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Is There a Link Between Pharmaceutical Consumption and Improved Health in OECD Countries?†

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Abstract

Objective: The objective of this study was to determine whether there is a measurable health return associated with high pharmaceutical consumption in a sample of developed countries.

Design and setting: The study focused on the production of health, disaggregating healthcare into pharmaceuticals and other healthcare. We controlled for wealth and lifestyle factors. The sample consisted of 21 Organization for Economic Cooperation and Development (OECD) countries and the measure of pharmaceutical consumption used was the best available for a *large* number of OECD countries. We proxied health with life expectancies at birth, at age 40, and age 60.

Main outcome measures and results: Pharmaceutical consumption had a positive and statistically significant effect on remaining life expectancy at age 40 and 60 years (significant at the 0.10 and 0.05 level, respectively, based on a 2-tailed test), although the effect on life expectancy at birth was small and not significant. Sensitivity analysis showed that these results were generally robust. A significant effect of pharmaceutical consumption on infant mortality was not demonstrated and results of the infant mortality model were very sensitive to small changes.

Conclusions: Increased pharmaceutical consumption helps improve mortality outcomes, especially for those at middle age and older.

Introduction

There have been many international studies of healthcare, especially in member countries of the Organization for Economic Cooperation and Development (OECD). Mostly driven by budgetary and cost-containment problems, the vast majority have focused on the determinants of healthcare expenditures. While the emphasis on healthcare spending is not entirely misplaced, more effort should be devoted to estimating the determinants of health itself. Such estimates can guide policy makers to most effectively allocate resources both among different types of healthcare goods and services and between healthcare and other goods. Only a small number of researchers from a variety of fields (economics, epidemiology, sociology and anthropology) have studied the effects of different factors on the production of health on an aggregate level. The results of these studies have been mixed and many of them have been flawed.

Our research focuses on the productivity of healthcare, with special attention paid to disaggre-

† These results are reported in greater detail in H.E. Frech III and Richard D. Miller.[1]

gating healthcare into pharmaceutical and other healthcare consumption. Pharmaceutical consumption varies considerably, even among the rich OECD countries. For instance, France's per capita pharmaceutical consumption was 5 times that of Denmark in 1990, while Italy's was roughly twice that of the US. At the same time, the conventional wisdom, especially among economists, is that the marginal return to healthcare consumption in general, and pharmaceutical consumption in particular, is negligible. This raises an obvious question: Are those people in high pharmaceutical consuming countries, e.g. France, acting irrationally or is there a measurable health return to this consumption?

To answer this question we analyse data from a sample of 21 OECD countries as of the early 1990s. Measuring pharmaceutical consumption is surprisingly tricky, but the measure we use here is the best one available for a large number of OECD countries. We measure each country's health objectively, if crudely, using life expectancies at birth, at age 40, and at age 60, along with infant mortality. The analysis consists of various multivariate regressions.

Previous Research

Certain results of previous studies appear strong and make sense. First, public health services, such as the provision of a potable water supply and sanitation services, provide the biggest payoffs in decreased mortality, for all age groups. These services are a matter of civil engineering rather than healthcare. This result has been found by all researchers who have studied underdeveloped countries, where there is sufficient variability in these public health infrastructure variables. Another striking and consistent result is that the expansion of healthcare services does not improve mortality rates to nearly the same extent. As previously stated by Stewart,[2] less developed countries should train more sanitation engineers and worry about training doctors only after the basic public health infrastructure is in place.

Environmental factors and per capita income have also been found to have a much higher impact on mortality than medical care.[3] Higher levels of education tend to be negatively related to mortality. Dietary factors have also been found to be important. Richer diets tend to decrease mortality from infectious diseases. At some point, though, mortality from degenerative diseases such as heart disease increases as diets become too rich. This is also generally true of income. At low income levels, higher income is associated with lower mortality rates. At higher income levels (in the most developed countries) higher income is associated with higher mortality rates, at least when education is controlled for. Studies have also found that variation in alcohol and cigarette consumption can explain variation in mortality. Finally, two studies have suggested that the political environment may play a role in determining mortality.[4,5] Generally, variation in medical care consumption has not been found to explain variation in mortality rates to the extent that the environmental variables have. This has led to the formation of the conventional wisdom that medical care itself does very little to improve mortality and that environmental factors are much more important.

While most studies have found little effect of increased medical care consumption on population mortality rates, a few good ones have found such consumption improves mortality. Hadley[6] examined variation in mortality across county groups in the US and found that increased medical care consumption was associated with decreased mortality for older cohorts. Similar results were demonstrated for younger cohorts in an earlier analysis.[7] Zweifel and Ferrari[8] found that increased medical care consumption has a positive effect on life expectancies for older individuals.

