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Material well-being and development: insights on the Preston curve

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Abstract

Life expectancy is a subject of natural interest, also because it is an obvious index of welfare. In 1975 Preston revealed a clear connection between the level of per capita income and life expectancy and this is the subject of this paper. Several authors show that the exact nature of this association is not clear and/or analyzed, and, moreover, the curve is subject to other limitations (e.g.: endogeneity). I show that the use of alternative variables, instead than income, gives very good results and makes a step forward in reducing the limits of the Preston curve.

JEL: O10, I15

Keywords: Preston Curve; Economic Development; Well-being

Material well-being and development: insights on the Preston curve[1]

Massimo Tamberi

Introduction

Life was "nasty, brutish and short" (Hobbes, cit. in Cutler et al., 2006). If this condition accompanied the human kind for hundreds of thousands of years, we have assisted at a spectacular rise in life conditions and length in recent times: life expectancy, as a consequence of the process of economic growth and development, is the subject of this paper.

In 1975 Preston revealed a clear connection between the level of per capita income and life expectancy; this relationship, on the one hand, is intriguing, but, on the other hand, elusive. In particular two limits are recognized: first, the fact that there are bi-directional causal links; second, the meaning of this relationship is not well clarified and income can be considered as a proxy for several other determinants, giving rise to a debate on the relative and historical role of different possible causes. This lack of clarity also provokes different policy recipes.

In this paper I estimate an "alternative" Preston curve, using data related to nutrition, sanitation and health systems for most world countries and for recent decades (even if it has to be recognized that some aspects of it are related to the long term); this approach potentially tackles all the problems arisen in the literature about the original Preston curve, eventually revealing specific channels of influence underlying the Preston curve itself. Moreover, statistical results seem clear and satisfying.

The Preston Curve

Life expectancy is a subject of natural interest, because of its immediate, intuitive meaning for all men and women; from the perspective of an economist, it is an obvious, albeit limited, index of welfare, material and probably also immaterial.

According to a very recent publication on long-term indexes of the quality of life, "Life expectancy seems to be fairly unique in showing unambiguous improvements at the same income level" (van Zanden, J.L., et al. (eds.), 2014, p. 35)

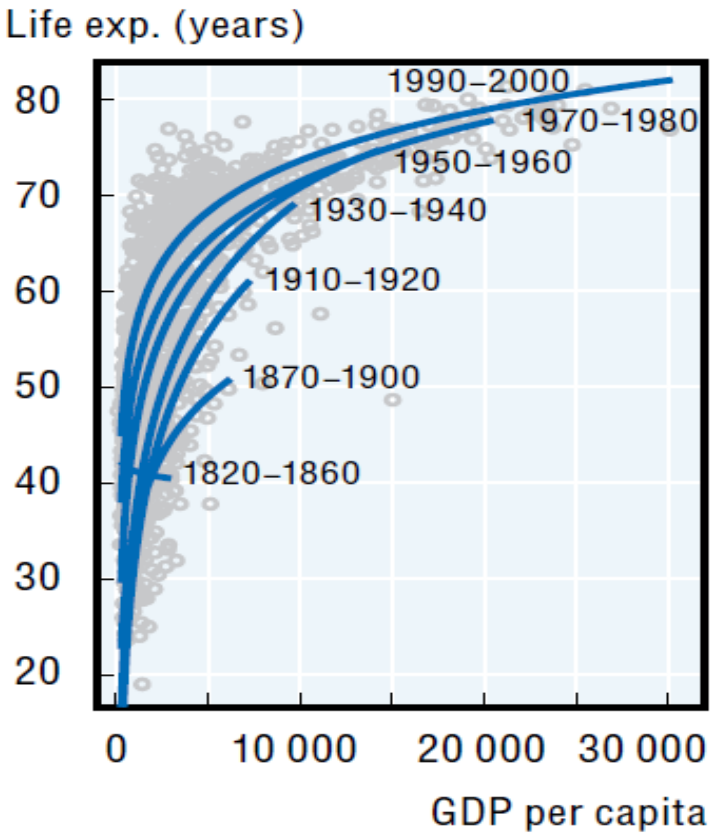
Considering, then, that for a scholar of economic growth and development, the most synthetic indicator of welfare has been, and perhaps still is, per capita income, we should not be surprised if life expectancy and income are correlated. In fact, in 1975 (and 2007) Preston revealed a clear connection between the level of

[1] *Thanks to Marco Gallegati, Roberto Esposti and the participants to the department seminar held on January 28th, 2015 for fruitful comments and discussion. The usual caveats apply*.

per capita income and life expectancy: a positive, non linear relation, shifting in time. The following figure 1 is the typical representation of the so called "Preston curve":

Figure 1

The Preston curve 1820-2000



Source: van Zanden, J.L., et al. (eds.) (2014)

This relationship says that per capita income and life expectancy are positively correlated, that there is a intrinsic nonlinearity at the cross-sectional level[2] and a progressive time shift.

Until very recently the Preston curve has attracted the attention of several scholars, as the paper of Dalgaard and Strulik (2014) testifies; nevertheless, and perhaps a little bit surprisingly, it has to be recognized that, in the past the subject did not arouse much attention among economists specifically: many papers and many references to the subject can be found in journals related to medicine, and the Preston paper is the first

[2] Oeppen and Vaupel (2002), and White (2002) suggest the possibility that life expectancy , if observed in time, does not show an upper limit and increases linearly, contrary to a common idea and to what the Preston curve itself seems to suggest. This result was reached statistically and was criticized from a biological perspective.

example of this[3]. Moreover, also in that field, the curve is more cited than analyzed, at least from a quantitative point of view.

