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Abstract

This paper investigates the factors that determine differences across OECD countries in health outcomes, using data on life expectancy at age 65, over the period 1960 to 2007. We estimate a production function where life expectancy depends on health and social spending, lifestyle variables, and medical innovation. Our first set of regressions includes a set of observed medical technologies by country. Our second set of regressions proxy technology using a spatial process. The paper also tests whether in the long-run countries tend to achieve similar levels of health outcomes. Our results show that health spending has a significant and mild effect on health outcomes, even after controlling for medical innovation. However, its short-run adjustments do not seem to have an impact on health care productivity. Spatial spill overs in life expectancy are significant and point to the existence of interdependence across countries in technology adoption. Furthermore, nations with initial low levels of life expectancy tend to catch up with those with longer-lived populations.

JEL No. C31, C33, H51

Key Words: Life expectancy, health care production, health expenditure, spatial dependence

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Francesco Moscone and Elisa Tosetti acknowledge financial support from ESRC (Ref. no. RES-061-25-0317). We thank two anonymous referees, Alberto Holly, Stephen Hall, John Mullahy, Edward Norton, Andrew Jones, and the participants of the II Health Econometrics Workshop, held in Rome in July 2010.

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1 Introduction

The last few decades have witnessed rapid growth in health expenditure. From 1960 to 2007, health care expenditure in OECD countries increased, on average, from 3.8 per cent to 9.0 per cent of GDP. Considerable attention has been given to understanding the factors that have produced such growth. This includes looking at the relationship between health spending and income, and reviving economic theories linked to the low productivity of the health sector, such as the Baumol (1967) cost disease theory. An alternative explanation for the rise in health spending is that over time people tend to demand and obtain higher quality of health care (Skinner et al., 2005). There continues to be a live discussion on whether, ceteris paribus, higher health spending corresponds to better health outcomes. A number of empirical studies support the hypothesis of a flat curve of health care expenditure, namely that more spending does not have a significant impact on health outcomes (Fisher et al., 2003; Skinner et al., 2005; Fisher et al., 2009). Other studies, for example the work by Baicker and Chandra (2004), even find a negative correlation between health quality measures and health spending.

Jones (2002) formalizes and empirically tests a model where health expenditure and life expectancy are endogenous variables driven by technological progress. He finds little association between changes in life expectancy and changes in health expenditure (as a share of GDP) in the US. However, interestingly enough, the author also finds that a large fraction of the increase in health spending over time is driven by medical advances. Hall and Jones (2007) estimate an health production function for the US that relates age-specific mortality rates to health spending and technology. Their finding support the theory that the rising health expenditure relative to income occurs as consumption of non-health goods and services grows more slowly than income. As people get richer and saturated with non-health consumption, they become more willing to devote their resources to purchase additional years of life. Skinner and Staiger (2009) develop a macroeconomic model of productivity and technology diffusion to explain persistent productivity differences across US hospitals. Focusing on US Medicare data, they find that cost-effective medical innovations explain a large fraction of persistent variability in hospital productivity, and swamp the impact of traditional factor inputs. Additionally, they argue that there is a clear polarization in health care productivity between hospitals that usually tend to adopt less technology, the so-called “tortoises”, and those that traditionally adopt more technology, the “tigers”. Survival rates in low-diffusion hospitals lag by roughly a decade behind high-diffusion hospitals.

That technological progress has an important impact both on health outcomes and spending is well known. Medical advances allow ill people that could not be treated in the past to be cured today. In some cases, technology progressively reduces the cost of treatments. For example, in the case of acute myocardial infarction, new technologies have the characteristic of being less invasive, ultimately reducing hospital stays, rehabilitation times, and health costs. The less invasive coronary stents delivered percutaneously, as well as drug eluting stents, are gradually taking over bypass surgery. Using US data, Cutler and Huckman (2003) examine the diffusion over the past two decades of percutaneous coronary interventions to treat coronary
artery disease. They find that percutaneous coronary interventions improve health productivity, especially when substituting more invasive and expensive interventions such as coronary artery bypass graft surgery. In recent years, pharmaceuticals such as statins were dispensed for prevention, proving to be effective in reabsorbing atherosclerotic plaques and hence reducing the need for angioplasty, and the associated costs. We refer to Moise (2003) for further discussion on how technological change affects health expenditures.