Very few studies have dealt with the effects of pharmaceutical consumption on mortality, either directly or indirectly. Those that have dealt with this relationship directly have had serious flaws. In a study that is closely related to ours, Babazono and Hillman[9] used OECD data to investigate the effects that different components of medical care

expenditures have on perinatal and infant mortality as well as male and female life expectancy. In a 1988 cross section of 21 countries, the authors found that per capita pharmaceutical expenditures have no effect on these basic health measures. They also found that total healthcare spending per capita and inpatient and outpatient utilisation are not related to health outcomes. This study has many substantial flaws. First, the researchers blindly used the OECD data 'as is', without giving critical thought to the difficulties inherent in comparing measures of different variables across countries and health systems. For example, the authors included a variable for average length of hospital stays in different countries without considering that hospitals serve quite different functions in different countries. In Japan, very basic convalescent homes are considered hospitals, whereas in the US they are not. Thus the authors compared average lengths of stay in very different types of institutions. Another flaw with the study is that the authors arrived at their final results using a stepwise regression analysis which results in misleading statistical inference.

Perhaps the most serious flaw, however, was that drug consumption was badly mismeasured in this study. The authors used per capita pharmaceutical expenditures, converted to US dollars by using gross domestic product (GDP) purchasing power parity (PPP) exchange rates. As we will see in the next section, these economy-wide PPPs provide very inaccurate measures of relative drug prices. The use of these PPPs led to mismeasurement of real drug consumption. Finally, the functional form used in the analysis is flawed, since it does not allow for diminishing marginal productivity of pharmaceutical consumption in the production of health.

In one of the better studies that has dealt with the effect of pharmaceutical consumption on mortality, Peltzman[10] looked at the effects of pharmaceutical regulations on national health indicators. He found that mandatory prescription laws are positively related to mortality from poisonings. This may reflect perverse effects of the regulations or it may only reflect reverse causation. Also, various micro-level studies,[11,12] and many studies of restricted formularies in the US,[13,14] have provided evidence that pharmaceutical consumption has a positive impact on health. It is our goal to investigate whether this effect can be found in a careful study of international aggregate data.

Data and Methodological Issues

OECD Data

We used data from the 1996 release of the OECD's Health Data database. The data include measures of various types of national per capita healthcare consumption as well as broad health system outcomes measures such as life expectancies and infant mortality. They also include information on various macroeconomic indicators such as GDP, education and employment. Finally, they include measures of environmental factors which are expected to affect health, such as alcohol and tobacco consumption as well as dietary make-up.

Health Indicators

As our measure of the performance of each country's healthcare system, we used life expectancies as of 1993 at the following ages: at birth, at age 40, and at age 60. We also used infant mortality as an alternative measure of this performance. According to the Office of Technology Assessment (1993) there appear to be no serious problems with the data from industrialised countries on life expectancy. Caution is prudent in using infant mortality in international comparisons, even among the richest countries. For example, physicians in the US are more likely to resuscitate extremely premature and low birth weight infants, who later die. These are classified as live births in the US and thus included in infant mortality calculations. Other countries are more likely to classify such births as fetal deaths.

Measuring Pharmaceutical Consumption

How one converts a nation's per capita pharmaceutical expenditures to US dollars for the purpose

Table I. Comparing measures of real pharmaceutical consumption by pharmaceutical purchasing power parity (Pharm PPP) and gross domestic product purchasing power parity (GDP PPP) exchange rates, 1990

Rank	Country	Pharm PPP	GDP PPP	Difference
1	France	560.927	256.278	304.649
2	Italy	448.200	242.235	205.965
3	Germany	374.138	311.483	62.655
4	Belgium	304.466	193.561	110.904
5	Spain	286.618	144.749	141.870
6	Portugal	247.092	153.211	93.881
7	US	231.000	231.000	0.000
8	Sweden	225.859	119.700	106.159
9	Norway	216.341	125.180	91.161
10	Greece	216.032	95.128	120.904
11	Canada	215.650	190.769	24.881
12	Australia	196.386	117.266	79.120
13	New Zealand	194.828	140.373	54.455
14	Austria	191.940	154.345	37.595
15	Finland	190.663	121.630	69.033
16	Switzerland	190.476	145.455	45.021
17	UK	183.721	131.667	52.054
18	The Netherlands	130.189	127.189	3.000
19	Ireland	120.690	101.449	19.241
20	Denmark	112.846	95.421	17.425
21	Turkey	62.115	34.869	27.246

