Title
The Productivity of Health Care and Pharmaceuticals: An International Comparison

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Publication Date
1996-12-01

I. Introduction

There have been many international studies of health care, especially in the OECD (Organization for Economic Cooperation and Development) countries. Mostly driven by budgetary and cost-containment problems, the vast majority have focused on the determinants of health care expenditures. The researchers have found that income, usually measured as gross domestic product, is a major determinant of health expenditures, with richer countries spending far more than poorer countries. Many have found results consistent with an income elasticity of greater than one, indicating that health care may be a luxury good in rich countries.[1] There have been mixed results on the impact of economic institutions and policies on health expenditures.

The emphasis on estimating the effects of different factors on health care spending is not entirely misplaced, but more effort should be devoted to estimating the determinants of health. This is so because such estimates can help guide policy makers in how to most effectively allocate resources both among different types of health care goods and services and between health care and other goods. A small number of researchers from a variety of fields, including economics, epidemiology, sociology and anthropology, have considered the effects of different factors on the production of health. The results of these studies have been mixed and many of them have been flawed. We devote Section II of this paper to a detailed description of this literature.

The particular focus of this research is the production of health, with special attention paid to disaggregating health care into pharmaceutical and other health care. We focus on the productivity of pharmaceuticals in particular because pharmaceutical consumption varies considerably, even among the rich countries of Europe, North

[1] We do not provide a detailed review of this literature since it is not the focus of our research. We do direct interested readers to papers by Joseph Newhouse (1977), and David Parkin, Alistair McGuire and Brian Yule (1987), and two papers by Ulf-Gerdtham and Jonsson (1991, 1992).

America, and Oceana. For instance, in 1990, France's per capita consumption of pharmaceuticals was five times that of Denmark, and Italy's was roughly twice that of the United States. It is also a common belief that, among rich countries, the marginal return to health care consumption in general, and pharmaceutical consumption in particular, is negligible. Taken together, this leads to the belief that many rich countries, such as France and Italy consume too many pharmaceuticals (*New York Times*, 1991; *Washington Post*, 1988). The popular press has even accused doctors in countries such as Japan of over-prescribing drugs (*The Economist*, 1996). The question is: Are those societies which consume more pharmaceuticals acting irrationally or is there a measurable health return to such consumption?

To attempt to answer this question a sample consisting of twenty one OECD countries as of the early 1990s is analyzed. Measuring pharmaceutical consumption is surprisingly tricky, but the measure of pharmaceutical consumption used here is the best one available for a large number of OECD countries. Each country's health is measured crudely, but objectively, using life expectancies at various ages (birth, age 40, and age 60), along with infant mortality. The data and methodological issues concerning measurement of variables of interest are discussed below in Section III.

The analysis consists of various multivariate regressions. By analogy to other production processes, the productivity of health should exhibit diminishing returns in each of its inputs. Thus, a functional form is used in the regression analysis that allows for this. The explanatory variables in each regression include pharmaceutical consumption, gross domestic product, other health care consumption, and three lifestyle variables: alcohol consumption, cigarette consumption, and richness of diet. The results indicate that pharmaceutical consumption is significantly positively related with increased life expectancy at the ages of 40 and 60, even when controlling for the above factors. The elasticities of pharmaceutical consumption for life expectancies at these ages are 0.017 and 0.040 respectively. Doubling pharmaceutical consumption would raise life expectancy by

about three years for those already 60. If the analysis is limited to Europe, the positive effect is even larger. The results also indicate that richness of diet is very important, but that returns to enriching a country's diet can become negative. A more detailed description of the analyses are provided in Section IV. We conclude and make additional remarks in Section V.

II. A Review of the Literature

II.1 Overview

There have been a number of studies that bear on the productivity of medical care in improving health, and even a few focusing particularly on the productivity of drugs themselves. The quality, purpose and approach of the studies varies greatly. In our review, we will focus on studies that relate most closely to our own study. This means studies that examine the production of health by geographic region and especially by country and also the very few studies looking at the productivity of drug consumption.

II.2 Aggregate studies of the Production of Health

Areas within the United States. In an important early study of the production of health using cross-sectional data from the United States, Richard Auster, Irving Leveson, and Deborah Sarachek (1969), considered the relationship between mortality and both medical care and environmental variables.[2] They used a regression analysis of state aggregates for 1960. Medical care was measured in two ways: first, by total per capita medical care expenditures, and second, by the output of a Cobb-Douglas production

[2] The sample was limited to whites, mostly for sample size reasons.

function, combining the services of physicians, other medical personnel, capital, and drugs. The environmental variables included per capita income, education levels, percent of population in urban areas, percent employed in manufacturing, alcohol consumption per capita, percent in white collar occupations, percent of females not in the labor force, and the presence of a medical school in the state. The authors presented results from both an ordinary least squares regression and from two stage least squares models where medical spending was treated as endogeneous.

The key result of this study is that the environmental variables are much more important in determining the age-adjusted death rate than medical care is. The authors found education to be negatively associated with mortality rates in all regressions, while they found per capita income to be positively associated with mortality rates. The authors believed that the latter result may have been due to the unfavorable diet, the sedentary and generally unhealthy lifestyle, and the greater psychological stress presumably associated with higher income. This negative partial effect of income on health is sometimes called the "fastlane lifestyle effect." The authors claim that this could explain the United States's poor performance in reducing mortality. Neither of the medical care measures significantly affected the age-adjusted death rate in the two-stage models.

In two studies, Jack Hadley (1982, 1988) estimated a series of health production functions using aggregate, U.S. cross-sectional data for 1970 and 1980. In the first study, he used the county group as the geographic unit of analysis[3] and focused on age-sex-race specific mortality rates for eight adult and four infant population cohorts. Data came from the 1970 Census and from special mortality files created under the sponsorship of the National Center for Health Statistics. Three approaches to defining medical care use were investigated: (1) the ratios of physicians, nurses and acute care hospital beds per 1,000

[3]County groups are defined by the Census Bureau and, in 1970, consisted of one or more whole counties with a minimum population of 250,000 people. The county groups are built to conform to local economic and market patterns and an individual county group can encompass counties in more than one state. As of 1970 there were over 400 county groups as defined by the Census Bureau.

people in the county group, (2) cohort-specific estimates of these ratios, and (3) Medicare expenditures per Medicare enrollee in the county group. Hadley found the third measure to be the best on both theoretical and empirical grounds. He estimated that, except for middle-aged males, a 10 percent increase in per capita medical care use is associated with about a 1.5 percent decrease in mortality rates. He also found that education is negatively related with mortality rates. For infants, he found that a 10 percent increase in total family income would lower infant mortality by between 1.6 and 2.2 percent (except for black male infants). He found the effect of total family income on adult mortality to be more ambiguous.

Hadley (1988) replicated his earlier study using 1980 data and focusing on the elderly. He used the redefined county group as the geographic unit of analysis.[4] For whites, he found that a 10% higher level of Medicare spending per beneficiary was associated with mortality rates that ranged from 2.53 to 4.40 percent lower, depending on age and sex. The impact on African-American mortality was even greater, usually at least twice as great as for whites. The age cohort which experienced the greatest impact was the 70-74 year old age group, regardless of race or sex. Not surprisingly, he also found that higher Medicare spending had the least impact on deaths from external causes, such as accidents and murder.