While some indications appear in Bishai and Kung (2007), a summary of the debate on the Preston curve can be found in Georgiadis et al. (2010).

In short, we can see that some authors (Schnabel and Eilers, 2009) suggested different technical methods in order to quantify the relationship, but remaining in the same general sphere of analysis; some others (Georgiadis and al. 2010), distinguishing between rich and poor countries, found that the role of income was not so relevant and progressively diminishing; others (Husain, 2011), performed causality tests in order to understand "what causes what" between life expectancy and income; it was then suggested (Mackenbach, 2013) that no smooth trend links the two variables, but that specific historical factors also have relevant impacts on life expectancy. Finally Cutler et al. (2006), suggest that behind the Preston curve there are different causes and mechanisms influencing life expectancy: nutrition, sanitation, and medical treatments. The role of these factors was probably different in different periods: from the middle of the XVIII century to the middle of the XIX nutrition improved; then, and until the beginning of the XX, public health mattered more. Finally from the 1930s on "has been the era of big medicine".

With a completely different and theoretical approach, Dalgaard and Strulik, (cit.) look for the causes of mortality in a model in which individuals "deliberate investments in slowing down the aging process, thereby postponing his or her "date of expiry". Through this kind of model they concentrate completely on the "health technology", linked to medical treatments and scientific progress.

What is striking is that "the interpretation of the Preston curve continued to be shrouded in mystery" (Dalgaard and Trulik, cit.), and "unravelling the exact nature of this association is deemed difficult and complicated" (Husain, 2011).

In particular, problems can be summarized as follows:

- It is not clear which is the fundamental cause of the curve, i.e. it is not clear which are the possible determinants "behind" income: "the Preston curve raises questions about what per capita GDP actually signifies for population" (Taylor, 2009)
- Moreover, another difficulty has to do with the fact that between life expectancy and per capita income there are bi-directional causal links: "health is a cause as well as a consequence of income growth" (Bloom, Canning, 2007), and this is true because of several causes, from trivial "energy" of men and women in their work activity, to more indirect linkages as the incentive of investing in human capital (weak, if life is short).
- The lack of clarity in the deep understanding of the relation, as consequence of the limits outlined above, also stimulated a debate about the proper policies for the reduction of mortality, especially in

[3] The Preston curve is included in popular web-sites related to bio-medical sciences: for example it is possible to find an illustration of it in GANFYD (http://www.ganfyd.org/index.php?title=Main_Page), or in CHEMISTRYMATTERS (www.chemistrymatters.ca).

developing countries; there are scholars supporting the idea that economic growth, i.e. growth of per capita GDP, is the best way to reduce it, while, according to others, governments should implement more specific policies.

The debate on the policies derive also from the fact that, in the literature, there are not papers investigating, from a quantitative point of view, the nature and the strength of the forces determining the shape of the Preston curve.
This paper tries to fill, at least in part, this empirical gap. As somehow already anticipated, Preston himself, and, recently, Cutler, Deaton, Lleras-Muney (2006) list the possible determinants of life expectancy (and mortality rates), linked to three different broad classes: nutrition, sanitation, and medical treatments. In any case, "there is still debate about the relative importance of these different factors" (Bloom, Canning, 2007). Following the previous indications, I introduce proxies of these three broad categories of determinants, i.e. nutrition, bio-medical sciences, and sanitary conditions, in the estimation of the Preston curve, with and without the contribution of income.

It seems to me that I can reach a natural and meaningful solution to several of the problems, both conceptual and of economic policy, which arise in relationship to the Preston curve.

Data

As I have just stated, I will use several variables, related to per capita income, in place of it: I intend them as a "substitute" of it and representative of the three above mentioned categories.

All my data come from two sources, World Bank and Food Agriculture Organization, and cover the period from 1990 to 2012.

Basic data defining the original Preston curve, i.e. per capita GDP (PPP, constant 2011 international $), in symbols *ypc*, and life expectancy at birth, *life*, are both from WB-WDI (World Bank – World Development Indicators), retrieved in June 2014.

I then have 7 different new variables, two for nutrition (*calor* and *fat*), three for sanitary conditions (*sanit*, *urbsanit* and *water*), and two for the efficiency of the health and medicine related system (*phys* and *immun*). Specifically:

- *cal*: Dietary Energy Consumption (kcal per person per day), whose source is from FAO (retrieved in June 2014 from http://www.fao.org/fileadmin/templates/ess/documents/food_security_statistics/FoodConsumptionNutrients_en.xls)
- *fat*: Dietary Fat Consumption (grams per person per day), again from FAO (same link)
- *sanit* : improved sanitation facilities (% of population with access), from WB-WDI
- *urbsanit*: improved sanitation facilities, urban (% of urban population with access), from WB-WDI

- *water*: access to an improved drinking water source (% of population with access), from WB-WDI
- *phys*: physicians (per 1000 people), from WB-WDI
- *immun*: immunization, DPT (combination of vaccines against Diphteria, Pertussi and Tetanus (% of children ages 12-23 months), from World Bank - Health, Nutrition and Population Statistics

Data from WB are available, with some missing for different years, countries and variables, for the whole period from 1990 to 2012, whereas FAO data are available for many countries but only as 3-year averages of four specific periods: 1990-92; 1995-97; 2000-02; 2005-2007 (or 2006-2008).