of cross-national comparisons is of crucial importance. One could use PPP exchange rates designed to convert total GDP to US dollars. This approach is appropriate only if pharmaceutical prices differ across countries in the same way that prices differ in general. Researchers who have looked at this issue in depth, including Szuba[15] and Danzon,[16] have demonstrated that this is far from the truth. Drug price regulation remains a national prerogative in many countries and trade barriers have traditionally been significant. Both price regulation and barriers vary widely. For instance, biased (in favour of domestic producers) price regulation is practised in France and Italy. Other OECD countries, such as the UK and Germany, also regulate pharmaceutical prices, albeit indirectly and typically much less stringently. The US and Denmark, at the other extreme, generally permit free pricing of pharmaceuticals, subject to market forces. For these reasons one might expect GDP PPP exchange rates to be unsatisfactory for converting pharmaceutical expenditures to US dollars for cross-national comparisons.

Luckily, PPP exchange rates designed specifically for converting pharmaceutical expenditures to US dollars are available for 1980, 1985, 1990 and 1993 in the OECD database. Table I compares measures of per capita pharmaceutical expenditures converted to US dollars using pharmaceutical PPP exchange rates and GDP PPP exchange rates for 1990. One should note that conversions using the GDP PPP exchange rates invariably underestimate actual pharmaceutical expenditures outside the US.

Danzon and Percy[17] argue that even the pharmaceutical PPP exchange rates provided by OECD are flawed and these investigators provide more accurate price indexes for a handful of countries to convert pharmaceutical expenditures to US dollars. These relative price measures should be regarded as the 'gold standard', as they are undoubtedly the most accurate. However, they are not available for any countries other than France, Italy, Germany and the UK. Szuba[15] also painstakingly assembled price ratios using detailed proprietary data,

though with a slightly different approach. His price coefficients are also excellent, when available.

Table II compares measures of real pharmaceutical expenditures for 1985 using the following conversion factors: market exchange rates, GDP PPPs, pharmaceutical PPPs, Danzon and Percy's Fisher price indexes and Szuba's price coefficients. Again we find differences, but a general pattern emerges. France seems to significantly outspend the other countries with Italy and Germany at the next tier, with expenditures significantly higher than those in the US. Switzerland and the UK tend to consume fewer pharmaceuticals than the US no matter which measure is used.

Table II also presents correlations among the different measures of real pharmaceutical expenditures for 1985. The measure that we use in our study is very highly correlated with both the Danzon and Percy measure and the Szuba measure for the countries for which all of the measures are available. Note that the measures that use simple market exchange rates and GDP PPP exchange rates are not highly correlated with the Danzon and Percy or Szuba measures. The analyses described above, along with conversations with officers at the OECD, lead us to believe that using the pharmaceutical PPP exchange rates is a significant step forward from earlier work which used the GDP PPP exchange rates. Since we wish to study more than the 5 countries for which Danzon and Percy provide price indexes, the pharmaceutical PPP exchange rates provided by OECD, however imperfect, are the best conversion factors available.

Therefore, our measure of pharmaceutical consumption is the 1985 per capita pharmaceutical expenditure for each country, converted to US dollars using the PPP exchange rates provided in the OECD database. Similarly, we construct a measure of healthcare consumption in 1985 using healthcare specific PPP exchange rates.

Other Explanatory Variables

We control for per capita income (measured as per capita GDP) and other lifestyle factors. The lifestyle factors we control for are tobacco use, alcohol consumption and richness of diet. Tobacco use is measured as the percentage of the population who smoke. Alcohol consumption is measured as litres consumed per capita. Finally, we measure dietary richness as the consumption of animal fat calories per capita.

The Model

We use regression analysis to determine the effect of each of our explanatory variables on each of our life expectancy measures and infant

Table II. Comparing measures of real pharmaceutical expenditures for 1985 in 6 countries, using various conversions to US dollars

	Exchange rate	GDP PPP	Pharm PPP	Danzon & Percy's Fisher price index[17]	Szuba's price coefficients[15]
Country					
France	129.48	176.36	401.38	387.16	556.2
Italy	94.30	148.00	269.96	258.97	457.06
Germany	174.15	229.60	257.29	290.83	256.79
Switzerland	102.44	115.07	135.84	NA	147.81
US	150.00	150.00	150.00	150.00	150.00
UK	66.67	94.55	119.85	103.44	126.01
Correlations among the various pharmaceutical consumption measures					
Exchange rate	1.000	0.988	0.527	0.686	0.225
GDP PPP		1.000	0.574	0.728	0.313
Pharm PPP			1.000	0.979	0.924
Danzon & Percy				1.000	0.851
Szuba					1.000