This paper models differences across OECD countries in health productivity as a function of traditional factor inputs, life styles conditions, technological progress. In our empirical exercise we first explore available data on medical technology to explain health productivity in the OECD countries. However, given the paucity of the data and the difficulty in measuring medical technology at the country level, we assume that technology is unobserved, and proxy for it by means of a spatial process. Our set-up is similar to that proposed by Ertur and Koch (2007) and Frischer (2010), where we allow technological progress in a country to be related to the technology adopted by neighboring countries. That technology may show a geographical pattern is well known in the economic literature (see, for example, Keller (2004)). In the medical literature, a consolidated body of research supports the important role of interpersonal communication and social networks in the diffusion of medical technologies (see, for example, the classic diffusion study by Coleman, Katz and Menzel, (1966)). We refer to Birke (2009) for a survey on the role of social networks in explaining individual choices in a large variety of economic, social and health behavior. Communication and information sharing may occur not only within national boundaries, but also across countries through social interaction in conferences, training or visiting programs, or the publication of results from clinical studies involving medical technologies. For example, Tu et al. (1998) demonstrated a strong correlation between the publication of studies on the use of a particular technology in the prevention of stroke and the corresponding rates of utilization in the US and Canada. They show that utilization rates increased dramatically between 1989 and 1995 following the publication of two influential clinical studies demonstrating the effectiveness of the procedure. Thus, international spill overs resulting from foreign knowledge and human capital externalities may impact technological progress in one country. In a recent paper, Papageorgiou et al. (2007) study the impact of a set of measures of international medical technology diffusion on health status, concluding that technology diffusion is an important determinant of life expectancy and mortality rates. Spatial interdependence in the adoption of medical technology may also occur if one country strategically mimics neighbouring health policies, for example by adopting the same vaccine to prevent the diffusion of a contagious disease. Similar policies may be adopted in neighbouring countries on the basis of new clinical evidence (e.g., from international multicenter studies) available to them.

Our model allows us to test a number of hypotheses. One important question is whether factor inputs still have an impact on health care productivity after having controlled for technological progress. This has important policy implications on the allocation of resources to the health sector. If, as some studies suggest, factor inputs are no longer effective in improving
health outcomes, then policy makers may decide to focus on reforms aimed at improving the efficiency of the health sector. For example, a nation could argue against further hospital expansion or recruitment of more specialists in over-supplied geographical areas. Another research question is whether there exist significant spatial spill overs in medical technology adoption across countries, and how these influence health outcomes. Finally, we wish to test if health productivity tend to converge to the same level in the OECD countries. Put it differently, our aim is to explore whether countries that started with lower health outcomes in the long-run catch up with countries that initially had higher levels of health outcomes. Failure to reach such convergence may call on institutions such as the World Health Organization, or the European Community to implement policies to help countries with persistent low health productivity.

The plan of the paper is as follows. Section 2 presents the empirical model. Section 3 briefly reviews the literature on the determinants of life expectancy. Section 4 presents the data. Section 5 summarizes our empirical results, and points to some of the limitations of our study. Section 6 gives some concluding remarks.

2 The health production function

Let $h_{it}$ be a measure of health outcome in country $i = 1, 2, ..., N$ at time $t = 1, 2, ..., T$. We assume a simple Cobb-Douglas production function in physical capital and labour

$$\ln h_{it} = \ln a_{it} + \beta_K \ln K_{it} + \beta_L \ln L_{it}, \quad (1)$$

where $a_{it}$ is the level of medical technology in country $i$ at time $t$. $L_{it}$ and $K_{it}$ represent labour and capital inputs per capita in the health sector in country $i$ at time $t$. The variable $K_{it}$ includes tangible assets such as building and equipment for the health care sector that may be accumulated for example using resources allocated from the rest of the economy.

In our framework, medical innovation $a_{it}$ includes all treatments, procedures, and devices that may be used to prevent, diagnose, and treat health problems. Following Ertur and Koch (2007), and Frischer (2010), we assume that these technologies are driven by the following spatial process:

$$\ln a_{it} = \mu_i + d_t + \rho \sum_{j=1}^{N} w_{ij} \ln a_{jt} + \theta \ln K_{it}, \quad (2)$$

where $\mu_i$ denotes a country-specific effect, $d_t$ denotes a time-specific effect, $w_{ij}$ are elements of a known $N \times N$ spatial weights matrix, which is row normalized, i.e., $\sum_{j=1}^{N} w_{ij} = 1$. The time-specific coefficients capture the stock of medical knowledge common to all countries, while the individual-specific effects capture heterogeneity at the country level.

The parameter $\rho$ measures the strength of interdependence in medical technological innovation between neighbouring countries. We assume that $0 \leq \rho < 1$. The parameter $\theta$ describes the strength of home externalities generated by physical capital accumulation.
Substituting (2) in equation (1) we obtain

\[ \ln h_{it} = \mu_i + d_t + \rho \sum_{j=1}^{N} w_{ij} \ln a_{jt} + (\theta + \beta_K) \ln K_{it} + \beta_L \ln L_{it}. \]  

(3)

To get rid of the spatial lag of technology, we subtract the spatial lag \( \rho \sum_{j=1}^{N} w_{ij} \ln h_{jt} \) from both sides of equation (3) to obtain

\[ \ln h_{it} = \mu_i + d_t + \rho \sum_{j=1}^{N} w_{ij} \ln h_{jt} + (\theta + \beta_K) \ln K_{it} + \beta_L \ln L_{it} \]

\[ -\beta_K \rho \sum_{j=1}^{N} w_{ij} \ln K_{jt} - \beta_L \rho \sum_{j=1}^{N} w_{ij} \ln L_{jt}. \]  

(4)

Following Skinner and Staiger (2009), we use total per capita health expenditure as a proxy for the a bundle of factor inputs, rather than capital and labour, separately.