International Studies. One early analysis of *international* data to determine the productivity of health care was conducted by Charles Stewart (1971). He divided the resources that are devoted to health into four categories: treatment, prevention, information, and research. Life expectancy was used as a dependent variable and was regressed upon treatment variables (such as numbers of medical personnel and numbers of hospital beds), literacy rates (a proxy for information), and the availability of potable water (a proxy for prevention). The study was a cross section analysis considering all of the

[4]For 1980, county groups were much larger, consisting of one or more whole counties with a minimum population of 1 million.

nations in the Western Hemisphere as of the mid 1960s. Stewart found literacy and the availability of potable water to both be significantly related to life expectancy, but found that none of the treatment variables mattered. Stewart notes that mortality rates are typically understated, and that the extent of the understatement is greatest in the least developed countries. Also literacy rate differences are probably understated in the data, because literacy standards tend to be lower in those countries with lower literacy rates.

Despite these misgivings about the data, Stewart comes to two general conclusions about the United States and its less developed neighbors. First, the United States seems to be on the flat of the curve as far as the productivity of health care is concerned. In other words, the marginal effect on mortality of any resource devoted to prevention, information, or treatment is small in the U.S. The best bet may be to increase research and development to improve the productivity of resources devoted to treatment. As far as the less developed countries of the Western Hemisphere are concerned, all health resources should be devoted to prevention. More sanitary engineers and fewer doctors should be trained in these countries. This is consistent with historical experience (Fuchs 1974: 31-53).

A. L. Cochrane, A. S. St. Leger, and F. Moore (1978) studied the relationship between various health service inputs and various mortality measures using a cross section of eighteen developed countries as of 1970. They limited their sample to those countries in which per capita GNP was at least roughly $2000 in 1970 and where the population was greater than 2 million persons. They also excluded Japan since they were concerned that genetic factors may account for a substantial proportion of any difference in mortality between it and the other, mostly European countries, that comprised the final sample. They do both simple correlations and regression analyses. In the regression analyses they regress maternal, infant and perinatal mortality, along with mortality rates for various age groups, on the number of doctors, per capita GNP, cigarette consumption, alcohol

consumption, population density, the percentage of health care provided by public funds, and sugar consumption.

In general, the results of the study are mixed. First, they find that a greater number of doctors is related to *higher* mortality rates in most age groups and for mothers and infants. This is even true of pediatricians and their effect on infant, perinatal and maternal mortality. They also find per capita GNP to be negatively correlated with all mortality measures. Cigarette consumption appears to be positively correlated with all types of mortality although it is only significant in the regressions for infant and perinatal mortality. The results for alcohol consumption are more mixed, as it has a positive effect on infant mortality (significant) and maternal mortality (insignificant), but has a negative, albeit insignificant, effect on mortality for those older than thirty five. Finally, the percentage of health care provided through public funds has a negative, though insignificant effect on mortality, except for those between the ages of 15 and 35, where the effect is significant.

Many studies have found per capita income a powerful variable in explaining certain measures of a society's health level. G. B. Rodgers (1979) included a measure of income inequality, the Gini Coefficient, to determine what effect it has on life expectancy at birth, life expectancy at the fifth birthday, and infant mortality across countries. It makes sense that income inequality would influence health, holding average income constant, because of strongly diminishing returns to the income/health relationship. In his sample of fifty six countries he finds that greater income inequality is associated with lower life expectancies both at birth and at the age of five and is associated with higher infant mortality rates. In his regressions he includes only per capita income and the Gini Coefficient. Still, his analysis has the advantage that he accounts for the decreasing returns of economic development, as measured by income per capita, in the production of health. He tries a number of different functional forms and his results are fairly robust. His basic result is that the difference in life expectancy between a fairly egalitarian society and a relatively nonegalitarian society is likely to be as much as five to ten years. It would be

interesting to see if this result were robust to the inclusion of environmental and health care spending variables.

Barbara Wolfe (1986) and Barbara Wolfe and Mary Gabay (1987) explore the hypothesis that lifestyle changes must be included to capture the true relationship between medical expenditures and health status in international comparisons. They consider 22 developed countries over a 20 year period. In the second paper, a simultaneous equation model is used. Changes in lifestyle, aging of the population and changes in occupational risk are modeled as influences on medical expenditures. In the second stage, changes in medical expenditures and changes in lifestyle are modeled as determinants of the change in health status. They find a positive relationship between changes in real medical care expenditures and changes in life expectancy (although these relationships are not statistically significant at the usual levels).

The lack of statistical significance could be due to an over-taxing of the data. Doing a two stage estimation in first differences was probably too much for such a small data set to bear. First differencing alone may result in mostly noise. See Anand and Ravallion (1996). Further, two stage least squares behaves badly when the first stage fits poorly (Startz and Nelson 1990). Although the first stage is not reported, we suspect that it does fit poorly.

In the study most similar to ours in its focus on OECD countries, Peter Zweifel and Matteo Ferrari (1992) considered what they called the Sisyphus syndrome in health care. The idea is that increased medical care expenditures tend to prolong life, especially at advanced ages, and that there is a positive feedback in that more consumers at advanced ages then drive up demand for health care and drive up expenditures. From a cost-containment perspective, the developed countries may be victims of their own medical care successes. Zweifel and Ferrari attempted to find evidence of this syndrome in aggregate data from the OECD countries.

The result of most interest to us concerns the first link in the Sisyphus syndrome. Zweifel and Ferrari found that there is a positive relationship between total health care expenditures and life expectancy beyond the age of 40 even when controlling for per capita gross domestic product, a result which is similar to Hadley's (1988). They found this result in a regression where they lagged both health care expenditures and per capita gross domestic product by ten years. Another interesting result that supports the "fastlane lifestyle" idea is the finding that lagged gross domestic product had a negative and significant effect on life expectancy, echoing Auster, Leveson and Sarachek (1969).

In an attempt to estimate the positive feedback relationship, Zweifel and Ferrari found that remaining life expectancies beyond 40 were not statistically significantly related to health care expenditures regardless of the specification or estimation method chosen. They conclude that the Sisyphus syndrome does not exert enough force to be detected in this data.

Sudhir Anand and Martin Ravallion (1993) examined the nature of the relationship between per capita real income and two measures human development which we also consider: life expectancy and infant mortality. Their sample consisted of a cross section of twenty two poorer, developing countries as of the mid 1980s. Three possible explanations are presented for the commonly found relationship between per capita income and health. First, capability to produce health is expanded through economic growth directly. Second, this capability is expanded through poverty reduction.[5] Finally, capability to produce health is expanded through expanded social services, particularly medical services.

To determine which of these explanations is most powerful, Anand and Ravallion first regressed life expectancy on per capita income alone and find that there is a strong and positive correlation between these two variables. They then added the proportion of

[5]This approach follows Rogers (1979) and flows from the general idea of diminishing returns to income in the production of health.

the population consuming less than $1 per day in 1985, to control for poverty, and a measure of public health spending per person. When they did this, the correlation between life expectancy and per capita income disappeared, while the coefficient on per capita public health spending was positive and significant and the coefficient on the poverty measure was negative and significant. They concluded that this is evidence that average affluence matters to the extent that it leads to lower poverty level and pays for better public health services. They then explained Sri Lanka's impressive record of progress in measured health, despite being a poor country, as a result of its high public health expenditures.