Let me spend a few words on the variables. Data on nutrition and sanitation seem very general, and they should capture most of the wanted potential effects. On the contrary, the two variables related to medical treatments/system on the one hand show evident differences in space and time and cannot be ignored, on the other can be, though as only a small part of the role that medicine plays in this story; in particular, we should also consider, together with the static efficiency, the progress in the scientific frontier in medicine, but my variables completely fail in measuring this aspect. In order to partly overcome this limit I will introduce a deterministic time trend in the empirical estimations presented in the following sections.

I can add that I also looked for other data, in particular related to the class of medical treatments, which is the weaker, as previously explained. Specifically it would be possible to use, at least theoretically:

- number of beds for 1000 people and per-capita health expenses, both private and public (all variables form WB-WDI). The problem is that these data are available for a limited number of countries (beds) or years (expenses), impeding an analysis similar to that of the paper. Notwithstanding this evident limitation, I used them with a reduced sample, but they were always not significant, in all the many combinations and functional forms that I experimented. As a consequence I decided not to go on with them.
- in principle data (variables) on immunization against other human diseases are available: vaccine called BCG against tuberculosis; immunization against hepatitis B; Haemophilus influenzae type B vaccine; finally the vaccination against polio. Nevertheless, the first is available for a reduced number of countries and I preferred to maintain the largest possible dataset (moreover it has a strong, albeit not perfect, correlation with the others); the remaining are almost perfectly correlated with the one that I decided to use throughout the paper, so they are useless.

Finally a word about *fat*: it could be observed that it is a less general variable with respect to *calor*; my initial aim was to test if in this field non-monotonic relationships were present (in this direction: Fogel, 1994): i.e., a positive impact of *fat* on *life* for low and middle levels of nutrition, negative for very high levels (i.e. the problem of obesity and related questions). Nevertheless from my numerous attempts this kind of effect does not ever emerge, and *fat* behaves similarly to *calor*, without any non-monotonicity of the linkage with *life*. I will not show specific results related to this point, and throughout the paper *fat* (and *calor*) will be used only in monotonic forms.

In order to overcome the presence of missing, and with the aim to maximize the cross-sectional data availability, I initially averaged all data over two periods, that I will call in the following, with some approximation, decades: 1990-2000 and 2001-2012. For FAO data I averaged 1990-92 and 1995-97, i.e. 6 years, as a proxy for the first decade, and 2000-02 and 2005-2007 (or 2006-2008) for the second. With this procedure I get data for all the selected variables for 154 countries, without any missing. Later on, I will also provide results based on 5-year averages, but for a lower number of countries.

Throughout the paper, different periods will be identified by a suffix indicating them added to the variable names, so that *cal90* will be *cal* for the first decade and, analogously, *cal2000* for the second. In analogy, *lncal* will be the natural log of *cal*.

The basic features of my data are shown in table 1, containing some descriptive statistics.

table 1

descriptive statistics

1990s

	ypc	*Life*	*Phys*	*immun*	*sanit*	*urbsanit*	*water*	*cal*	*fat*
Mean	11040	65,1	1,3	79,1	63,9	73,3	80,1	2564,4	71,7
median	6227	68,1	0,9	86,5	73,0	81,5	87,0	2500,0	66,3
st.dev	14421	10,0	1,3	18,1	32,8	27,0	19,8	497,0	33,9
Max	107851	79,9	5,0	99,0	100,0	100,0	100,0	3615,0	163,5
Min	307	35,0	0,0	23,4	3,9	12,3	19,9	1550,0	13,0

2000s

	ypc	*life*	*Phys*	*immun*	*sanit*	*urbsanit*	*water*	*cal*	*fat*
Mean	13745	68,0	1,5	87,1	68,8	76,2	85,4	2699,0	77,9
Median	8271	71,4	1,1	92,6	80,0	86,3	92,3	2690,0	71,0
st.dev	15398	9,9	1,4	12,8	30,8	25,5	15,8	491,3	34,5
Max	89751	82,3	6,3	99,0	100,0	100,0	100,0	3795,0	167,5
Min	595	42,7	0,0	29,0	7,8	18,0	41,2	1555,0	12,0

Given the fact that the sample of countries is large, we have a lot of variability in the levels of the variables. If we look at the mean values in time, we see that all variables have increased; differently we can note that the standard deviation decreased for several of them, namely for all but *ypc* and *phys*.

All the selected "alternative" variables are positively correlated to per capita income, and this correlation is higher in the second period, as shown in table 2.

table 2

correlation with per capita income

	phys	*immun*	*sanit*	*urbsanit*	*water*	*cal*	*fat*
1990s	0,37	0,39	0,57	0,52	0,52	0,59	0,65
2000s	0,55	0,40	0,63	0,58	0,55	0,67	0,72

We can observe that variables related to nutrition have the highest correlation with income, variables related to medical treatments have the lowest, while sanitation variables are in between.