As a measure of health outcomes we focus on life expectancy for males at age 65. This is measured as the average number of years that a male person at age 65 can be expected to live assuming that age-specific mortality levels remain constant. This can be considered as a summary of the mortality conditions at this age and at all subsequent ages. By focusing on life expectancy for males at age 65, we aim at eliminating the heterogeneity in life conditions, gender differences existing at the country-level that may affect the analysis of general mortality rate, or life expectancy at birth.

The coefficient attached to the spatial lag in equation (4) measures how the health outcome in one country is correlated with health outcomes in neighbouring countries due to technological diffusion. However, we realize that observed similarities in health outcomes could also be the effect of other factors, both observable or unobservable, that influence health outcomes and that are correlated across countries (Manski, 1993).

In the next section, we provide a brief survey of the determinants of life expectancy.

3 A brief review of the determinants of life expectancy

Shaw et al. (2005) look at the geographical patterns in life expectancy at age 40 and 65 (for both males and females) across 19 OECD countries in 1997 as a function of income, health and pharmaceutical expenditures and a set of risk factors temporally lagged. They find that health spending has a positive influence on the dependent variable, thus, finding evidence against the hypothesis of a flat cost curve. They also find that pharmaceutical expenditure has a positive effect on life expectancy both at middle and advanced ages, though this effect changes when one controls for the age distribution of the population. Schoder and Zweifel (2009) study the inequality in life expectancy within country and, following the work by Hanada (1983), construct
a Gini coefficient for the distribution of length of life. Using OECD health data for 24 countries between 1960 and 2004, the authors suggest that medical and non-medical inputs have a negative effect on the second moment of the distribution. Although the inputs do have an impact on the dependent variable, this result, in light of the law of diminishing marginal productivity, supports the hypothesis of a flat cost curve. Akkoyunlu et al. (2009) address the issue of spurious correlation in the production of health, by estimating a conditional error correction model for life expectancy. They apply the bounds testing procedure developed by Pesaran et al. (2001). The authors find a significant relationship between life expectancy, pharmaceutical innovation, and public health care expenditure in the US. Crémieux et al. (1999, 2005) study the relationship between health expenditure and health outcomes in Canadian provinces, finding that lower spending is associated with a statistically significant increase in infant mortality and a decrease in life expectancy. Using data on 63 countries over the period 1961 to 1995, Papageorgiou et al. (2007) study the impact on life expectancy and mortality of a set of measures of diffusion in medical innovation. They construct a set of measures of flows of medical R&D originating from advanced economies and directed to the so-called “non-frontier” countries. The authors conclude that technology diffusion is an important factor in explaining variations in the long-run averages of life expectancy and mortality in “non-frontier” countries.

A different approach in studying life expectancy is taken by Hall and Jones (2007). The authors develop an economic model that explains the evolution in the value of life and its relation with health spending. They calculate the marginal cost of saving a life at different ages and over time in the US, and find that its growth over time may explain the observed rise in health spending.

4 Data and empirical specification

From the discussion in Section 2, we adopt the following empirical specification

\[
\ln h_{it} = \mu_i + d_t + \rho \ln \bar{h}_{it} + \beta_1 \ln h_{exp, it} + \beta_2 \ln h_{exp, it} + u_{it},
\]

where \( h_{it} \) is life expectancy for males at age 65, and \( \mu_i \) and \( d_t \) are country-specific and year-specific effects. The variable \( h_{exp, it} \) is total per-capita health expenditure,\(^1\) and \( \ln \bar{h}_{it} \) and \( \ln h_{exp, it} \) are the spatial lags of \( \ln h_{it} \) and \( \ln h_{exp, it} \).

We used a weights matrix based on the inverse distance expressed in kilometers between countries. Other geographical metrics can be used such as economic proximity or similarity and social proximity (e.g. Baicker, 2005).

We gathered data on 25 OECD countries observed over the period 1960 to 2007.\(^2\) This rich data set contains over 1200 variables, including various measures of health status, health care

\(^1\)Total health expenditure is defined by the OECD as the sum of spending on activities that has the goals of promoting health and preventing disease. See OECD (2009).

\(^2\)The data source is OECD Health Data 2010. Due to the missing observations problem, we have excluded Poland, Portugal, Slovak Republic, Spain and Italy from our sample.
resources and utilization, health spending and financing. Drawing from this data, we incorporate in the regression a number of variables to control for differences across countries and over time in lifestyles. Specifically, we consider three important variables related to lifestyle, given by daily fat intake, alcohol and tobacco consumption (see Table 1 for a description). Further, we include social expenditure for old people, defined as all benefits and financial contributions to support the elderly during circumstances which adversely affect their welfare. We note that the variable social spending is only available for the years 1980 to 2005. Both health expenditure and social expenditure are expressed in per-capita terms and have been adjusted for purchasing power parity. We recognize that other factors, such as body weight and education may affect life expectancy (Deaton and Paxon, 2001; Hendricks and Graves, 2009; Culter et al. 2006). However, for many countries, data on these additional variables are either not available or available for a very short time period.

Table 1 shows some descriptive statistics on the variables included in the model. We observe that our data set is highly unbalanced; in particular the sample size drops significantly when the variable social expenditure is added to the regression.