Howard Wall (1996) argues that Anand and Ravallion's results likely are due to the fact that they did not control for fixed effects, such as diet, culture, and location, that are correlated with their regressors. Wall ran similar regressions using panel data from two years in the early 1980s on twenty five developing countries using country dummy variables to control for the fixed effects. He found that differences in per capita public health expenditure do not affect the positive relationship between per capita income and life expectancy. At the same time, he found that per capita income has only a tenuous effect on life expectancy, as the t-statistic is only 1.74 (which is statistically significant at the 95 percent level, based on a one-tailed test) in the fixed-effects regression including only per capita income and a year dummy.

This tenuous effect of income on life expectancy leads Anand and Ravallion (1996) to reply that Wall's result is most likely due to measurement error bias, which tends to be more problematic in fixed-effects models. Measurement error in the regressors in fixed-effects models will tend to bias coefficient estimates toward zero, which could explain the lack of explanatory power in per capita income in Wall's regression. It could also explain the lack of explanatory power of per capita public health spending, a variable which is likely to be measured with more error than per capita income.

Still, Anand's and Ravallion's analysis could be improved. First, they put much emphasis on *public* health expenditure per capita, but they never investigate whether their measure of public health expenditure is acting merely as a proxy for *total* (public + private) health expenditure. There is also data available on nutritional factors (see Gage and O'Connor, 1994) which could be included. Over all, it seems that public health expenditures should have an impact in poorer countries, since the public sector provides those health services (sanitation, clean water, etc.) that have the biggest impact on health in poorer countries, but making the above improvements would make their argument stronger.

Sam Peltzman (1987) found similar effects of income distribution in a study which focused on the effects of pharmaceutical prescriptions. He chose 22 middle-income countries with per capita incomes between 5 percent and 50 percent of the United States' level. He examined the poisoning death rate and the infectious disease death rate for people between the ages of 20 and 64. The most interesting results follow.

First, median income is negatively related to mortality, but holding the median constant, a higher mean income (greater income inequality) implies higher mortality. This result is quite similar to Rodgers's (1979) and Anand and Ravillion's (1995), using different measures of income inequality. Second, the only pharmaceutical regulation effect that was significant was a strong positive relationship between such regulation and the poisoning death rate. Perhaps this results from forcing consumers to use balck market drugs and perhaps it results from reverse causation. Mandatory prescription requirements also appeared to have a positive effect on the demand for doctors, and for some illnesses this indirect effect appeared to improve health. He found no direct effect of regulation on the degree to which consumers realize benefits from pharmaceutical utilization.

Finally, a broad measure of government intervention, government health expenditures per dollar of GNP, appeared, if anything, negatively related with life expectancy. Peltzman finally points out the sharp contrast between the effect of regulation

and government intervention (bad) and the effect of diffuse economic development (good). This is exacly the opposite of the results and interpretation of Anand and Ravallion (1993) who argue that economic development leads to better health through increased spending on public health services. It should be noted that Peltzman studied more developed countries, where the very productive public investments in sanitation and clean water were more or less complete.

Political scientists Edmund Wnuk-Lipinki and Raymond Illsley (1990) examined the experience of health and inequality in the former Eastern Bloc countries of Bulgaria, Hungary, Poland, and the Soviet Union. In simple cross-correlation and regression analyses, they found evidence that dysfunctions of these health systems were only minor contributing factors in the poor performance in these countries in mortality and life expectancy. The more fundamental causes of this poor performance were cultural and collective behavior and the priorities of the political and economic systems of these countries. Again this is evidence of the importance of lifestyle and environmental factors in yet another set of countries.

Another example of work in this area by non-economists is that of Hugh Lena and Bruce London (1993), who investigated the impact of selected political and economic processes on the well-being of populations within samples of fifty to eighty four peripheral and noncore (sociological term for developing) nations. More specifically, the authors studied the impact of regime ideology, state strength (as measured by government expenditures as a proportion of GNP), multinational corporate penetration, and position in the world economy, on infant mortality, child death rate, and life expectancy. Even when controlling for per capita gross national product, the authors found that political systems matter in determining health and well-being. High levels of democracy and strong left wing regimes are associated with positive health outcomes and strong right wing regimes are associated with lower life expectancies and higher mortality levels. This could be

proxying for more egalitarian income distributions, in which case the results are consistent with Rodgers's (1979), Anand and Ravallion (1995) and Peltzman's (1987).

Erica Hertz, James Herbert and Joan Landon (1994) used data from United Nations sources to conduct an international comparison of infant and maternal mortality rates and life expectancy at birth. Their sample consisted of a cross section of 66 countries representing all levels of economic development. Given that the sample included both developing and rich countries it is not surprising that they found that availability of sanitation facilities showed the strongest association with all three dependent variables, lowering both infant and maternal mortality and raising life expectancy. They also found total literacy rate, total calorie consumption and dietary composition to be important determinants of health. Medical care resource availability variables such as availability of medical care personnel and number of hospital beds per capita did not significantly affect any of the dependent variables. These results are quite similar to those found by earlier researchers, such as Stewart (1971) and Auster et al (1969) in the economics literature. A major flaw with the study is the use of stepwise regression techniques, which undermines statistical inference and interpretation (see Greene, 1993).

Timothy Gage and Kathleen O'Connor (1994) examined the associations between nutrition and mortality at the national level. Again, their sample included both developing and rich countries. The results indicate that life expectancy is positively related to total calories, the overall quality and quantity of diet, and the ratio of fats to protein. The ratio of carbohydrates to fats is positively associated with life expectancy. Higher ratios of fats to proteins are healthy initially, but the effect is reversed when diets are rich. All the effects of nutrition diminish considerably at high nutrient availability. To an economist this is not surprising since it indicates that increased diet quality exhibits diminishing returns in the production of health.

This finding also reflects what is known as the epidemiological transition. The epidemiological transition is the shift from infectious to degenerative causes of mortality.

As a population's wealth increases, more resources can be made available for sanitation and clean water, and diets can become richer. At lower income levels, richer diets decrease mortality from infectious pathogens such as parasites and bacteria. At higher income levels, diets may become too rich and this would lead in increase in the number of cases of heart disease, cancer and strokes, which are leading causes of mortality in richer countries. Also, degenerative diseases are more likely to strike at advanced ages, so the epidemiological transition is partially a byproduct of the success in fighting off infectious diseases, and thus living to advanced ages. A flaw in the study of Gage and O'Connor is that income and other environmental variables such as education levels and the availability of clean water and sanitation are not included. Thus the decrease in mortality which is attributed to the nutritional factors, may be at least partially due to these other factors. The study does indicate that nutrient availability may be important, at least for developing countries.