I believe that what is interesting and relevant is that the use of these "alternative" variables, in place of per capita income, simultaneously and naturally solves the problems of the income-life expectancy relationship highlighted above:

- first, because the causal direction is clearly univocal, since nutrition, sanitation and efficiency of the health system have a possible impact on life expectancy and not vice-versa (unless very minor effects that we can imagine)
- second, each variable provides a clear meaning to the "curve", i.e. they are meaningful determinants of the increase (or decrease) of life expectancy

Estimations and results: the benchmark

I will compare three types of estimations, whose names partially derive from the literature on the convergence hypothesis of the empirics of economic growth; so I have:

- the original Preston curve, that I call "unconditional Preston curve", i.e. estimation of an equation where simply *life = f(ypc)*;
- then I will propose an "alternative Preston curve", i.e. an estimation where *ypc* is not included among the explanatory variables and *life = f(X)*, where X is a set of new explanatory variables
- finally I will also propose the "conditional Preston curve", i.e. an estimation with *ypc* and other "conditioning" variables: life = *f(ypc, X)*

In order to have a benchmark for comparison, I initially present the result for the unconditional Preston curve.

I present three different forms of it: both variables in the original values (lin-lin), only for comparison, then both variables in natural logs (log-log) and a mix of them (lin-log); results are presented in table 3.
While in the literature it is common to use logs, in principle we could also employ a polynomial form. From visual analysis and from several econometric attempts, the polynomial form does not come out as a good

approximation for data, if compared to the logarithmic form which I will use throughout this paper, and I decided to abandon it.

Since in this paper I will follow a framework from general to more specific estimations, and I will start with the estimation of my "alternative curve" with a pooled OLS, I believe that it is useful to start also with pooled OLS estimations for the unconditional Preston curve just discussed: these results are presented in table 4, just after previous table 3 (where cross-section regressions are presented). As evident, and expected, results of the pooled OLS are quite similar to the estimations for the separated decades, in the sense of similar explained variability, similar levels of significance (of joint and single variables) , and similar coefficients values.

Table 3

Unconditional Preston curve

Cross section – 10 year averages

	life90	life90	lnlife90	life2000	life2000	lnlife2000
ypc90	0.000					
	(8.21)***					
lnypc90		6.947	0.113			
		(18.19)***	(16.59)***			
ypc2000				0.000		
				(9.59)***		
lnypc2000					6.535	0.101
					(16.07)***	(14.88)***
Constant	60.861	4.936	3.180	62.592	9.757	3.312
	(71.98)***	(1.48)	(53.20)***	(74.14)***	(2.67)***	(54.52)***
F statistic	67.45	331.05	275.34	91.95	258.13	221.49
Adjusted R-squared	0.30	0.68	0.64	0.37	0.63	0.59
num.obs	154					
num.obs.		154	154	154	154	154

* p<0.1; ** p<0.05; *** p<0.01

Table 4

Unconditional Preston curve

Pooled OLS – 10 year averages

	life	life	lnlife
ypc	0.000		
	(12.75)***		
lnypc		6.792	0.108
		(24.41)***	(22.46)***
Constant	61.660	6.868	3.238
	(102.87)***	(2.78)***	(76.13)***
F statistic	162.65	595.95	504.62
Adjusted R-squared	0.34	0.66	0.62
num. obs.	308	308	308

* p<0.1; ** p<0.05; *** p<0.01

All estimations show the positive relationship between the two variables, but while the lin-lin regression is, as expected, the worst, both log-log and lin-log have good results, with the latter providing results that are globally better in both decades (second and fifth columns of table 3), as well as in the pooled estimation (second column of table 4).

For this reason, which will be confirmed by further analysis, throughout the paper I will present only tables in which the dependent variable is in its original (linear) values, while I will have (some) logs in the RHS of equations.

Estimations and results: functional forms and relevant variables

As explained above, I intend to use my variables in place of *ypc*, at least initially, to understand if we can "explain" life expectancy with variables that can reasonably capture in detail the deep meaning of the original Preston curve.

The causal link between my explanatory variables and *life* is not demonstrated but somehow "assumed"; nevertheless the choice of the variables has been led by the conclusions of a serious and conspicuous literature based on long term historical evidence and analysis (as summarized in Cutler et al., cit.).

Initially I ran many simple regressions of what I call the "alternative" curve, where life was "explained" by some combinations of the new explanatory variables that I selected, using one variable of the same class at a time, but considering all classes simultaneously. In practice I had many possible combinations, also considering different functional forms. Results were somehow (positively) disturbing: I always got very good results, with all variables always highly significant and high goodness of fit (and better than in the unconditional curve).

The fact that I got similar results partly depends on the fact that variables within each class are correlated: I have to select which are the better, if there are any. Moreover, I have to choose among different functional forms.
I initially start from this last choice, and I proceed using two different tools: simple pairwise correlations between life and each one of the explanatory variables; cross validation analysis.