GDP PPP = gross domestic product purchasing power parities; **NA** = not applicable; **Pharm PPP** = pharmaceutical purchasing power parities.

mortality. We lag the explanatory variables by roughly 10 years (by using health indicators from 1993 and pharmaceutical and healthcare consumption measures from 1985) because we believe that lifestyle factors and medical care consumption have a cumulative rather than an instantaneous effect on health. As an example of a cumulative effect, smoking does not kill people immediately, but rather over a period of years. A full model of this general type would require several lags of each explanatory variable. Because of data and sample size limitations we include only one lag here. First, there are only 21 countries in our sample. Adding a large number of lagged explanatory variables would lead to a decrease in precious degrees of freedom. Second, the PPP conversions for healthcare and pharmaceuticals are available only for 1980, 1985 and 1990. Thus, we would only be able to include additional lagged terms for 1980 and 1990. Including lags from the 1960s or 1970s might be worthwhile, but the necessary data do not exist.

Finally, we use a double-log, or constant elasticity, functional form. There are a couple of reasons for this. First, a coefficient from a double-log regression is interpreted as an elasticity: the percentage change in the value of the dependent variable associated with a 1% change in the value of an explanatory variable. Second, we desire a model that allows for diminishing returns to all of the independent variables. In the double-log model the elasticity is held constant and the absolute value of the marginal effect of each explanatory variable is forced to fall at higher and higher values of the explanatory variable. The data to which one applies such a model determines the rate at which the absolute marginal effect decreases.

Empirical Results for Life Expectancy

The key empirical results for life expectancy are reported in table III. In these regressions life expectancies at birth, at 40 years of age and at 60 years of age are analysed for our full sample of 21 OECD countries.

Originally, regressions were run for each sex at each age separately and the results indicated that observations could be pooled across sexes. To test for this, we pooled observations for each age-specific life expectancy across sexes and included a dummy variable for female life expectancy to allow the intercept to differ by sex. As part of the test, interaction terms were included to allow the slope coefficients to vary by sex. They were not statistically significant as a block. The only interaction term that was significant individually in any of the pooled regression was the interaction of the female dummy and alcohol consumption. We therefore decided to pool across sexes including this interaction. It should be noted that the ln(SMOKE) variable is equal to ln(FEMSMOKE) for those observations on female life expectancy and equal to ln(MALESMOKE) for those observations on male life expectancy.

Effect of Pharmaceutical Consumption

The results in table III indicate that pharmaceutical consumption is surprisingly productive. It has positive and statistically significant relationships with the life expectancies at the ages of 40 and 60 years (significant at the 0.10 and 0.05 level, respectively, based on a 2-tailed test). Doubling pharmaceutical expenditures would increase life expectancy at 40 by roughly 2% and life expectancy at 60 by about 4%. For example, a typical 60-year-old male living in a country with the average life expectancy at age 60 could expect to see his remaining life expectancy rise from 18.5 to 19.2 years. A typical 60-year-old female living in a country with average life expectancy at age 60 could expect to see her life expectancy rise from 22.5 to 23.5 years. These results are sharply different from those found in the flawed study by Babazono and Hillman,[9] where it was found that pharmaceutical consumption had no significant effect on life expectancy in OECD countries. At the same time, they are consistent with the results of others who have found, in micro-studies and in studies of restricted formularies, that pharmaceutical consumption is associated with lower mortality rates and better health outcomes.

Table III. Constant elasticity regression results of life expectancy for full sample (t statistics in parentheses)