The Productivity of Drugs Across Nations. Akira Babazono and Alan Hillman (1994) used OECD data to investigate the effects that different components of medical care expenditures have on perinatal and infant mortality and male and female life expectancy. This work is closely related to ours because it examines the effects of drug use. In a 1988 cross section of twenty one OECD countries, the authors found that per capita pharmaceutical expenditures have no effect on these basic health measures. They also found that total health care spending per capita and inpatient and outpatient utilization 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 primitive convalescent homes are considered hospitals whereas in the U. S. 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 leads to misleading statistical inference.

Perhaps most serious, drug consumption was badly mismeasured in this study. The authors used per capita pharmaceutical expenditures, converted to U.S. dollars by using gross domestic product purchasing power parity exchange rates. As we will see in the next section, these economy-wide purchasing power parities provide very inaccurate measures of relative drug prices. The use of these purchasing power parities 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.

Other Studies of the Productivity of Pharmaceuticals. Other researchers have looked at micro-level data to determine the productivity of pharmaceuticals. Frank Lichtenberg (1996) analyzed the effects of changes in the quantity and types of pharmaceuticals prescribed by doctors on rates of hospitalization, surgical procedures, and mortality. He obtained data on drugs prescribed by physicians from the 1980 and 1991 National Ambulatory Medical Care Survey (NAMCS) Drug Mentions files. In order to analyze the relationship between changes in the pattern of drug utilization and changes in other medical inputs and mortality, he computed disease-level aggregate statistics from six additional sources, including the NAMCS 1980 and 1991 patient files, the 1980 and 1992 National Hospital Discharge Survey files, and the 1980 and 1991 Vital Statistics-Mortality Detail files. He found that for those diseases where there were the greatest increases in both quantity and novelty of pharmaceuticals, the number of hospital stays, bed-days, and surgical procedures declined most rapidly. He also found that increases in pharmaceutical quantity and novelty are associated with reductions in both the number of hospital deaths and deaths per hospital stay. Nonhospital mortality is unaffected as is the mean age of death.

A number of studies have also investigated the effects of restricted formularies imposed upon the Medicaid population by some U.S. states. In the U.S. Medicaid program, the federal government does not require the states to cover prescription drugs and thus states can select which drugs to reimburse patients for and which drugs not to reimburse them for. Under restricted formularies, states limit the availability of pharmaceuticals to their Medicaid populations (Grabowski, 1988). As of 1990, twenty states had such restricted formularies.

William Moore and Robert Newman (1993) used a multivariate regression model to analyze pooled cross sectional state data for the years 1985-89. They estimated the effect of restricted formularies on total Medicaid expenditures, Medicaid prescription drug reimbursements, and Medicaid reimbursements made for other types of health care. Both state and various Medicaid program characteristics were controlled for. The results indicate that the formularies decrease expenditures on prescription drugs (by 13 percent), but leave total Medicaid expenditures unchanged. This is due mostly to a 33 percent increase in mental patient hospital expenses and a 25 percent increase in physician services expenses. This indicates that prescription drug consumption is productive in the Medicaid population and that without access to the drugs Medicaid patients must go to other sources to preserve their health stock.

In two other studies, Stephen Soumerai and colleagues (1991 and 1994) considered the effects of formularies on the elderly and on patients with schizophrenia. In the first study they analyzed thirty six months of Medicaid claims data from New Hampshire, which had a three-drug limit per patient for eleven of those months, and from New Jersey, which had no restrictions. The study patients were over 60 years of age. Survival and time series analyses were conducted to determine the effect of the restrictions in New Hampshire on admissions to hospitals and nursing homes. They found that when the restrictions were instituted, drug use fell by thirty five percent and that this decrease was associated with an increase of admissions to nursing homes. No changes were

observed in the New Jersey comparison group. When the restrictions were discontinued, the excess risk of nursing home admission ceased. This result is very similar to Moore and Newman's and can be interpreted similarly.

In the second study by Soumerai et al (1994), the authors again investigated the effects of the restrictions in New Hampshire, but this time they focused on a population of schizophrenic patients. The study and comparison patients were permanently disabled, non-institutionalized, and aged nineteen to 60 years of age. The restrictions resulted in reductions in the use of all psychotropic drugs (by fourteen to 45 percent) and increases of one to two visits per patient per month to community mental health centers. Sharp increases in the use of emergency mental health services and partial hospitalization also occurred. After the restrictions were discontinued the use of medications and mental health services reverted to the level they had been at before. Again, this is evidence that pharmaceuticals are important in producing health.

II.4 In summary

Where does this literature survey leave us? Certain results appear very strong and that make perfect sense. First, basic public health services, in the form of a potable water supply and sanitation services, provide the biggest payoffs in decreased mortality, for all age groups. These services are matters of civil engineering, not health care. This result has been found by all researchers who have studied underdeveloped countries, and thus have introduced sufficient variability in these public health infrastructure variables. Another striking and consistent result is that the expansion of medical care services does not improve mortality rates to anywhere near the extent that public health infrastructure development does. As Stewart (1971) commented, the best thing for the less developed countries to do would be to 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 been found to have a much greater effect on mortality than medical care. Higher levels of education are negatively related to mortality. Dietary factors have been found to be important, as richer diets tend to decrease mortality from infectious diseases, although at some point the rate of deaths from degenerative diseases begins to increase as diets become too rich. This is also true of income. At low income levels, increases in income tend to be associated with lower mortality rates. At higher income levels (in the most developed countries) income is positively related to 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 that variation in medical care utilization can not. Finally, a couple of studies have suggested that the political environment may play a role in determining mortality.

While most studies have found little effect of more medical care on population mortality rates, the best studies have found small negative effects. Hadley (1988) and Zweifel and Ferrari (1992) found such a relationship for older cohorts for areas within the U.S. and across countries. Hadley (1982) also found such a relationship for younger cohorts in the U.S., although he measured the health care consumption of older people.

As far as the effect of pharmaceutical consumption on mortality rates is concerned, very few studies have dealt with this either directly or indirectly. Those studies which have dealt with this relationship directly have had serious flaws. In one of the better studies, Peltzman (1987) considered the effects of pharmaceutical regulations on national health indicators and 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. The poisonings may have been the reason why such regulations were enacted in the first place. The micro study by Lichtenberg (1996) and many studies of restricted formularies in the United States have also provided evidence that pharmaceutical consumption has a positive impact on health. It is our goal to investigate whether this effect can be found in an international comparison study.

III. Data and Methodological Issues

III.1 The data

The data which is analyzed here were secured from the Organization for Economic Cooperation and Development (OECD). The OECD data set includes data on health care outcomes such as life expectancy for both males and females, potential years of life lost attributable to a number of causes, and infant and perinatal mortality. Many other premature mortality and morbidity measures are included, but they are not available for many countries nor for long time periods. This is true of many of the other data series as well.

The data set also includes data on medical determinants of health such as the number of inpatient beds per 1,000 inhabitants, the number of physicians and other health care professionals, the number of hospital admissions, etc. It also includes data on health insurance coverage provided by public agencies, but not private health insurance. This exclusion renders the insurance data almost useless. Total and public expenditures on various health care inputs, including pharmaceuticals, are also available. In order to facilitate cross-national comparisons, various exchange rates are provided to convert these expenditure levels into U.S. dollars. For the years 1980, 1985, 1990, and 1993 separate purchasing power parity (PPP) exchange rates are provided for pharmaceuticals and medical services. This is true for almost all countries in 1990 and 1993 and for most countries for 1980 and 1985.