In table 5, correlations between life and the other variables are shown; in the first panel (*lin-lin*) all variables are in their original values; in the second (*lin-log*), life is as before, but the other variables are transformed in logs; finally in the third (*log-log*), all variables are in logs.

table 5

correlations with life

	phys	*immun*	*sanit*	*urbsanit*	*water*	*cal*	*Fat*
lin-lin							
1990s	0,64	0,73	0,84	0,82	0,81	0,74	0,71
2000s	0,68	0,64	0,83	0,82	0,75	0,73	0,70
lin-log							
90s	0,85	0,70	0,80	0,77	0,77	0,76	0,73
2000s	0,83	0,64	0,75	0,75	0,69	0,70	0,66
log-log							
90s	0,84	0,70	0,78	0,76	0,76	0,74	0,71
2000s	0,83	0,61	0,78	0,77	0,71	0,71	0,67

Some results should be stressed:

- correlation indexes are always high
- in the case of *phys* the non linear relationship with *life* seems preferable: differences in the correlation indexes of different forms are really strong (and also a visualization of a scatterplot easily confirms this non-linearity, differently from the case of the other variables)
- on the contrary, *sanit*, *urbsanit* and *water* seem to have a linear relationship with *life*
- for the remaining variables, a clear advantage of using one or another of the tested functional forms does not emerge

This outcome suggests that it would be inappropriate to simply compare estimations of the type lin-log and log-log, and that it is possible that a mixed form, in which some explanatory variables are introduced in logs and other in original values, would be better.

In order to make my choice I used, as test for the model selection, Cross Validation. In particular I used the routine provided by GRETL, which calculates the test by means of the so called LOOCV, or Leave-One-Out Cross Validation. Cross Validation is a test for non nested models; differently from other popular tests, like

the AIC and BIC, CV has no penalties for the loss of degrees of freedom. In my case I compare models with the same degrees of freedom.

I then ran several regressions, using variables pertaining to the same category in alternative: i.e., I alternatively introduce *cal* or *fat*, *phys* or *immun*, *sanit* or *urbsanit* or *water*, in linear or in log form.

From the bulk of estimations, in which different mix of variables and different functional forms are used, I derive some clear indications, partly confirming the impressions deduced from table 5, and precisely

- estimations using *lnphys* are always better (in terms of cross-validation and adj. R2) than the corresponding estimations (i.e., other variables being identical) where *phys* is used
- on the contrary, *sanit* and *urbasanit* always improve results in comparison with *lnsanit* and *lnurbsanit*
- there are no univocal indications for the other variables

In any case, I have to stress a couple of features: first, all variables in both forms (original values or logs) and in all combinations are always highly significant (let us say, in short, with "three stars"); second, adj R2 are always very high, ranging from around 0.79 to 0.82. This is to underline that there are no fundamental differences between different models.

Having decided which variables are to be introduced in a linear or nonlinear form, I pass on to the another step: the selection of the best variables, if there are any, within each of three categories (nutrition, sanitation, and medicine). For this purpose, I ran regressions, in which variables pertaining to the same broad class are used together in the RHS. Results are the following

- when I use *cal* and *fat*, in logs or not, *cal* always remains significant while *fat* does not.
- when I use *sanit*, *urbsanit* and *water*, in logs or not, only *sanit* remains always significant
- Finally, and differently from the previous two points, both *phys* and *immun* remain significant when they are used simultaneously

Summing up the previous two steps, in the end my "preferred" model is the model in which *life* depends on: *lnphys*, *immunm* (or *lnimmun*), *sanit*, *cal* (or *lncal*).

In addition to the chosen variables, I also introduce two more variables:

- first, a dummy for African countries (*africa*), which can be a proper procedure for several reasons (as suggested by results of Filmer and Pritchett, 1999, and Husain, 2011)[4].

[4] From a strictly econometric point of view, I also used other regional variables, for Latin America, Asia, and so on. None of them, except that for Africa, resulted as significant.

- besides, the pooled OLS structure allows me to introduce a deterministic time trend (*ttrend*). In this case, since I have only two periods, this time trend is very simple, just *t=1,2*. The reason for this is also that I would like to capture, even if roughly, the path of scientific progress in medicine.

Results of my pooled OLS estimations of the "alternative" curve are presented in the following table 6.

Table 6

The alternative Preston curve

Pooled OLS - 10 year averages

	life	life	life	life
africa	-6.662	-6.691	-6.682	-6.713
	(8.88)***	(8.90)***	(8.92)***	(8.95)***
ttrend	1.172	1.167	1.120	1.117
	(2.38)**	(2.37)**	(2.26)**	(2.26)**
lnphys	1.204	1.237	1.219	1.253
	(3.48)***	(3.58)***	(3.52)***	(3.63)***
lnimmun	4.580	4.871		
	(3.46)***	(3.68)***		
lncal	12.865		12.818	
	(6.82)***		(6.80)***	
sanit	0.069	0.067	0.065	0.063
	(4.18)***	(4.04)***	(3.83)***	(3.69)***
cal		0.005		0.005
		(6.80)***		(6.77)***
immun			0.076	0.081
			(3.52)***	(3.72)***
Constant	-58.554	28.492	-44.038	43.641
	(3.89)***	(5.13)***	(3.05)***	(19.45)***
F statistic	250.47	250.29	250.92	250.58
Adjusted R-squared	0.83	0.83	0.83	0.83
num. obs.	308	308	308	308

* p<0.1; ** p<0.05; *** p<0.01

There are two very evident features

- first, estimates are satisfying; variables used in place of *ypc* have the expected signs and are always strongly significant, F values are always very good, and adjusted R2 are sensibly higher than in the unconditional estimations, meaning that we can explain a higher degree of variability of *life* . This outcome depends only marginally on the presence of *africa* and *ttrend*: omitting them we only get a minor worsening of the estimation.
- second, results of different models are almost indistinguishable in terms of goodness of fit and significance (i.e., if I alternatively use *cal* or *lncal*, *immun* or *lnimmun*).
- finally, both *africa* and *ttrend* are significant and with the expected sign.