Regressor	Dependent variable: life expectancy		
	at birth	age 40y	age 60y
CONSTANT	–0.5344[b]	–0.0256	–0.8954
	(–1.7130)	(–0.0370)	(–0.8130)
FEMALE	0.0392[a]	0.0996[a]	0.1369[a]
	(4.2480)	(5.4290)	(5.1200)
ln(PHPC)	0.0050	0.0172[b]	0.0401[a]
	(1.0860)	(1.7400)	(2.6950)
ln(GDPPC)	0.0121	0.0572[a]	0.0876[a]
	(0.8310)	(2.1470)	(2.3190)
ln(HEPC)	0.0052	–0.0111	–0.0145
	(0.7820)	(–0.8270)	(–0.7290)
ln(SMOKE)	–0.0071	–0.0102	0.0020
	(–1.1120)	(–0.7460)	(0.1020)
ln(ALCOHOL)	–0.0093[a]	–0.0143	–0.0188
	(–2.3890)	(–1.6230)	(–1.3410)
ln(ALCOHOL) × FEMALE	0.0167[a]	0.0150[a]	0.0312[a]
	(4.8500)	(2.1060)	(2.9850)
ln(ANFAT)	1.4040[a]	0.9548[a]	0.9096[a]
	(13.7640)	(4.1660)	(2.5180)
ln(ANFAT)-squared	–0.1045[a]	–0.0728[a]	–0.0706[a]
	(–13.3550)	(–4.1710)	(–2.5690)
R-squared	0.9621	0.9308	0.9290
Adjusted R-squared	0.9515	0.9113	0.9090
Shapiro-Wilkes p value	0.7068	0.5968	0.5310
Sample size	42	42	42

a Significant at the 0.05 level based on a 2-tailed test.

b Significant at the 0.10 level based on a 2-tailed test.

ANFAT = animal fat consumption per capita; **GDPPC** = gross domestic product per capita; **HEPC** = nonpharmaceutical healthcare consumption per capita; **PHPC** = pharmaceutical consumption per capita.

Figure 1 presents the marginal effect of drug consumption on male and female life expectancies at age 60 and at age 40 in France, Italy, the US and Ireland. We chose these 4 countries because they represent, respectively, a very high use country, a high use country, an average use country and a low use country. The marginal effects are measured in additional days of life expected per additional US dollar spent on pharmaceuticals in 1985. The most interesting result here is that France and Italy (the biggest consumers of drugs) stand to gain the least by marginally increasing their drug consumption. On the other hand, the beneficial effect of increased drug consumption is much higher in a low drug consumption country like Ireland, where the effect is roughly 5 times greater than in France. In countries like the US, where drug consumption is near the average, the effect is higher than in high consumption countries, but lower than in the low consumption countries.

The fact that the countries which spend the most on pharmaceuticals show the smallest marginal gains in life expectancies is determined by the functional form used in our analysis. The double-log model forces the marginal effect to decrease as pharmaceutical expenditure levels increase. The log-log functional form implies that doubling drug consumption would cut the marginal effect in half. Attempts to empirically test the extent of the flattening of the curve by adding a second order term for drug use were unsuccessful. The fact that the productivity of drugs (and other inputs) diminishes

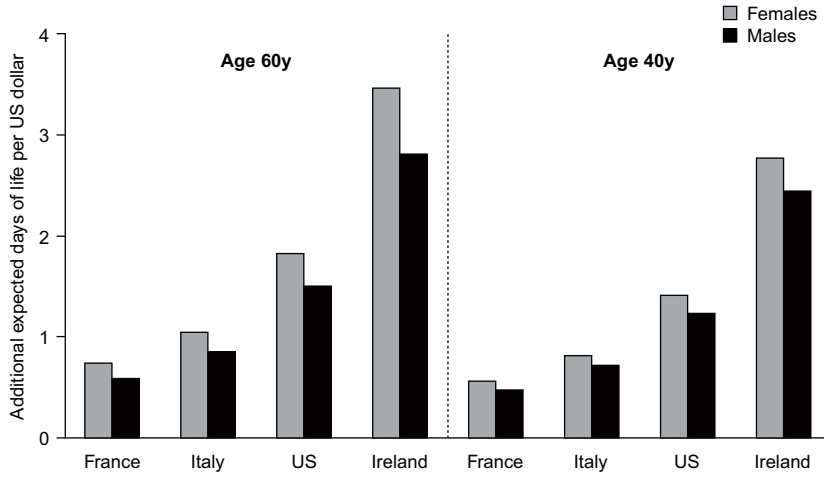

Fig. 1. The marginal effect of pharmaceutical consumption on life expectancy at ages 60 and 40 years, in selected countries. Additional expected days of life per US dollar spent on pharmaceuticals in 1985.**((←Author: the second sentence has been added so the figure can 'stand on its own' as per our house style. Please approve or amend))**

at high use levels makes excellent sense theoretically and fits the data well. We can be less certain of how rapidly diminishing returns set in. Still, it is clear that big spenders on pharmaceuticals like France and Italy could cut back on pharmaceutical consumption with less effect on life expectancy than the low spenders such as Ireland.