### Table 1: Definition of variables and descriptive statistics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Description</th>
<th>Mean</th>
<th>St. dev.</th>
<th>N obs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>$h$</td>
<td>N. of years</td>
<td>14.1</td>
<td>1.7</td>
<td>1,284</td>
</tr>
<tr>
<td>hexp</td>
<td>Per-capita, in US$ at 2000 PPP rates</td>
<td>1,605.4</td>
<td>905.4</td>
<td>935</td>
</tr>
<tr>
<td>fat</td>
<td>Grammes per capita per day</td>
<td>119.4</td>
<td>28.6</td>
<td>1,183</td>
</tr>
<tr>
<td>tobacco</td>
<td>Annual per capita in grammes</td>
<td>2,326.5</td>
<td>690.8</td>
<td>919</td>
</tr>
<tr>
<td>alcohol</td>
<td>Annual per capita in liters</td>
<td>10.0</td>
<td>3.9</td>
<td>1,241</td>
</tr>
<tr>
<td>socexp</td>
<td>Per-capita, in US$ at 2000 PPP rates</td>
<td>1,264.9</td>
<td>809.3</td>
<td>660</td>
</tr>
</tbody>
</table>

Notes: (*): per capita in this case means divided by population aged 15 years and over.

### 5 Empirical findings

Figure 1 shows life expectancy for males at age 65 in the OECD countries in 1960 and in 2007. During these years, life expectancy has increased markedly, rising from an average of 12.7 years in 1960 to 16.8 in 2007. That this measure of health outcome has risen greatly among developed countries is well known, suggesting not only that greater numbers of individuals are reaching old age but also that elderly people are living longer (Jagger et al., 2008; Cutler et al., 2006).
However, it is important to observe that populations are not ageing uniformly in all nations. Australia and Japan experienced particular strong gains in life expectancy over time, placing them at the top of the ranking in recent years. In contrast, countries from Eastern Europe, such as Hungary and the Slovak Republic show the lowest values for life expectancy throughout the sample period. According to the OECD (2009) health report, the gains in life expectancy registered in the OECD countries can be explained in part by a marked reduction in death rates from heart disease and celebro-vascular diseases (stroke) among elderly people.

Figure 2 reports the time series patterns of life expectancy for the OECD countries. Note that, towards the end of the sample period, life expectancy patterns in most countries tend to get closer. Only five countries diverge substantially from this trend and show a low life expectancy throughout the sample period. These are Hungary, Slovak Republic, Turkey, Poland and the Czech Republic. Later in the paper, we will test whether in the long-run countries tend to achieve similar levels of health outcomes.

Figure 3 shows the plot of the average life expectancy at age 65 and average health spending across countries for the period 1969 to 2007. As expected, both series trend up (as also confirmed by our non-stationary tests reported in Table 4 below). Life expectancy shows a stable increase over time, while health spending seems to rise more rapidly at the beginning and at the end of our sample period.
Figure 2: Life expectancy at age 65 in the OECD countries over the period 1960-2007

Figure 3: Life expectancy at age 65 and health spending over the period 1969-2007
Figure 4 shows the standardized Moran statistic\(^3\) for the variable \(\ln h\) in the OECD countries for the period 1969 to 2007. Note that all values of this statistic above the red line are statistically significant at the 5 per cent significance level. These results show a significant Moran statistic for the years 1980-1984 and from 1990 onwards. This initial exploratory analysis indicates the presence of geographical concentration of the variable life expectancy at age 65, which will be incorporated in our empirical model. It is also suggested by the economic theory discussed in Section 2.

First, we discuss the estimation results of our production function using some observed measures of medical technology available at the country level. Table 2 presents a set of technology variables for the treatment of problems of the cardiovascular system, which are known to be the leading cause of morbidity and mortality in older adults (OECD, 2009). These variables are the number of percutaneous coronary interventions (PCI), the number of coronary bypass and stents placed on patients with cardiovascular problems, the number of daily doses of lipid modifying and beta-blocking agents. We gathered these variables from the OECD Health Data 2010.

The first two technologies have been used by Cutler and Huckman (2003) to study the impact of technology diffusion on health productivity in New York state. Moise (2003) has also studied the mechanisms of diffusion of these procedures in the OECD countries, showing

\(^3\)For each time period the Moran statistic has been standardized by using the moments of the empirical distribution generated by a random permutation procedure.
that the most important determinants of their utilization are GDP and hospital characteristics such as technology regulation and payment methods for hospitals and physicians. Coronary stents represent perhaps the most important improvement to PCI since the mid-1990s. Lipid modifying and beta-blocking agents are drugs aimed at preventing and treating cardiovascular disease (Dickson and Jacobzone, 2003). We note that bypass surgery, widely diffused prior to the early 1980s, is an invasive procedure that has been progressively substituted by the less traumatic coronary stents delivered percutaneously. With the exception of bypass surgery, the technology variables we have chosen have the characteristic of being minimally invasive and less costly than existing technologies for which they are often substitutes.