III.2 Measuring pharmaceutical consumption

How one converts a nation's per capita pharmaceutical expenditures to U.S. dollars for the purpose of cross-national comparisons is of crucial importance. In the Babazono and Hillman (1994) paper cited above, the authors used PPP exchange rates designed to convert total gross domestic product (GDP) to U.S. dollars. This approach is only appropriate 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 Tadeusz Szuba (1986) and Patricia Danzon and Allison Percy (1995), have demonstrated that this is far from the truth. Drug price regulation remains a national prerogative in many countries and non-tariff trade barriers have traditionally been significant. Both price regulation and barriers vary widely.

For instance, biased (in favor of domestic producers) price regulation is practiced in France and Italy. In this situation, a manufacturer's price must be approved for a product to be reimbursed by social insurance programs. This keeps pharmaceutical prices particularly low in France and in Italy. Spain also strictly regulates pharmaceutical prices. Other OECD countries, such as the United Kingdom and Germany, also regulate pharmaceutical prices, albeit indirectly and typically much less stringently.[6] The U.S. and Denmark, at the other extreme, generally permit free pricing of pharmaceuticals, subject to market forces. For these reasons one might expect gross domestic product PPP exchange rates to be unsatisfactory for converting pharmaceutical expenditures to U.S. dollars for cross-national comparisons.

Luckily, PPP exchange rates designed specifically for converting pharmaceutical expenditures to U.S. dollars are available for 1980, 1985, 1990, and 1993. Table 1 presents measures of per capita pharmaceutical expenditures converted to U.S. dollars using pharmaceutical PPP exchange rates and gross domestic product PPP exchange rates for 1990. One should note that conversions using gross domestic product PPP exchange

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[6] See Garattini et al. (1994) for an excellent comparison of the pharmaceutical markets and price regulation in Italy, France, Germany, and the United Kingdom.

Table 1 : Comparing Measures of Real Pharmaceutical Consumption Using Pharmaceutical Purchasing Power Parity and GDP Purchasing Power Parity Exchange Rates, 1990

Rank	Country	Pharm PPPs	GDP PPPs	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	United States	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	United Kingdom	183.721	131.667	52.054
18	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

rates invariably underestimate actual pharmaceutical expenditures outside of the U.S. For instance, using the pharmaceutical PPP exchange rate, one obtains a measure of $561 for France, whereas the value is only $256 using the gross domestic product PPP exchange rate. A similar result is found for Italy. The biggest differences are found for those countries with the strictest price regulations, France and Italy, which we take as evidence of the desirability of the pharmaceutical PPPs.

Danzon and Percy (1995) argue that even the pharmaceutical PPP exchange rates are flawed and provide more accurate Fisher price indexes for a handful of countries to convert pharmaceutical expenditures to U.S. dollars.[7] These price indexes are painstakingly calculated using detailed proprietary data that is only available for a few countries. These relative prices should be regarded as the "gold standard" as they are undoubtedly the most accurate. But, they are not available for any countries other than France, Italy, Germany and the United Kingdom. Further, the detailed prices were converted from other currencies into dollars using market exchange rates, rather than purchasing power parity exchange rates. This seems to have had little effect on the rankings of consumption of various countries, but it does seem to have compressed the consumption, relative to U.S. consumption.

One can convert pharmaceutical consumption in France, Italy, Germany and the United Kingdom to U.S. dollars using the pharmaceutical PPP and the gross domestic product PPP exchange rates for 1980, 1985 and 1990. Danzon and Percy measures are also available for these countries in these four years. Correlations among the three measures of pharmaceutical consumption for these years and countries were calculated. The correlation between the Danzon and Percy measure and the pharmaceutical purchasing power parity measure is fairly high at 0.872. The other two correlation

[7]The Fisher price index is the geometric mean of the Laspeyres and Paasche price indexes. Like both the Laspeyres and Paasche indexes, it is intransitive. For example, the product of the indexes between the U.S. and Canada and between Canada and Denmark is not equal to the index between the U.S. and Denmark. Unlike the other two indexes, the Fisher price index yields results which are invariant to which country is used as a base.

coefficients are both roughly 0.5. This indicates that the consumption measure using the pharmaceutical purchasing power parity exchange rates compares well with the Danzon and Percy measure.

Szuba (1986) also painstaking assembled price ratios using detailed proprietary data, though with a slightly different approach. His price coefficients are also excellent--when available. Szuba's price coefficients are applied to expenditure data after they have been converted to U.S. dollars using conventional market exchange rates (like Danzon and Percy). He found that in 1983 Italy had the lowest pharmaceutical prices and that the U.S. had the highest pharmaceutical prices among the six countries he studied (those countries listed in Table 2a). We apply his price coefficients to 1985 expenditure estimates which have been converted to U.S. dollars using the market exchange rates.

In Table 2a we compare measures of real pharmaceutical expenditures for 1985 using the following conversion factors: market exchange rates, gross domestic product purchasing power parities, pharmaceuticals purchasing power parities, Danzon and Percy's Fisher price indexes, and Szuba's(1986) 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 United States. Switzerland and the United Kingdom tend to consume fewer pharmaceuticals than the United States no matter which measure is used.

Table 2b presents correlations among the different measures for 1985. The measure that we use in our study is very highly correlated with the Danzon and Percy measure for the countries for which all of the measures are available. Note that the measures that use simple market exchange rates and gross domestic product purchasing power parity exchange rates are not highly correlated with the Danzon and Percy or Szuba measures. The simple 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 gross domestic product PPP

Table 2a: Comparing Measures of Real Pharmaceutical Expenditures for 1985 in Six Countries Using Various Conversions to U.S. Dollars

Country	Exchange Rates	GDP Purchasing Power Parities	Pharm. Purchasing Power Parities	Danzon & Percy's Fisher Price Index	Szuba's Price Coefficients
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
U. S.	150.00	150.00	150.00	150.00	150.00
U. K.	66.67	94.55	119.85	103..44	126.01

Table 2b: Correlations Among the Various Pharmaceutical Consumtpion Measures

	Exchange Rate	GDP PPP	Pharm. PPP	Danzon & Percy	Szuba
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

exchange rates. Since we wish to study more than the five 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.

Our measure of pharmaceutical consumption should still be viewed as only a reliable approximation. The measure can not capture all of the more subtle differences in pharmaceutical use across countries. Two countries may spend approximately the same amount of money on pharmaceuticals but still exhibit very different patterns of consumption. For example, Livio Garratini and his colleagues (1994) attempted to analyze the prices of the 100 best selling pharmaceuticals in 1992 in Italy, Germany, France, and the United Kingdom. Only eight products were common to all four best seller lists. They also found that the proportions of pharmaceuticals sold in three different therapeutic classes varied considerably across the four countries. Danzon and Percy and Szuba also found this to be the case. Thus, the measure of pharmaceutical consumption used in this research is hardly uniform across countries.