It could be interesting to understand what happens if we re-introduce *ypc* among the explanatory variables. This is done in the next step, whose results, the "Conditional Preston curve", are shown in table 7; the presence of *ypc* is the only difference from results presented in table 6.

Table 7

The conditional Preston curve 1990-2012

pooled OLS – 10 year averages

	life	life	life	life
africa	-6.799	-6.811	-6.823	-6.837
	(9.35)***	(9.34)***	(9.39)***	(9.39)***
ttrend	1.060	1.061	1.013	1.015
	(2.22)**	(2.22)**	(2.11)**	(2.11)**
lnphys	0.872	0.903	0.889	0.922
	(2.54)**	(2.64)***	(2.59)**	(2.69)***
lnimmun	4.949	5.153		
	(3.85)***	(4.01)***		
lncal	8.960		8.953	
	(4.45)***		(4.44)***	
sanit	0.039	0.038	0.035	0.034
	(2.24)**	(2.19)**	(1.97)**	(1.92)*
lnypc	1.865	1.852	1.848	1.837
	(4.58)***	(4.51)***	(4.54)***	(4.48)***
cal		0.003		0.003
		(4.35)***		(4.33)***
immun			0.081	0.084
			(3.87)***	(4.01)***
Constant	-43.818	16.975	-28.275	33.135
	(2.93)***	(2.85)***	(1.96)*	(10.35)***
F statistic	231.94	231.24	232.06	231.24
Adjusted R-squared	0.84	0.84	0.84	0.84
num. obs.	308	308	308	308

* p<0.1; ** p<0.05; *** p<0.01

We note that:

- The overall significance of the curve and the goodness of fit are substantially unchanged
- Most coefficients remain highly significant, albeit there is a marginal loss of significance for *ttrend* and *sanit*
- All coefficient values remain unchanged and, if they change, it does not happen dramatically
- Finally the most notable outcome is that, while *lnypc* is still significant, its coefficient is strongly reduced compared to the unconditional Preston Curve, passing from a value of 6.8 in table 4 (second column) to around 1.8 in table 7.

This last point, evaluating the impact of *ypc* on *life,* can be improved. Since, as I said, also the log-log regressions have good and almost comparable results, I use them in order to get constant elasticities. I do not present all the results, but I limit my attention to the following considerations: in the second decades (similar results to the first), in the unconditional Preston curve the elasticity for *ypc* was about 0.11 (see table 1, last column); in the conditional curve its value is reduced to at a level between 1/3 and 1/4 of the original value. This can be translated in an example: the minimum of *life*, in my sample, is 42,7 years in 2000s: with the unconditional estimation, an increase of 1% of *ypc* meant an increase up to 47.4 years (five years added); in the conditional estimation its effect is limited to 43,9 years (an addition of about 1 year).

SOMETHING ON ROBUSTNESS

The previous section already provides some indication of robustness, since I used different proxies and different functional forms with similar results.

In this section I follow this strategy: I "split" the whole sample in subsamples in three ways: first I separately run cross-sectional regressions for my two decades, both for the alternative and the conditional curves; then I calculated 5 year averages of all variables, obtaining a panel with four periods (but a lower number of countries); finally, I separately considered rich and poor countries.

Let us pass to the first step, where I present results for the 1990s and the 2000s. Obviously, with respect to previous estimates, I had to drop the *ttrend* variable. Table 8 shows results for the alternative curve.

Table 8

The alternative Preston curve

cross sections 1990s and 2000s

	life90	life2000
lnphys90	1.567	
	(3.34)***	
lnimmun90	4.964	
	(3.05)***	
lncal90	12.617	
	(4.90)***	
sanit90	0.046	
	(2.06)**	
africa	-5.893	-7.702
	(5.39)***	(6.81)***
lnphys2000		0.980
		(1.89)*
lnimmun2000		6.419
		(2.61)***
lncal2000		12.694
		(4.50)***
sanit2000		0.058
		(2.26)**
Constant	-55.435	-61.784
	(2.76)***	(2.52)**
F statistic	154.52	135.23
Adjusted R-squared	0.83	0.81
num. obs.	154	154

* p<0.1; ** p<0.05; *** p<0.01

This outcome, completely cross-section, is quite similar to the pooled estimations of table 6 (that, differently from here, also include *ttrend*); we note that, while the values of the parameters are sufficiently stable passing from the first period to the second, in 2000s there is an acceptable loss of significance for some of them (*lnphys*, *lnimmun*, *sanit*) and, in parallel, a slight decrease in the goodness of fit. This probably depends on the general process of "catching-up" at world level.

As before, I re-introduce now per capita income; in practice, I get again the "conditional" Preston curves but for both decades. Results of this step are shown in table 9, whose structure is perfectly overlapping with that of table 8, and the only difference is the presence of the log of *ypc* as an additional explanatory variable:

A relevant feature is that now *sanit* is not significant in both periods, and *lnphys* loses its significance in the second period.. The African dummy remains always strongly significant.