It is important to note that these technologies are only a subset of all possible technologies that may adopted to prevent, diagnose, and treat health problems for people aged over 65. We refer to Comin and Hobijn (2009, 2010) for an extensive discussion of existing medical and non-medical technologies. Most of these variables are available only from 1990, and even then, only for a few countries. Table 2 shows the number of observations per technology variable.

Table 2: Technology measures and their correlation with life expectancy and its spatial lag

<table>
<thead>
<tr>
<th>Technology</th>
<th>Description</th>
<th>n.obs.</th>
<th>Corr. with ln h</th>
<th>ln h</th>
</tr>
</thead>
<tbody>
<tr>
<td>ln perc</td>
<td>n. percutaneous coronary interv.(^{(1)})</td>
<td>285</td>
<td>0.281*</td>
<td>0.361*</td>
</tr>
<tr>
<td>ln bypass</td>
<td>n. coronary bypass(^{(1)})</td>
<td>293</td>
<td>0.042</td>
<td>0.024</td>
</tr>
<tr>
<td>ln stent</td>
<td>n. coronary stenting(^{(1)})</td>
<td>181</td>
<td>0.142*</td>
<td>0.199*</td>
</tr>
<tr>
<td>ln statin</td>
<td>Cons. of lipid modifying agents(^{(2)})</td>
<td>189</td>
<td>0.740</td>
<td>0.887*</td>
</tr>
<tr>
<td>ln betabli</td>
<td>Cons. of beta-blocking agents(^{(2)})</td>
<td>278</td>
<td>-0.045</td>
<td>0.247*</td>
</tr>
</tbody>
</table>

\(^{(1)}\): expressed in n. procedures per 100,000 population (in-patients). \(^{(2)}\): Expressed as n. of defined daily doses (DDD) per 1,000 inhabitants. (\(^{*}\)): Significant at 5 per cent significance level.

While aware of these data limitations, Table 3 explores the role in the production function of each technology separately, given that the presence of missing values prevented us from building a composite index of medical technology. The reported regressions do not include the spatial lags of life expectancy, due to their high correlation with the technology variables, as reported in Table 2. Further, time dummies have not been included due to the little variation over time of our variables, caused by data limitations. For comparison purposes, we also show a regression with no technology variables (see column I). Note that, when including our observed measures of innovation, the sample size drops significantly.

Under the classic FE specification, health spending has a significant impact on life expectancy. All technologies except for coronary stents have a positive and significant impact before 1995, data on all these technology variables are available for no more than 9 countries.
on our health outcome. Further, the coefficient attached to health spending is significant, ranging from 0.059 when the variable measuring consumption of beta-blocking agents is included in the regression, to 0.264 in the case of technology coronary stents. Again the latter number is based on a fewer number of observations and should be interpreted with caution. Among the lifestyle variables, consumption of tobacco is statistically significant with the correct sign in all regressions except for the one with Statin, where it is positive but insignificant. Consumption of alcohol has a negative but statistically insignificant effect on life expectancy in all regressions except for the one with beta blockers, where it is negative and significant. Fat intake has a negative and significant effect on life expectancy in the FE regression, but is positive and insignificant in most technology variable regressions. Again this may be due to the number of observations lost due to the paucity of these technology variables.

Table 3: Estimation of the health production function including observed medical technology

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<td></td>
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<tr>
<td>ln percc</td>
<td>0.020*</td>
<td>0.005</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>ln bypass</td>
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<td>0.017*</td>
<td>0.005</td>
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<td>-</td>
</tr>
<tr>
<td>ln stent</td>
<td>-</td>
<td></td>
<td>-</td>
<td></td>
<td>-0.002</td>
<td>0.003</td>
<td>-</td>
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<td>ln statin</td>
<td>-</td>
<td></td>
<td>-</td>
<td></td>
<td>-</td>
<td></td>
<td>0.022*</td>
<td>0.009</td>
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<tr>
<td>ln betabl</td>
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<td>-</td>
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<td></td>
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<tr>
<td>ln hexp</td>
<td>0.127*</td>
<td>0.028</td>
<td>0.119*</td>
<td>0.025</td>
<td>0.264*</td>
<td>0.036</td>
<td>0.172*</td>
<td>0.035</td>
<td>0.059*</td>
<td>0.024</td>
</tr>
<tr>
<td>ln tobacco</td>
<td>-0.077*</td>
<td>0.017</td>
<td>-0.079*</td>
<td>0.019</td>
<td>-0.071*</td>
<td>0.021</td>
<td>0.006</td>
<td>0.029</td>
<td>-0.104*</td>
<td>0.019</td>
</tr>
<tr>
<td>ln alcohol</td>
<td>-0.017</td>
<td>0.027</td>
<td>0.012</td>
<td>0.030</td>
<td>-0.030</td>
<td>0.034</td>
<td>-0.239</td>
<td>0.052</td>
<td>-0.056*</td>
<td>0.026</td>
</tr>
<tr>
<td>ln fat</td>
<td>0.032</td>
<td>0.050</td>
<td>0.009</td>
<td>0.051</td>
<td>0.017</td>
<td>0.050</td>
<td>0.254*</td>
<td>0.086</td>
<td>0.032</td>
<td>0.060</td>
</tr>
<tr>
<td>ln socsp</td>
<td>0.013</td>
<td>0.022</td>
<td>0.059*</td>
<td>0.019</td>
<td>-0.020</td>
<td>0.025</td>
<td>-0.016</td>
<td>0.045</td>
<td>0.082*</td>
<td>0.012</td>
</tr>
<tr>
<td>n. obs</td>
<td>139</td>
<td>146</td>
<td>77</td>
<td>83</td>
<td>146</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: individual fixed effects have been included included. (*): Significant at 5 per cent significance level.