Figure 1 also illustrates that the marginal effect of pharmaceutical consumption on life expectancy increases with age. For instance, the results indicate that in the US a dollar spent on pharmaceuticals would increase life expectancy for a 40-year-old male by 1.2 days, and would increase life expectancy for a 60-year-old male by 1.5 days. The same pattern follows in all countries and for both males and females. Furthermore, the marginal effect of pharmaceutical consumption is greater for females than for males. In Ireland, a dollar spent on pharmaceuticals would increase life expectancy for a 60-year-old female by 3.5 days and for a 60-year-old male by 2.7 days. Again, the same pattern follows in all countries for life expectancies at all ages. Figure 2 presents estimates of the lifetime per capita pharmaceutical expenditures necessary to extend life by 1 year, by age and by gender. We again consider the same 4 countries. The estimates are fairly conservative because they are based on the assumption that pharmaceutical expenditures are constant at 1985 levels over the entire lifetimes of the individuals. These results are highly sensitive to assumptions made about the histories of pharmaceutical consumption in each country.

Expressed in US dollars per life-year, figure 2 is commensurate with analyses for new drugs or technologies. Of course, the results here tell the same story as those in figure 1. Again, the highest expenditures are necessary in France and Italy, the lowest are necessary in Ireland, and the US is somewhere in between. The costs are also higher for 40-year-olds than for 60-year-olds and higher for males than for females. Again, the magnitudes reflect our conservative assumptions. More realistic assumptions about actual lifetime drug use will lead to lower estimates of cost per additional year of life.

Effects of Other Factors

Wealth, as measured by lagged GDP per capita, has a positive effect on life expectancy, although the effect is significant only for life expectancies at 40 and 60 years of age (table III). The results indicate that doubling GDP would increase life expec-

tancy at 40 years by roughly 6% and at 60 by roughly 9%. These are large increases, and not terribly surprising, since economic growth has been associated with rising life expectancies over the last 2 centuries. These results are consistent with most previous studies, the most notable exceptions being those by Auster et al.[3] and Zweifel and Ferrari.[8] In these 2 studies, increased per capita wealth was associated with higher mortality rates and lower life expectancies, respectively.

Nonpharmaceutical healthcare consumption has virtually no effect on life expectancy at birth and a slight negative effect on life expectancy at the ages of 40 and 60 years, although the relationship is not statistically significant in any of the analyses (table III). The negative relationship is quite small. The elasticity is never greater in absolute magnitude than –0.0145. Our data reject any large effect of either sign. Ours is not the first study to find a weak negative relationship between life expectancy and healthcare consumption, especially among developed countries. Our result could indicate that developed countries are on the flat of the curve when it comes to nonpharmaceutical healthcare consumption or it could simply reflect a bias due to the endogeneity of healthcare consumption and a nation's health.

It appears that the lifestyle variable that has the greatest effect on life expectancy at all ages is the consumption of animal fat (table III). The results indicate that consumption of animal fat has a very strong and large positive effect on all 3 life expectancy measures at low levels of consumption, and this effect is estimated quite precisely. The negative coefficient on the second order term indicates that this effect falls in magnitude as consumption of animal fat increases, and even goes negative at a high enough level of animal fat intake. At low levels of fat consumption, enriching a diet is beneficial to health but, at some point, further consumption of fat decreases life expectancy. This is a sensible result given that clinical researchers have found excessive fat intake to be linked to degenerative diseases such as heart disease and cancer.

The point estimates also indicate that smoking has a small negative effect on life expectancy at birth and at age 40 and no effect on life expectancy at the age of 60 (table III). However, this is not very precisely estimated.

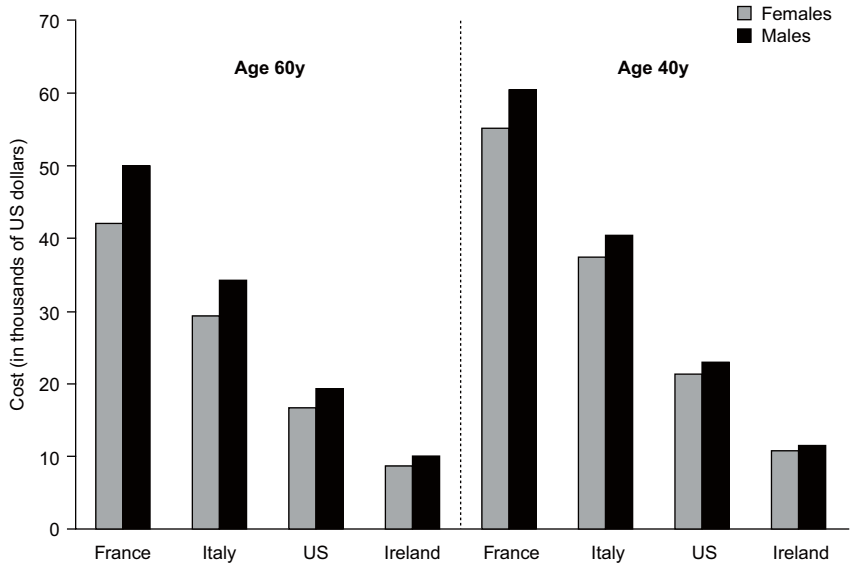

Fig. 2. Lifetime cost ($US) of extending life by 1 year at ages 60 and 40 years, in selected countries **((Author: in 1985?))**.

