decentralization of health care to regions. Related to health care powers, the process began in 1981 and ended in 2002, according to three models distinguished among the 17 regions:

(i) Two "foral" regions (Basque Country (1987) and Navarre (1991)) that were both fiscally and politically accountable for the running of almost all public service provision within their boundaries. While they were granted autonomy in financing health care, they also enjoyed a high level of tax autonomy.

(ii) Five regions (Catalonia (1981), Andalusia (1984), Valencian Community (1987), Galicia (1991), and Canary Islands (1994)) kept health powers, but with fiscal responsibility limited. They were held politically more than fiscally accountable. Most resources devoted to health care in those regions came from specific grants, with self-financing strongly constrained and playing a minor role.

(iii) The remaining regions (Aragon, Asturias, Balearic Islands, Cantabria, Castile-La Mancha, Castile and Leon, Madrid, Extremadura, La Rioja, Murcia) had no health powers until 2002. So, the central government carried out all responsibility for health care there until this date.

The process of health care decentralization has then extended to all regions. The new effective system departs from the previous model of specific health care financing, by integrating it into the general one. Nowadays, health care financing is covered through regional taxes, shared taxes and block-grants from the central government, and pharmaceutical co-payments.

3.3 Empirical strategy

We first evaluate the direct impact of infant mortality on economic growth. In order to estimate Eq. (3) we start by applying linear panel data models. Though, it is well known in studying growth regressions that problems of endogeneity and reverse causality arise. We then use the generalized method of moment estimator (GMM). Besides, as Bond et al. (2001) suggest that more plausible results can be achieved using the system-GMM estimator developed by Arellano and Bover (1995) and Blundell and Bond (1998), it is also applied.

Next, we estimate Eq. (4) by applying instrumental variable (IV) estimators (Schaffer 2010). We then examine infant mortality related to investment share, education attainments, and fertility rate with two goals. On one hand, these relationships are interesting in their own right as evidence for the horizon effect of mortality. On the other hand, it is a first step toward the latter decomposition of the total effect of infant mortality on income growth through the channels. Last, we focus on the different channels through growth is affected by health, for which we specified the structural model Eq. (5)−(6). Econometric methodology relies on Three-Stage Least Squares estimation (3SLS), where dependent variables are explicitly taken to be endogenous to the system and are treated as correlated with the disturbances in the system's equations[1]. Summing up, the effect of each channel on income growth multiplied by the effect of infant mortality on this channel would provide the indirect effect of health on growth.

The remaining issue to discuss is how we obtain valid instruments for mortality and the channels. In some referred studies for countries, climatic factors and geographic characteristics are used. However, for regions within a country such differences might not be sensitive. So, we instrument for lagged variables of both infant mortality and the channels whenever they appear on the right-hand-side of equations[2]. These are obviously not exogenous, but with regard to the current values of the endogenous variables, they may be regarded as having already been determined.

[1] As infant mortality is treated here as an endogenous regressor, we relate it to its lagged values used as instruments.
[2] For the sake, LMW obtained broadly consistent results when instruments for geography characteristics or lagged variables were used.

The deciding factor is whether or not they have uncorrelated with the current disturbances, which we may assume (Greene 2012). We also examine the issue of the validity of instruments through tests of overidentifying restrictions.

4 Results

In this section we apply the estimation procedure described above to the model. Our agenda is twofold. We first estimate models that account for the mortality status of Spanish regions over the period 1980-2007. Then, in line of the typical pattern of channel equations, we also check the relative importance of these channels through which growth is affected. As regard to identification, two points need to be made. On the one hand, it is important to emphasize that our paper does not quantify the reducing-income effect of poor health as distinct from the horizon effect of infant mortality. On the other hand, it should be noted that extended life may result in an increase in working years. Health improvements enhancing the quality of life may boost the per capita income each year of life (Mirvis and Bloom 2008).

4.1 Reduced-form regressions

Our econometric estimates for the baseline model (3) are reported in Table 3. The results for the linear panel levels, GMM and system-GMM estimators are qualitatively similar. On the whole, we notice that results support the hypothesis of the impact of the channels on per capita income growth. The coefficients on the investment share and education attainments have the expected positive sign and are strongly significant. So is the coefficient on lagged income, the negative sign is consistent with the hypothesis of convergence between regions.