We now turn to the estimation of our production function keeping technology unobserved, as outlined in model (5). This allows us to expand the sample size considerably.

Our exploratory data analysis suggested that both life expectancy and health spending may be non-stationary raising some concern on the validity of our OLS estimates (Engle and Granger, 1987). Table 4 reports the Pesaran (2007) CIPS panel unit root tests on our variables. The low power of country by country test is one of the major motivation for the use of panel unit root tests. A Monte Carlo exercise reported in Baltagi et al. (2007) has shown that CIPS test is quite robust to the presence of spatial dependence as explicitly modeled in our framework. The output provides evidence of non-stationarity for all variables, both when an intercept only is included in the specification or when an intercept and a trend are included. The results are
obtained including 3 lags in the ADF regressions\(^5\). Non-stationarity of life expectancy is justified by the declining mortality pattern for the elderly. According to the UN-World Bank Population database, life expectancy on average for men at age 65 is likely to be 18.1 years in 2040 in the OECD countries (see also Hendricks and Graves, 2009).

Note that all variables except for fat intake and social expenditure for old people are stationary when first differences are applied. We have also computed a CIPS statistic for the spatial lag of the variable life expectancy, namely \(\ln h_{it}\). Given the non-stationary nature of \(\ln h_{it}\), we verify whether applying the spatial operator may render the dependent variable stationary. However, the CIPS test does not reject the null hypothesis, thus indicating that \(\ln h_{it}\) is non-stationary.

Table 4: Panel unit root tests and cointegration analysis

<table>
<thead>
<tr>
<th>Variables</th>
<th>Intercept only</th>
<th>Intercept and trend</th>
<th>In first diff. (interc. only)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\ln h)</td>
<td>1.287</td>
<td>0.693</td>
<td>-7.072*</td>
</tr>
<tr>
<td>(\ln h)</td>
<td>1.252</td>
<td>-2.360*</td>
<td>-13.709*</td>
</tr>
<tr>
<td>(\ln h_{exp})</td>
<td>3.082</td>
<td>1.676</td>
<td>-3.029*</td>
</tr>
<tr>
<td>(\ln tobacco)</td>
<td>1.572</td>
<td>-3.100</td>
<td>-3.596*</td>
</tr>
<tr>
<td>(\ln alcohol)</td>
<td>2.735</td>
<td>0.799</td>
<td>-8.054*</td>
</tr>
<tr>
<td>(\ln fat)</td>
<td>-0.447</td>
<td>0.873</td>
<td>-5.212*</td>
</tr>
<tr>
<td>(\ln socsp)</td>
<td>5.456</td>
<td>2.361</td>
<td>-6.407*</td>
</tr>
</tbody>
</table>

(*): Significant at 5 per cent significance level.

Next, we checked whether our variables are cointegrated, by computing the Pedroni (1999) parametric group \(t\)-statistic to the residuals from a regression of life expectancy on its spatial lag and on the variables provided in Table 1. Table 5 reports the estimation of model (5) and the Pedroni (1999) and Kao (1999) cointegration tests. As noted by Beenstock and Felsenstein (2010), if variables are cointegrated, the OLS estimator for regression parameters in (5) is super-consistent, regardless the endogeneity of the spatial lag \(\ln h_{it}\) appearing on the right hand side of the equation. For this reason, there is no need to use spatial techniques such as IV or ML, to deal with the endogeneity of \(\ln h_{it}\). We refer to Stock (1983) for further details on super-consistency of the OLS estimator. As a further check, in Column (II) we report estimation of (5) also by the IV approach, where instruments are given by the spatial lags of the included regressors (Kelejian and Prucha, 1998), and the temporal lag of the spatial lag, namely \(\Delta \ln h_{it-1}\). Results show very similar coefficients to the OLS estimates (Column I). Both Pedroni and Kao cointegration tests reported are significant, suggesting the existence of a long-run economic relationship between health productivity, expenditure, the lifestyle variables and social spending for the elderly. In

\(^5\)Some robustness checks show that the results reported do not change when varying the number of lags included in the ADF regression.
the last column of Table 5 (Column (III)) we also report the dynamic fixed effects estimator of the long-run coefficients and of the error correction term (Pesaran, Shin, and Smith, 1999). Results confirm the significant effect of health spending on the dependent variable, as well as the presence of a sizeable spatial effect. Specifically, a one percentage increase in health spending induces a rise of 0.03-0.05 per cent in life expectancy on average. This corresponds to almost an extra half year of longevity. All risk factors show, as expected, a negative effect on the dependent variable. A rise of 1 per cent in annual tobacco per capita consumption implies a reduction in life expectancy of around 0.04-0.06 per cent. This corresponds to over a half year reduction in longevity on average. As for fat intake, a 1 per cent increase in per capita consumption reduces the dependent variable by 0.07-0.09 per cent. This is almost one additional year of life expectancy, on average.