Table 9

The conditional Preston curve

cross sections 1990s and 2000s

	life90	life2000
lnphys90	1.217	
	(2.72)***	
lnimmun90	5.325	
	(3.48)***	
lncal90	7.626	
	(2.88)***	
sanit90	0.006	
	(0.24)	
lnypc90	2.431	
	(4.61)***	
africa	-6.050	-7.829
	(5.90)***	(6.97)***
lnphys2000		0.721
		(1.36)
lnimmun2000		6.639
		(2.72)***
lncal2000		10.087
		(3.25)***
sanit2000		0.038
		(1.40)
lnypc2000		1.231
		(1.93)*
Constant	-36.511	-51.880
	(1.90)*	(2.09)**
F statistic	149.91	115.40
Adjusted R-squared	0.85	0.82
num. obs.	154	154

* p<0.1; ** p<0.05; *** p<0.01

The overall capacity of the model to explain life does not change a lot, but, in any case, it is increased in the adj R2 values (compared to table 8). As evident, *ypc* comes out as again strongly significant in the 1990s, but only weakly in the second decade.

As a further step, and as anticipated, I passed to 5 year averages. I can work only with 4 periods: 1990-1994; 1995-1999; 2000-2004; 2005-2009. This implies two consequences:

- with this procedure, I miss the final years (2010-2012); this depends also on the fact that I have, as discussed previously, only limited information about nutritional variables, the last period for which is the 3 year average 2005-2007 in the case of *fat*, and 2006-2008 in the case of *cal*.
- second and obviously, the number of countries reduces sensibly, passing from 154 to 101.

In this section I will show that results worsen; nevertheless this is especially true when I simultaneously introduce deterministic space and time variables (i.e. country fixed effects, and *ttrend* or time fixed effects). Instead, if I introduce them separately, results show only minor changes.

As an example, if we run a pooled OLS similar to those based on ten year averages presented in table 7, but here based on 5 year averages (and a reduced number of countries), the conditional curve still appears effective and the results are quite similar to the previous ones: see table 11.

A word on the introduction of time: in principle we can chose a time dummy and/or *ttrend* as before (in this case *ttrend* = 1, ..., 4). I prefer the second option for two reasons: first in order to directly compare the new outcome to the previous analysis; second, because *ttrend* results, in my estimations, dominant on time dummies, when I introduce them together.

table 11

Conditional Preston Curve
Pooled OLS - 5 years averages

	life	life	life	life
africa	-6.655	-6.656	-6.768	-6.772
	(9.43)***	(9.41)***	(9.57)***	(9.56)***
ttrend	0.400	0.403	0.399	0.403
	(2.09)**	(2.10)**	(2.08)**	(2.09)**
lnphys	0.553	0.571	0.554	0.576
	(1.87)*	(1.93)*	(1.86)*	(1.93)*
lnimmun	2.765		3.158	
	(2.46)**		(2.82)***	
lncal	9.186	9.313		
	(4.82)***	(4.89)***		
sanit	0.059	0.058	0.058	0.058
	(3.89)***	(3.80)***	(3.85)***	(3.75)***
lnypc	1.839	1.816	1.841	1.819
	(5.10)***	(5.03)***	(5.01)***	(4.94)***
immun		0.040		0.046
		(2.22)**		(2.55)**
cal			0.003	0.003
			(4.52)***	(4.57)***
Constant	-36.783	-28.746	25.125	35.303
	(2.70)***	(2.15)**	(5.07)***	(14.07)***
F statistic	257.29	256.44	255.27	254.19
Adjusted R-squared	0.82	0.81	0.81	0.81
num. obs.	408	408	408	408

* p<0.1; ** p<0.05; *** p<0.01

It is easy to notice that there are no relevant changes compared to those of the corresponding table 7, only minor ones.

As mentioned, with this new structure of the dataset, based on 5 year averages, I can run a panel estimation adding country dummies to the pooled structure. Results of this this F.E. estimation, excluding, for the moment, *ttrend*, are presented in table 12

Table 12

conditional Preston Curve

Panel regression with fixed effects - 5 year averages

	life	life	life	life
lnphys	-0.048	-0.011	-0.059	-0.023
	(0.12)	(0.03)	(0.15)	(0.06)
lnimmun	4.068		4.229	
	(5.00)***		(5.28)***	
lncal	5.529	6.121		
	(2.46)**	(2.72)***		
sanit	0.059	0.061	0.061	0.065
	(2.02)**	(2.10)**	(2.13)**	(2.25)**
lnypc	3.979	3.845	3.937	3.815
	(6.68)***	(6.41)***	(6.49)***	(6.24)***
immun		0.061		0.063
		(4.52)***		(4.76)***
cal			0.002	0.002
			(2.36)**	(2.52)**
Constant	-33.370	-24.199	4.185	17.925
	(2.18)**	(1.54)	(0.84)	(4.18)***
F statistic	50.79	49.24	50.62	48.86
Adjusted R-squared	0.27	0.26	0.27	0.25
num. obs.	408	408	408	408

* p<0.1; ** p<0.05; *** p<0.01

While the F tests for the joint significance of the coefficients of country dummies, not showed in table 12, are always high (Prob > F = 0.0000), the main change is that *phys* comes out to be insignificant, while other variables, *immun* included, maintain their significance.

The reduction of adj. R2 and F values (still good) depends on the loss of degrees of freedom, perhaps signalling that the more parsimonious model without F.E. would be preferable to the panel model.