If we believe the Sys-GMM estimates in column 5, for example, the convergence effect would imply a catch-up of about 4% between Madrid (top) and Extremadura (bottom)[3]. In addition, it is noteworthy the corresponding coefficients suggest a negative effect of infant mortality. According to our findings, a 1% increase in the infant mortality rate would reduce per capita income growth by around 2%. In accordance to our GMM results, it should reduce up to 1.3% growth rate of per capita income in the sample period[4]. Further, we tested for potential specification problems. On the one hand, the Hausman tests for unobservable heterogeneity lead to reject in column 2 the null hypothesis that differences in linear panel estimates are not systematic. Thus, we used the fixed effects instead of random effects estimator. On the other hand, the Sargan tests of over-identifying restrictions do not detect any problems with the validity of the instruments.

('insert table 3 here')

The next step has been to estimate Eq. (4) that allows for the impact of health on the channels of growth. The results are displayed in Table 4. To address endogeneity issues, mortality, investment, school, and fertility are instrumented by their lagged variable. In all regressions we failed to reject the null hypothesis of validity of the instruments. Overall, the estimated relations between health and the three channels are consistent with the hypothesis stated above. Our estimates indicate a long-run relationship between those variables. The elasticity of investment and education to infant mortality is significant negative, while it is positive for fertility, as suggested by theory. Using estimates in the regression of column (2), a one standard deviation increase in infant mortality (equal to 0.39) is associated with 3.5 percentage points difference in investment. It is also worth mentioning that health power exerts a positive impact on the channels.

('insert table 4 here')

[3] With respectively 14,870 and 7,240 euros of per capita income (sample average) in Madrid and Extremadura, respectively, the convergence effect vehicles a catch-up of 0.05xlog (14,870 /7,240)) = 3.9 percentage points.

[4] Being infant mortality 2 per thousand (time average for La Rioja) instead of 1.14 per thousand (the one for Balearic Islands) implies a growth gap of -0.023 x log ((2/1.14)) = 1.3 percentage points.

4.2 Structural model regressions

As discussed in the previous section, we assume that infant mortality may impact investment in physical and human capital, as well as fertility, which in turn affect growth. We proceed with the econometric exercise by estimating the system of structural equations. Now let us briefly describe the corresponding estimation results. On the one hand, the estimated elasticities of income growth to the channels to some extent differ from those of reduced-form regressions (column 1 of Table 5 versus Table 3). On the other hand, the elasticities of the channels with respect to infant mortality are quite consistent with the ones previously found (column 2 of Table 5 versus Table 4). This again connects with a significant impact (negative for investment and school, and positive for fertility) from infant mortality. At this stage of the analysis, the overall impact on income growth from health can be straightforwardly computed. On the basis of the parameters reported in columns 1−2, our overall impact of health on growth through the three channels jumps to -0.10. We can now attempt to capture the order of magnitude of the indirect impact by comparing overall to direct elasticity. Taking into account that the point estimate of our baseline model equals to -0.02 the former is fivefold as large compared to the later.

('insert table 5 here')

Overall, our results support the hypothesis that early-life health can be seen as a robust determinant of the growth rate of per capita income. Our findings support both the effect of health on channels and the effect of these on growth matter for income growth. While the impact seems to occur directly, somewhat unsurprisingly it is likely to work more strongly through investment in physical and human capital, as well as fertility. It is also of interest to note that the accuracy of our estimates is very strong. All statistics are high, for instance *p*-values below 0.01, thereby indicating the robustness of our regressions.

5 Conclusions

Recent literature provides mixed answers regarding the effect of health on economic growth. In this paper we investigate these effects for the Spanish regions over the period 1980-2007. Our findings support those in the literature who found that worse health status, in the sense of higher early-life death, is growth-reducing. Along with Aghion et al. (2011) and Mayer (2001) we show the positive effect of health on accelerating economic growth. Besides, in line with Lorentzen, McMillan and Wacziarg (2008) we found there is supportive evidence of even larger indirect impacts driven through the growth channels.

Our results have a number of consequences from a policy economic perspective. On the one hand, early-life health would be related to the economy. As said by Muižnieks (2013), budgetary adjustments in the years of financial downturn have had a disproportionate impact on children's access to health care in Spain. As a result, one of the main objectives for policymakers should be clearly directed to improve population health, especially at an early age. This assumption is on primary importance in current debates on the cost and benefits of health care programs. On the other hand, being health one of the most sensitive indicators to reflect development, it is important to aware its significance in order to avoid social inequalities. In accordance to OECD (2014), Spain's poorest has been affected the most among OECD countries during the crisis years of 2008-2013. Indeed, children are highly sensitive to the quality of health and other social services provided to them.