III.3 Health indicators

Thus far we have discussed problems in the measurement of our explanatory variable of greatest interest. Now we turn to the dependent variables. One reason that so many previous researchers have focused on mortality is that it is widely considered the most reliable health indicator, especially amongst the industrialized countries which routinely report vital statistics (see U.S. Congress, Office of Technology Assessment [OTA], 1993)[8] . Focusing on mortality rates for less developed countries can be more problematic. As Murray (1987) points out, recent empirical information on mortality

[8] There are other dimensions of health that are nearly impossible to measure objectively. These include the level of comfort and the general issue of the quality, as well as length, of life. The availability of these measures tends to be rather spotty even for the OECD countries and the quality of these measures is yet to be determined. Thus, we focus on life expectancy.

exists for only a handful of the developing countries. Many published estimates of mortality in most developing countries are therefore based on old surveys updated over the years with *assumed* rates of improvement in mortality. This is not a problem with our analysis, since we have limited our study to industrialized members of the OECD, which all routinely publish vital statistics on a yearly basis. The mechanistic updating of old data does undermine the credibility of the research on the developing countries.

Life expectancies at birth and at various advanced ages have been used as health indicators in many of the previous studies cited above, and we use them also. Infant mortality rates are also used as a health indicator. According to the Office of Technology Assessment (1993) there appear to be no serious problems with the data from industrialized countries on life expectancy. It does urge caution in using cause-specific mortality rates due to international differences in diagnostic techniques, the use of autopsies, and the training of medical personnel.

Even among the richest countries, caution is prudent in using infant mortality in international comparisons. For example, physicians in the United States are more likely to resuscitate extremely premature and low birth weight infants, who later die. These are classified as live births in the U.S. and thus included in infant mortality calculations. Other countries are more likely to classify such births as fetal deaths. Also in some countries, infants may be classified as stillbirths if they die before their births are registered, sometimes as many as two days after birth. In Japan, this problem is exacerbated by the fact that many infant deaths are recorded as stillbirths because the latter are not recorded in Koseki, the Japanese family registration system (OTA, 1993). We exclude Japan from our analysis due to this and other problems with Japanese health care data.

Measures of infant mortality and female and male life expectancy for the twenty one countries included in the study for the years 1980, 1985, 1990, and 1993 are presented in Table 3. The data show that each country experienced improvements in each of these health indicators throughout the 1980s and into the early 1990s, carrying on long-

Table 3 : Health Indicator Measures for Countries of Interest

COUNTRY	1980	1985	1990	1993
United States				
Infant Mortality	12.6	10.6	9.2	8.4
Female Life Expectancy	77.4	78.2	78.8	78.8
Male Life Expectancy	70.0	71.1	71.8	72.2
France				
Infant Mortality	10.0	8.3	7.3	6.4
Female Life Expectancy	78.4	79.4	80.9	81.4
Male Life Expectancy	70.2	71.3	72.7	73.3
Australia				
Infant Mortality	10.7	9.9	8.2	6.1
Female Life Expectancy	78.1	78.8	80.1	80.9
Male Life Expectancy	71.0	72.4	73.9	75.0
Austria				
Infant Mortality	14.3	11.2	7.8	6.5
Female Life Expectancy	76.1	77.3	78.9	79.4
Male Life Expectancy	69.0	70.4	72.4	73.0
Belgium				
Infant Mortality	12.1	9.8	8.0	8.0
Female Life Expectancy	76.8	77.7	79.1	79.8
Male Life Expectancy	70.0	70.9	72.4	73.0
Canada				
Infant Mortality	10.4	8.0	6.8	6.8
Female Life Expectancy	79.1	79.7	80.4	81.2
Male Life Expectancy	71.9	73.0	73.8	74.9
Denmark				
Infant Mortality	8.4	7.9	7.5	5.4
Female Life Eexpectancy	77.6	77.5	77.7	77.6
Male Life Expectancy	71.4	71.6	72.0	72.3
Finland				
Infant Mortality	7.6	6.3	5.6	4.4
Female Life Expectancy	77.6	78.5	78.9	79.5
Male LifeExpectancy	69.2	70.1	70.9	72.1
Germany, Federal Republic of				
Infant Mortality	12.7	8.9	7.1	5.8
Female Life Expectancy	76.6	78.1	79.1	79.3
Male Life Expectancy	69.9	71.5	72.7	73.8
Greece				
Infant Mortality	17.9	14.1	9.7	8.5
Female Life Expectancy	76.6	78.9	79.4	79.9
Male Life Expectancy	72.2	74.1	74.6	74.9

Table 3 (Continued)

COUNTRY	1980	1985	1990	1993
Ireland				
Infant Mortality	11.1	8.9	8.2	5.9
Female Life Expectancy	75.0	76.4	77.5	78.2
Male Life Expectancy	69.5	70.8	72.0	72.7
Italy				
Infant Mortality	14.6	10.5	8.2	7.3
Female Life Expectancy	77.4	78.6	80.0	80.9
Male Life Expectancy	70.6	72.0	73.5	74.5
Netherlands				
Infant Mortality	8.6	8.0	7.1	6.3
Female Life Expectancy	79.2	79.7	80.1	80.0
Male Life Expectancy	72.4	73.1	73.8	74.0
New Zealand				
Infant Mortality	12.9	10.8	8.4	7.3
Female Life Expectancy	76.3	77.3	78.3	78.9
Male Life Expectancy	70.1	71.0	72.4	73.1
Norway				
Infant Mortality	8.1	8.5	7.0	5.1
Female Life Expectancy	79.2	79.4	79.8	80.2
Male Life Expectancy	72.3	72.6	73.4	74.2
Portugal				
Infant Mortality	24.3	17.8	11.0	8.7
Female Life Expectancy	75.9	76.7	77.9	78.0
Male Life Expectancy	67.7	69.7	70.9	70.8
Spain				
Infant Mortality	12.3	8.9	7.6	6.8
Female Life Expectancy	78.6	79.7	80.4	80.9
Male Life Expectancy	72.5	73.3	73.4	73.3
Sweden				
Infant Mortality	6.9	6.8	6.0	4.8
Female Life Expectancy	78.8	79.7	80.4	80.8
Male Life Expectancy	72.8	73.8	74.8	75.5
Switzerland				
Infant Mortality	9.1	6.9	6.8	5.6
Female Life Expectancy	78.8	80.1	80.9	81.4
Male Life Expectancy	72.3	73.4	74.0	74.7

Table 3 (Continued)

COUNTRY	1980	1985	1990	1993
Turkey				
Infant Mortality	95.3	75.3	59.3	52.6
Female Life Expectancy	64.3	65.4	68.4	70.0
Male Life Expectancy	58.2	60.7	64.1	65.4
United Kingdom				
Infant Mortality	12.1	9.4	7.9	6.3
Female Life Expectancy	75.9	77.4	78.6	78.9
Male Life Expectancy	70.2	71.5	72.9	73.6

term trends. As of 1993, infant mortality ranged from a low of 4.4 deaths per 1000 live births in Finland to a high of 52.6 deaths per 1,000 live births in Turkey. Though still high by OECD standards, Turkey's infant mortality rate in 1993 was a significant improvement over what it had been only thirteen years earlier (95.3 deaths per 1,000 live births in 1980). Greece, Portugal, and Spain also experienced significant improvements in infant mortality throughout the 1980s and into the early 1990s.