Table 5: Estimation of the health production function

<table>
<thead>
<tr>
<th>Variables</th>
<th>(I) Static FE</th>
<th>(II) IV FE</th>
<th>(III) Dynamic FE</th>
</tr>
</thead>
<tbody>
<tr>
<td>ln(h)</td>
<td>0.364*</td>
<td>0.159</td>
<td>0.360*</td>
</tr>
<tr>
<td>ln(hexp)</td>
<td>0.032*</td>
<td>0.011</td>
<td>0.032*</td>
</tr>
<tr>
<td>ln(tobacco)</td>
<td>-0.046*</td>
<td>0.007</td>
<td>-0.045*</td>
</tr>
<tr>
<td>ln(alcohol)</td>
<td>-0.010</td>
<td>0.010</td>
<td>-0.011</td>
</tr>
<tr>
<td>ln(fat)</td>
<td>-0.073*</td>
<td>0.022</td>
<td>-0.071*</td>
</tr>
<tr>
<td>ln(socsp)</td>
<td>0.011*</td>
<td>0.006</td>
<td>0.010</td>
</tr>
<tr>
<td>Error correction term</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>n.obs</td>
<td>426</td>
<td></td>
<td>425</td>
</tr>
<tr>
<td>Pedroni ADF group test</td>
<td>-9.596*</td>
<td>(0.00)</td>
<td></td>
</tr>
<tr>
<td>Kao test</td>
<td>-2.347*</td>
<td>(0.01)</td>
<td></td>
</tr>
</tbody>
</table>

Notes: Individual fixed effects and time dummies are included.
(*) : significant at 5 per cent significance level. p-values in parenthesis.

One may argue that life expectancy in a country is affected not only by the current level of resources deployed in the health sector, but also by what has been allocated in the past. Hence, as a robustness check we have also tried including in our regression the average of health spending over a pre-specified interval of time. Specifically, in (5) we have replaced \( \ln(hexp_t) \) with the variable \( \ln(hexp_t^n) = \sum_{s=0}^{n} \ln(hexp_{t-s}) \), where we have set \( n \) equal to 4, 9 and then 14 years. Results for the static fixed effects estimation are reported in Table 6. Comparing these results to those in Table 5, the influence of health resources spent over a given period of time on life expectancy is similar to the impact of current level of health resources.
Table 6: Estimation of the health production function using average health spending

<table>
<thead>
<tr>
<th>Variables</th>
<th>(I) n=4</th>
<th></th>
<th>(II) n=9</th>
<th></th>
<th>(III) n=14</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ln h</td>
<td>0.359* 0.158</td>
<td>0.375* 0.159</td>
<td>0.358* 0.159</td>
<td>0.375* 0.159</td>
<td>0.358* 0.159</td>
<td>0.375* 0.159</td>
</tr>
<tr>
<td>ln hexp</td>
<td>0.040* 0.012</td>
<td>0.031* 0.014</td>
<td>0.035* 0.014</td>
<td>0.031* 0.014</td>
<td>0.035* 0.014</td>
<td>0.031* 0.014</td>
</tr>
<tr>
<td>ln tobacco</td>
<td>-0.044* 0.007</td>
<td>-0.045* 0.007</td>
<td>-0.045* 0.007</td>
<td>-0.045* 0.007</td>
<td>-0.045* 0.007</td>
<td>-0.045* 0.007</td>
</tr>
<tr>
<td>ln alcohol</td>
<td>-0.009 0.010</td>
<td>-0.007 0.010</td>
<td>-0.007 0.010</td>
<td>-0.007 0.010</td>
<td>-0.007 0.010</td>
<td>-0.007 0.010</td>
</tr>
<tr>
<td>ln fat</td>
<td>-0.071* 0.022</td>
<td>-0.078* 0.022</td>
<td>-0.083* 0.022</td>
<td>-0.078* 0.022</td>
<td>-0.083* 0.022</td>
<td>-0.078* 0.022</td>
</tr>
<tr>
<td>ln socsp</td>
<td>0.007 0.007</td>
<td>0.009 0.007</td>
<td>0.010 0.007</td>
<td>0.009 0.007</td>
<td>0.010 0.007</td>
<td>0.009 0.007</td>
</tr>
<tr>
<td>n.obs</td>
<td>435</td>
<td>437</td>
<td>437</td>
<td>437</td>
<td>437</td>
<td>437</td>
</tr>
</tbody>
</table>