Because health-related growth is an area in which greater effort must be made to generate empirical information for policymakers, this article could be extended. It would be valuable to test the results by using other health proxies than infant mortality, and also to focus on specific causes of death. These and other extensions of the analysis of this paper are left for further research, when there will be available new data on health indicators.

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TABLES

Table 1 Variables and data sources

Variable	Definition	Units	Data Source
y	Per capita income (per capita gross value added, GVA).	Thousands of euros (2000 constant prices)	Regional database of the Spanish economy (BD.MORES)
imr	Infant mortality rate.	Ratio. Deaths per thousand inhabitants 0-4 years	Spanish National Institute of Statistics (INE)
health power	Health care power, 1 when the region gets it, 0 otherwise.	Dummy	Authors' elaboration
investment	GFCF/GVA.	Ratio	BD.MORES
school	Human capital.	Average schooling years based on 1970 General Education Law	Valencian Economic Research Institute (IVIE)
fertility	Fertility rate.	Ratio. Births per thousand women	INE

Table 2 Descriptive statistics

Variable	Mean	Std. Dev.	Min.	Max.	Observations
y (per capita income)	11.21	3.28	4.39	20.20	476
imr	1.59	0.63	0.54	3.60	476
health power	0.43	0.50	0.00	1.00	476
investment	0.20	0.04	0.08	0.44	476
school	7.55	1.12	5.12	10.51	476
fertility	42.64	10.43	23.38	82.77	476

Table 3 Estimates of the growth regression. Dependent variable: per capita income growth

	Linear Panel		GMM		Sys-GMM	
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**
log y_{-1}	-0.021 ** (2.03)	-0.191 *** (7.23)	-0.067 *** (4.45)	-0.154 *** (6.52)	-0.053 *** (4.27)	-0.113 *** (6.31)
log imr	-0.009 (1.07)		-0.023 ** (2.44)		-0.015 * (1.86)	
health power	0.002 (0.34)	-0.017 ** (2.01)	0.005 (0.76)	-0.009 (1.22)	0.004 (0.66)	-0.012 ** (2.02)
log investment		0.037 ** (2.28)		0.025 * (1.65)		0.019 (1.61)
log school		0.042 *** (5.63)		0.030 *** (4.59)		0.024 *** (4.76)
log fertility		-0.030 (1.60)		-0.016 (0.89)		-0.007 (0.55)
constant	0.076 *** (2.92)	0.333 *** (3.57)	0.190 *** (5.22)	0.264 *** (3.17)	0.155 *** (2.22)	0.170 *** (2.75)
Hausman test: *p*-value	0.38	0.00				
Sargan test: *p*-value			0.22	0.65	0.42	0.62
Observations	459	459	408	408	408	408

z and t-statistics in parentheses. ***, **, and * denote significant at 1%, 5%, and 10% respectively. Instruments: lagged values of the endogenous variables.

	investment		*school*		*fertility*	
	(1)	***(2)***	***(3)***	***(4)***	***(5)***	***(6)***
log imr	-0.207 *** (6.16)	-0.090 ** (1.99)	-2.321 *** (23.91)	-1.439 *** (16.62)	0.317 *** (11.09)	0.224 *** (5.89)
health power	0.110 *** (4.41)	0.082 *** (3.38)	0.484 *** (6.73)	0.271 *** (5.83)	0.027 (1.28)	0.049 ** (2.40)
log y_{-1}		0.251 *** (4.21)		1.889 *** (16.58)		-0.198 *** (3.95)
Sargan test: *p*-value	0.11	0.25	0.09	0.59	0.38	0.68

Table 4 IV estimates of the channel equations

Observations: 442. *z*-statistics in parentheses. ***, **, and * denote significant at 1%, 5%, and 10% respectively. Instruments: lagged values of the endogenous variables.

Table 5 System estimates of the infant mortality effects. 3SLS estimation

	Effect of channel on growth	**Effect of infant mortality on channel**
	(1)	***(2)***
investment effect	0.199*** (3.16)	-0.182*** (4.87)
school effect	0.032*** (4.02)	-1.144*** (-16.66)
fertility effect	-0.189*** (3.29)	0.191*** (5.42)
TOTAL EFFECT		-0.108

z-statistics in parentheses; ***, **, and * denote significant at 1%, 5%, and 10% respectively.