Female life expectancy at birth in 1993 was highest in France and Switzerland at 81.4 years, and no fewer than nine of the countries included enjoyed female life expectancies over 80 years. Again, Turkey ranked lowest in female life expectancy in 1993, as it had in earlier years, at 70 years. As far as male life expectancy at birth is concerned, Sweden ranked first in 1993 with a life expectancy of 75.5 years. Once again, Turkey ranked last with a male life expectancy of 65.4 years. To keep the Turkish experience in perspective, this showing is also a vast improvement over the life expectancy of a male Turk in 1980 which was only 58.2 years.

IV. The Analysis

IV.1 Specification Issues

Our primary interest is in the determinants of the health of a nation's citizens. One useful starting point is to consider an individual's health as being determined by a number of different factors through a household production function.[9] These factors include the consumption of goods which may have either positive or negative effects on an individual's health. For example, consumption of cigarettes has been found to lead to increases in mortality, or lower life expectancies, due to emphysema, lung cancer, and heart disease. Meanwhile, nutritional variables have been found in epidemiological studies to affect life

[9] Michael Grossman (1972a,b) did the seminal work on the household production of health.

expectancy (Gage and O'Connor, 1994). The studies cited in the literature review indicate that the consumption of non-medical goods, so-called lifestyle choices, are an important determinant of the health of a population, especially for rich countries.

Some of the better previous studies have indicated that goods and services provided by the medical care sector also help to determine the health of a population. Pharmaceutical products fall into this general category along with other health care services. One would expect the consumption of such goods and services to augment a population's health. In a market-oriented economy, any services that did not augment health would not be demanded for very long. Even in more centralized economies, one would expect public choice mechanisms to favor productive health care over useless health care.

We, therefore, can think of a nation's health as being a function of pharmaceutical consumption, other health care consumption, wealth, and lifestyle and environmental factors. Specifically we proxy the health of a nation's population using the life expectancies of males and females at birth, at the age of 40, and at the age of 60, as of the year 1993. Infant mortality is also considered separately. These measures are discussed more fully in Section III.3.

We measure per capita pharmaceutical consumption by converting 1985 per capita pharmaceutical expenditures to 1990 U.S. dollars. The choice of conversion method is based on the discussion of the previous section. We first apply the nation's 1985 pharmaceutical purchasing power parity exchange rate and then inflate the resulting figure to 1990 U.S. dollars by applying the U.S. consumer price index. No pharmaceutical purchasing power parity exchange rate is available for Switzerland in 1985, though it is available for 1990. Thus, we convert Swiss pharmaceutical expenditures to 1990 U.S. dollars by first inflating Swiss pharmaceutical expenditures to 1990 Swiss Francs using the Swiss index of consumer prices and then converting this figure to U.S. dollars using the Swiss pharmaceutical purchasing power parity exchange rate for 1990. A similar

approach is taken with the United Kingdom. Partly based on conversations with OECD officials, we believe that the United Kingdom's pharmaceutical purchasing power parity exchange rate for 1985 is problematic.

The measure of other health care consumption is likewise constructed by converting 1985 per capita health care expenditures to 1990 U.S. dollars. For this we first apply the nation's 1985 *health care* purchasing power parity exchange rate and then the U.S. consumer price index. From this figure the measure of per capita pharmaceutical consumption is subtracted. For Switzerland, the approach taken is similar to the one described above for pharmaceuticals, because the Swiss health care purchasing power parity exchange rate for 1985 is unavailable.

Living standards and other lifestyle factors are also controlled for. First, we include each nation's 1985 per capita gross domestic product converted to 1990 U.S. dollars. For the conversion we use each nation's 1985 gross domestic product purchasing power parity exchange rate and the U.S. consumer price index. We also control for cigarette smoking, by including the percentage of females or males aged fifteen years or older who smoked as of 1983. Alcohol consumption is controlled for by including each nation's 1983 alcohol consumption in litres per capita per year. Finally, richness of diet is controlled for by including a measure of animal fat consumed per capita per day. This measure is constructed by subtracting the average animal protein calories consumed per day from the average total animal calories consumed per day. See Exhibit 1 for the list of definitions of the variables used in the study.

Exhibit 1: Definitions of Variables

Variable	Definition

MLE	Male life expectancy at birth, 1993
MLE40	Male life expectancy at age 40, 1993
MLE60	Male life expectancy at age 60, 1993
FLE	Female life expectancy at birth, 1993
FLE40	Female life expectancy at age 40, 1993
FLE60	Female life expectancy at age 60, 1993
INFMORT	Infant Mortality (deaths per 1000 live births), 1990
PHPC	Pharmaceutical expenditures per capita, 1985 (in 1990 US $)
GDPPC	Gross domestic product per capita, 1985 (in 1990 US $)
HEPC	Health expenditures (not including pharmaceuticals) per capita, 1985 (in 1990 US $)
FEMSMOKE	Percentage of females aged 15 years or older who smoke, 1983
MALESMOKE	Percentage of males aged 15 years or older who smoke, 1983
ALCOHOL	Alcohol consumption in litres per capita, 1983
ANFAT	Animal fat consumption in calories per capita per day, 1983

In our specification, the explanatory variables are lagged by roughly ten years, because it is believed that lifestyle factors and medical care consumption will have a cumulative rather than a contemporaneous effect on health.[10] In this, we follow Zweifel and Ferrari (1992). 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 idea would require several lags of each explanatory variable. Both data and sample size limitations lead us to include only one lag. First, there are only twenty one countries in our sample. Adding a large number of lagged explanatory variables would lead to a huge decrease in precious degrees of freedom. Second, purchasing power parity conversions for total health care and pharmaceuticals are available only for 1980, 1985, and 1990. Thus, we would only be able to include lagged terms for 1980 or 1990. Given the persistence of the explanatory variables over time, this would most likely lead to a multicollinearity problem. Including lags from the 1960s or the 1970s could be worthwhile, but the necessary data do not exist.

Finally, a double-log, or constant elasticity, functional form is chosen. There are a couple of reasons for this. First, the coefficients from the double log regression are

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[10] The exact lag depends on data availability.

interpreted as elasticities. Second, a specification was desired which would allow for diminishing returns to all of the independent variables.

Other specifications were considered. A linear specification was rejected since it would not allow for diminishing returns. Second order linear specifications were estimated, and the results confirmed the importance of diminishing returns, but the estimated residuals from the regressions were highly skewed and we could reject the hypothesis that they were normally distributed using a Shapiro-Wilk test (1965). Also, multicollinearity was a major problem. Second order double logarithmic specifications were also tried, and they were preferable according to Hausman specification tests, but so much multicollinearity was introduced, that the results were uninterpretable. Condition indexes from these regressions were in the range of 15000 which indicated severe multicollinearity. Conventionally, an index of 45 indicates a serious problem (see Greene, 1993). Ultimately, a second order logarithmic term for animal fat calories was included because we did not want to constrain the effect to be monotonic. This also introduced a fair amount of collinearity but improved model performance overall.[11]

IV.2 Descriptive Statistics

Descriptive statistics presented in Tables 4a and 4b, indicate that there is a good deal of variation in both the dependent variables to be explained and in the regressors to do the explaining. For example, pharmaceutical consumption per capita varies by a factor of almost 20, from $28.64 in Turkey to $490.68 in France. Gross domestic product per capita varies by a factor of five, from $4080 in Turkey to $19876.53 in the United States. Lifestyles also vary widely among the countries in our sample. For convenience in checking and critiquing our work, the entire data set used for this study is presented in Tables B1, B2 and B3, in the appendix.