Having established the existence of a cointegration relationship, we now turn to the estimation of the following error correction model

\[
\Delta \ln h_{it} = \mu + d_t + \alpha (\bar{\Delta h}_{it}) + \delta \Delta \ln h_{it} + \gamma_1 \Delta \ln h_{exp}_{it} + \gamma_2 \Delta \ln h_{exp}_{it} + \varepsilon_{it},
\]

where in the parenthesis we have the previous period cointegration relation. The coefficient \( \alpha \) measures the speed of adjustment of life expectancy to a deviation from the long-run equilibrium relation between the dependent variable and the regressors. Again, we estimate the above model using 30 countries followed over 49 years. For estimation, we adopt an IV approach, where, again, instruments are given by the spatial lags of the included regressors (Kelejian and Prucha, 1998), and the temporal lag of the spatial lag, namely \( \Delta \ln h_{it-1} \). In this case, we drop from the regression \( \Delta \ln h_{exp}_{it} \) as it is highly correlated with \( \Delta \ln h_{it} \). Results are reported in Table 7. The variable health expenditure is statistically insignificant, suggesting that short-run adjustments in traditional input factors do not have an impact on health care productivity.\(^6\) There is also further evidence of high degree of spatial correlation suggesting that the adoption of technologies in a country is mostly driven by the adoption of the same technologies in neighbouring countries. While short-run adjustments in risk factors are statistically insignificant, fluctuations in social expenditure for the elderly seem to play a role in explaining life expectancy.

As previously observed in the descriptive data analysis, some Eastern European countries, at the beginning of our sample period are characterized by low life expectancies. We wish to test whether these countries in the long-run continue to experience low levels of life expectancy.

---

\(^6\) We have also checked the sensitivity of our estimates by removing one country at a time from the sample. The coefficient estimate of health spending varies very little. The only exception is when we remove the United States. In this case, the estimated coefficient of health spending increases from 0.035 to 0.05 for the FE specification. This indicates that the US exerts an influential set of observations in these regressions because the US is characterized by low longevity accompanied by high health expenditure levels (Preston and Ho, 2009).
or instead tend to catch up with the remaining countries. We do this by checking whether there exists beta convergence in life expectancy in the OECD countries (Barro and Sala-i-Martin, 1995). An empirical observation of beta convergence would suggest that countries tend to achieve similar levels of health outcomes in the long-run.

In order to do this, we regress the average growth rate of life expectancy over the period 1975 to 2006 on the initial level of life expectancy. Hence, our dependent variable is \( \frac{1}{32} \ln \left( \frac{h_{i,2006}}{h_{i,1975}} \right) \), while our key regressor is \( \ln h_{i,1975} \). Due to the unbalancedness of the data set, we focus only on 25 OECD countries. The results are reported in Table 8, Column (I). In Column (II) we also control for health expenditure in the initial period, while in Column (III) we include the spatial lag of life expectancy in the initial period to control for technological interdependence.

The estimated coefficient of \( \ln h_{1975} \) in Column (I) is negative and significant, suggesting that countries with a lower initial level of life expectancy have a faster health care growth than those with a higher initial level of life expectancy, and that they all converge to the same steady state. A similar result is obtained when controlling for initial level of health spending, as well as technological interdependence.
Table 8: The beta convergence of life expectancy

<table>
<thead>
<tr>
<th>Variables</th>
<th>(I)</th>
<th>(II)</th>
<th>(III)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \ln h_{i,1975} )</td>
<td>-0.015*</td>
<td>-0.021*</td>
<td>-0.022*</td>
</tr>
<tr>
<td>( \ln h_{exp_{i,1975}} )</td>
<td>-</td>
<td>-0.000</td>
<td>0.001</td>
</tr>
<tr>
<td>( \ln \bar{h}_{i,1975} )</td>
<td>-</td>
<td>-</td>
<td>-0.012</td>
</tr>
<tr>
<td>Speed of conv.</td>
<td>0.0004</td>
<td>0.0007</td>
<td>0.0007</td>
</tr>
<tr>
<td>( R^2 )</td>
<td>0.169</td>
<td>0.579</td>
<td>0.598</td>
</tr>
</tbody>
</table>

6 Concluding remarks

This paper studied the spatio-temporal variations in health productivity using panel data on life expectancy (at age 65), in the OECD countries, over the last three decades. We have estimated a production function where life expectancy depends on health and social spending, lifestyle, and medical innovation. The latter has been approximated by means of a set of technology variables such as percutaneous coronary interventions, and also using a spatial process. Our results show that health spending does have a significant but mild effect on health outcomes, even after controlling for medical innovation. However, its short-run adjustments do not seem to have an impact on health care productivity. One lesson to learn is that strategies aimed at reducing public resources in the long-run may contribute to slower improvements in life expectancy of the elderly.

Our study finds the presence of sizeable spatial spill overs in life expectancy, confirming the interdependency in technology adoption across countries. It also finds that in the long-run countries tend to achieve similar levels of health outcomes. It is interesting to observe that countries that initially have low levels of life expectancy tend to catch up with those with higher longevity, and that the initial levels of expenditure and medical technology do not affect the long-run growth in productivity. It is important to emphasize that our results should be interpreted with care, due to data limitations, and given the complexity of the phenomenon and the limited set of variables included in our analysis.

References


[34] Moise P. (2003), The technology-health expenditure link a perspective from the ageing-related diseases study. In OECD (2003), A disease-based comparison of health systems: what is best and at what cost?.