Alcohol consumption has a negative and statistically significant effect on male life expectancy at birth and a nearly statistically significant negative effect on male life expectancy at age 40 (table III), but the magnitudes of the effects are small, indicating that doubling alcohol consumption would lower life expectancy by at most only 1.5%. This is somewhat surprising, given the epidemiological research showing that moderate (up to 3 drinks a day) drinking substantially reduces the risk of heart disease.[18] The interactions between alcohol consumption and the female dummy variable indicate that alcohol consumption has roughly no effect on female life expectancies. This most likely reflects a difference in alcohol consumption between women and men.

Sensitivity of the Results

We tried a number of different variants of our model to test for sensitivity to small changes. For one, we re-estimated the models, excluding Turkey, because Turkey is quite different from the rest of the countries in our sample in such key measures as wealth, pharmaceutical consumption and life expectancy. Doing this had no meaningful effect on the results. We also ran regressions after restricting the sample to the European countries. The results, again, were very similar to those from our original inclusive regressions, the main difference being that the effect of pharmaceutical consumption was bigger and the effect of wealth was smaller. We also tried a model in which we did not lag the explanatory variables, but in which they were contemporaneous with the life expectancies. Again, the results did not change meaningfully. Finally, we included other factors that may help determine life expectancy, but none appeared satisfactory.

Empirical Results for Infant Mortality

Table IV presents results from the analysis of infant mortality. Two constant elasticity regressions are presented. In the first one, infant mortality in 1990 is explained by contemporaneous levels of the regressors. In the second regression it is explained by values of the regressors which have been lagged by 5 to 7 years (as in the life expectancy regressions). We present both specifications for 2 reasons. First, the *a priori* case for lagged regressors in explaining infant mortality is not as strong as it is for life expectancy. Second, unlike in the case of life expectancy, the choice of contemporaneous versus lagged regressors makes a big difference in the results.

Effect of Pharmaceutical Consumption

In each regression, pharmaceutical consumption per capita has a small positive effect on infant mortality. The effect is very small and not significant in the contemporaneous regression, while it is larger and marginally significant in the lagged regression, where the t statistic is 1.56 (table IV). The point estimate seems to indicate that, after controlling for wealth, other healthcare consumption and lifestyle factors, increased pharmaceutical consumption may actually increase infant mortality, although the contemporaneous regression results indicate that pharmaceutical consumption has no effect on infant mortality. The result is not very precise. Given the usual standard for statistical inference, we can not rule out the possibility that there is as much as a 2% negative effect of pharmaceutical consumption on infant mortality.

Effects of Other Factors

The results of the effects of wealth on infant mortality are mixed. GDP per capita has the expected negative effect on infant mortality in the contemporaneous regression. In the lagged regression, however, it has a small insignificant positive effect on infant mortality (table IV). The results in the contemporaneous model indicate that a doubling of GDP per capita would cut infant mortality by nearly 50%. The average country would see a fall in infant mortality from roughly 10 infant deaths to only 5 infant deaths per 1000 live births. The results in the lagged regression indicate that, for the average country, a doubling of GDP would slightly increase infant mortality, raising it by roughly 3%. Again, note that this effect is imprecisely measured.

Table IV. Constant elasticity regression results for infant mortality in 1990 (t statistics in parentheses)

Regressor	Specification	
	contemporaneous	lagged 5 to 7years
CONSTANT	53.7210*	51.390*
	(11.463)	(17.225)
ln(PHPC)	0.0026	0.0753
	(0.0300)	(1.5610)
ln(GDPPC)	–0.4943	0.0307
	(–1.7900)	(0.1880)
ln(HEPC)	0.1642	–0.1899*
	(0.9770)	(–3.2360)
ln(FEMSMOKE)	–0.0294	0.1654
	(–0.1460)	(1.6690)
ln(ALCOHOL)	–0.0233	–0.0246
	(–0.2670)	(–0.6970)
ln(ANFAT)	–13.800*	–14.350*
	(–9.589)	(–13.770)
ln(ANFAT)-squared	0.9915*	1.044*
	(9.4370)	(12.911)
R-squared	0.9436	0.9659
Adjusted R-squared	0.9132	0.9475
Shapiro-Wilkes p value	0.3814	0.1493
Sample size	21	21

ANFAT = animal fat consumption; **GDPPC** = gross domestic product per capita; **HEPC** = nonpharmaceutical healthcare consumption per capita; **PHPC** = pharmaceutical consumption per capita;
* Indicates significant at the 0.05 level based on a 2-tailed test.