As anticipated, the simultaneous introduction of space and time deterministic variables produces some relevant changes

table 13

Conditional Preston Curve

FE + ttrend

	life	life	life	life
lnphys	-0.274	-0.233	-0.285	-0.246
	(0.72)	(0.61)	(0.75)	(0.64)
lnimmun	3.131		3.229	
	(3.88)***		(4.04)***	
lncal	2.650	3.295		
	(1.18)	(1.46)		
sanit	0.023	0.028	0.025	0.031
	(0.81)	(0.96)	(0.88)	(1.07)
lnypc	2.877	2.778	2.896	2.806
	(4.66)***	(4.47)***	(4.65)***	(4.47)***
ttrend	0.568	0.568	0.573	0.575
	(4.86)***	(4.74)***	(4.85)***	(4.75)***
immun		0.042		0.043
		(3.08)***		(3.22)***
cal			0.001	0.001
			(0.91)	(1.10)
Constant	4.256	10.011	22.232	32.787
	(0.26)	(0.60)	(3.65)***	(6.32)***
F statistic	49.45	47.70	49.27	47.40
Adjusted R-squared	0.32	0.31	0.32	0.30
num. obs.	408	408	408	408

* p<0.1; ** p<0.05; *** p<0.01

It is well evident, from table 13, that in this case something happens: several variables lose their significance.

Nevertheless, I add a couple of observations. First, I believe that this result does not diminish the scope and the meaning of the previous analysis: simply, the space and time variability of some of my variables is strongly correlated to deterministic space and time effects. Furthermore, I would like to recall what said a few lines before: the reduction of adj. R2, because of the loss of degrees of freedom, denotes that the introduction of FE do not add so much to the explicative strength of the model, and that the model without F.E. is perhaps preferable.

Finally, since the Preston curve is intrinsically not linear, we could expect stronger (weaker) linkages for poor (rich) countries. So, as a final proof, I divided countries in two groups: RICH (those with *ypc* above the average), and POOR (the remaining countries). To have an idea of what it means in my sample, consider that among RICH I have, in the low band, countries like Chile, Mexico and Turkey.

I limited the analysis to the pooled OLS based on ten year averages, in order to get sufficiently large subsets of countries; given the well known asymmetry of world income distribution, about 100 observations (50 countries) are included in the rich group, 200 (100 countries) in the poor one.
Results of this last step, not shown, can be easily summarized:

- goodness of fit and joint significance are higher in the case of the POOR subset, but in both cases satisfying;
- *lnypc*, remain significant for both groups, with a coefficient close to 1.7 for POOR and 1.5 for RICH
- *ttrend* and variables of the sanitation class are not significant for the POOR,
- *ttrend* is highly significant for the RICH (differently from what was found for the previous point)
- *phy* and *immun* lose their explanatory power in the RICH subsample.

In conclusion some surprises, but on the whole a confirmation of previous results with some changes.

Among those changes, slightly surprising are: the similar role for *ypc*; the loss of significance of the variables related to sanitation for the POOR.

Instead, expected were: the role of *ttrend* exclusively in the RICH group, if it measures, in some way, the role of technological progress; the loss of significance for *phys* and *immun* in the RICH group[5], finally, the overall better performance of the estimations in the POOR case.

CONCLUSIONS

Notwithstanding some weaknesses in the robustness section, I believe that this paper provides sufficiently firm and clear evidence of the existence of an "alternative" formulation of the Preston curve: alimentation, sanitation, and medicine together seem good "explanations" of the role of income as a determinant of life expectancy. This does not eliminate a role for income itself, at least if considered within the limits of the dataset that I have used.

One relevant feature of my results is that the use of those variables naturally solves most of the problems highlighted in the literature, as already stressed: there are no problems of endogeneity, since causality is reasonably only one-directional; the used variables are not proxies of something else, as in the case of income, but are themselves clearly meaningful.

It should be added that one strong and rather paradoxical result, with respect to my initial purposes, is that *ypc* appears as the most robust variable; in any case, even if income remains significant in all estimations, its impact on life expectancy is largely reduced when compared to the result obtained in the unconditional curve.

These results, if confirmed, could have some relevant policy implications. As stressed initially, one field of disagreement among scholars is about sound policies for improving life expectancy in poor countries: according to some of them policies for an increase of per capita income are sufficient, as suggested by the original Preston curve, while others believe that better results can be achieved with policies specifically directed to nutrition, etc.

[5] *Phys*: consider than in RICH countries many medical resources are dedicated to activities not linked to diseases, like plastic surgery. *Immun*: many countries already at 100% of coverage.

I chose my variables exactly because they are correlated to income; we then expect that, in a long run perspective, an increase in per capita income is intimately related to improvements in nutrition, sanitation and of the health system. Nevertheless, it is also true that a government has the possibility to redirect its policy efforts from some areas to others; as an example, it can reduce military expenses, or in some kind of infrastructures, etc., not to speak of the possible results that could be reached in poor countries redirecting resources for corruption to those sectors. If my results are correct, they support the idea that it is possible to make some positive steps (increase of life expectancy) through policies explicitly directed to nutrition (probably of specific classes of people), sanitation and medicine. Moreover, if we bear in mind the bi-directional causality between life expectancy and per capita income, it is possible that those specific policies positively contribute to a general process of economic growth.

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