The results are also mixed on the effect of nonpharmaceutical healthcare. In the contemporaneous regression the effect is positive and insignificant, whereas in the lagged regression the effect is negative and significant (table IV). The result from the lagged regression indicates that doubling healthcare consumption would lower infant mortality in the average country from roughly 10 deaths per 1000 live births to roughly 8 deaths per 1000 live births.

Lifestyle factors appear to be major determinants of infant mortality. In both regressions reported in table IV one can reject the hypothesis that all 4 of the lifestyle coefficients are zero. The amount of animal fat calories consumed per capita per day is significantly negatively related to infant mortality in both regressions, although the positive coefficient on the second order term indicates that the beneficial effect of increased animal fat consumption diminishes as more and more is consumed. Animal fat consumption actually becomes harmful at consumption levels above 1053 calories in the contemporaneous regression and above 965 calories in the lagged regression. The results would make sense to epidemiologists. The diseases which are most likely to kill infants are those for which a rich diet may be protective. The bad effects of a diet that is too rich would most likely be due to issues pertaining to maternal behaviour at the prenatal stage.

The results for smoking are more problematical. In the contemporaneous regression, female smoking appears to have a very small negative and statistically insignificant effect on infant mortality (table IV). The result is quite imprecise. In the lagged regression, female smoking has a strong, marginally statistically significant positive effect on infant mortality, which is consistent with previous studies. While the more reasonable result from the better-fitting lagged regression is com-

forting, the result from the contemporaneous regression is troubling. This is so because of the general belief that smoking during pregnancy leads to birth defects and low birth weight. Perhaps there is a subtle selection problem here. Smoking during pregnancy may lead to poorer fetal health, and thus more miscarriages and stillbirths. But perhaps there is not much effect on infant mortality for fetuses that survive to a live birth.

The results on alcohol consumption are not mixed. In both regressions alcohol consumption has a slight, although statistically insignificant, negative association with infant mortality (table IV).

Sensitivity of the Results

Generally, the infant mortality model was much less successful than the life expectancy models. Most results were sensitive to small, defensible changes to our model. The infant mortality results must be counted as suggestive at best.

Conclusions

In this study we have estimated a production function for health which is a function of pharmaceutical consumption, other healthcare consumption, GDP and 3 lifestyle measures: alcohol consumption, the percentage of the population which smokes, and the richness of diet. PPP exchange rates for pharmaceuticals and healthcare were used to transform pharmaceutical expenditures and healthcare expenditures to US dollars. The dependent variables were life expectancies at birth, at age 40, and at age 60, and infant mortality.

In a sample of relatively rich, developed countries, we found that pharmaceutical consumption has a positive and significant (both statistically and economically) effect on remaining life expectancy at age 40 and at age 60 (significant at the 0.10 and 0.05 level, respectively, based on a 2-tailed test). It has a small, positive and statistically insignificant effect on life expectancy at birth. The elasticities of pharmaceutical consumption on life expectancy are roughly 0.017 at age 40 and 0.040 at age 60. The estimates are not very sensitive to small changes in the basic statistical models.

A significant effect of pharmaceutical consumption on infant mortality was not found. Taken at face value, one of the models even suggests that, controlling for lifestyle factors, increased pharmaceutical consumption may be related to slightly increased infant mortality. Unfortunately, the results of the infant mortality model were very sensitive to small changes, which does not inspire much confidence in infant mortality as a health output measure, at least not in this data set. This measurement problem also affects life expectancy at birth to some extent.

Our work should add to the discussion over how and whether governments in developed countries should regulate the pharmaceutical industry. It improves on much of the existing literature in that it uses better measures of pharmaceutical and other healthcare consumption and uses a functional form that allows for diminishing returns. The results have been surprising, but they have also been fairly robust. The final conclusion is that increased pharmaceutical consumption helps improve mortality outcomes, especially for those at middle age and beyond.

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