[11] Adding additional second-order terms did not improve the model's performance.

Table 4a: Descriptive Statistics for Dependent Variables

Variable	Mean	Stand. Dev.	Minimum	Maximum
MLE	73.01	2.52	63.3	75.5
ln(MLE)	4.29	0.036	4.15	4.32
MLE40	35.60	1.34	31.7	37.3
ln(MLE40)	3.57	0.038	3.46	3.62
MLE60	18.44	0.99	16.0	19.7
ln(MLE60)	2.91	0.055	2.77	2.98
FLE	79.14	3.22	66.0	81.4
ln(FLE)	4.37	0.044	4.19	4.40
FLE40	40.87	1.58	36.0	42.9
ln(FLE40)	3.71	0.040	3.58	3.76
FLE60	22.65	1.36	18.4	24.6
ln(FLE60)	3.12	0.063	2.91	3.20
INFMORT	9.91	10.83	5.60	59.3

Table 4b: Descriptive Statistics for Regressors

Variable	Mean	Stand. Dev.	Minimum	Maximum
PHPC	201.59	96.00	28.64	490.68
ln(PHPC)	5.19	0.56	3.35	6.20
GDPPC	13968.87	3988.46	4080.08	19876.53
ln(GDPPC)	9.49	0.37	8.31	9.90
HEPC	1221.38	568.22	141.36	2335.83
ln(HEPC)	6.95	0.67	4.95	7.76
FEMSMOKE	27.10	7.30	9.00	42.5
ln(FEMSMOKE)	3.26	0.33	2.20	3.75
MALESMOKE	43.56	9.09	31.8	62.8
ln(MALESMOKE)	3.75	0.20	3.46	4.14
ALCOHOL	10.51	4.31	1.20	19.1
ln(ALCOHOL)	2.21	0.65	0.18	2.95
ANFAT	1026.43	292.46	252	1324
ln(ANFAT)	6.87	0.40	5.53	7.19

IV.3 Empirical Results for Life Expectancy

The main body of the analysis consists of the regressions reported in Table 5. In these regressions life expectancies at birth, at 40 years of age and at 60 years of age are analyzed for both our full sample of twenty one 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.

The Effect of Lifestyle. First, and surprisingly, it appears that the lifestyle variable that has the greatest effect on life expectancy at all ages is the consumption of animal fat. The results indicate that consumption of animal fat has a very strong and large positive effect on all three 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. This is further evidence of the epidemiological transition (see Gage and O'Connor, 1994). At low levels of fat consumption, enriching a diet is beneficial to health, but at some point, further

Table 5: Constant Elasticity Regression Results for Full Sample
(t-Statistics in parentheses)

	Dependent Variable: Life Expectancy		
Regressor	At Birth	Age 40	AGE 60
CONSTANT	-0.5344*	-0.0256	-0.8954
	(-1.7130)	(-0.0370)	(-0.8130)
FEMALE	0.0392**	0.0996**	0.1369**
	(4.2480)	(5.4290)	(5.1200)
ln(PHPC)	0.0050	0.0172*	0.0401**
	(1.0860)	(1.7400)	(2.6950)
ln(GDPPC)	0.0121	0.0572**	0.0876**
	(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**	-0.0143	-0.0188
	(-2.3890)	(-1.6230)	(-1.3410)
ln(ALCOHOL) X FEMALE	0.0167**	0.0150**	0.0312**
	(4.8500)	(2.1060)	(2.9850)
ln(ANFAT)	1.4040**	0.9548**	0.9096**
	(13.7640)	(4.1660)	(2.5180)
ln(ANFAT) - Squared	-0.1045**	-0.0728**	-0.0706**
	(-13.3550)	(-4.1710)	(-2.5690)
R-squared	0.9621	0.9308	0.9290
Adj. R-squared	0.9515	0.9113	0.9090
Shapiro-Wilkes p-value	0.7068	0.5968	0.5310
Sample Size	42	42	42

* Significant at the 0.10 level based on a two-tailed test.
** Significant at the 0.05 level based on a two-tailed test

consumption of fat decreases life expectancy. Excessive fat intake has been found to be linked to degenerative diseases such as heart disease and cancer.

How much animal fat is too much? The results indicate that the effect of animal fat consumption becomes negative for life expectancies at birth, age 40 and age 60, beyond consumption levels of 827 calories per day, 705 calories per day, and 628 calories per day respectively. The caloric consumption measures translate to 92 grams, 78 grams, and 70 grams respectively. These results are interesting for two reasons. First, given the epidemiological transition, one would expect animal fat to be more helpful at birth than at advanced ages, since a rich diet would decrease mortality early in life infectious disease is a major killer, but be less effective and more likely to increase mortality at more advanced ages when degenerative disease becomes important. The detrimental effects of too rich a diet at advanced ages are offset by the positive effects of a rich diet in the early years of life when we consider life expectancy at birth. When we consider the life expectancies at ages 40 and 60, this positive effect is not as important, so we find negative returns to fat consumption at lower levels. It is also interesting to note that the numbers are very close to the U.S. Food and Drug Administration's dietary guidelines which call for fat consumption to be no higher than 80 grams in a 2500 calorie diet.

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. However, this is not very precisely estimated. The 95 percent confidence interval includes an elasticity of about positive 4 percent at age 60.

Alcohol consumption has negative and significant effects on male life expectancy at birth and at age 40, but the magnitudes of the effects are small, indicating that doubling alcohol consumption would only lower life expectancy by at most one and a half percent. This is somewhat surprising, given the epidemiological research showing that moderate (up to three drinks a day) drinking substantially reduces the risk of heart disease (Gaziano, et al, 1993). The interactions between alcohol consumption and the female dummy

indicate that alcohol consumption has roughly no effect on female life expectancies. This most likely reflects alcohol consumption difference between women and men. If men drink most of the alcohol in all countries, variation in aggregate alcohol consumption will be largely variation in male alcohol consumption and have a bigger effect on male life expectancy than on female life expectancy. The above results are quite different from those found in some previous studies, especially Cochrane, St. Leger, and Moore (1978) who found that cigarette smoking and alcohol consumption are important determinants of mortality.

The Effect of Wealth. Wealth, as measured by lagged gross domestic product 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. The results indicate that doubling gross domestic product would increase life expectancy at 40 by roughly six percent, and life expectancy at 60 by roughly nine percent. In terms of remaining years of life, this means that a 60 year old man living in a country with the average life expectancy at 60 could expect to see his remaining life expectancy increase from roughly 18.5 years to roughly 20 years. A 60 year old female living in such a country could expect to see her remaining life expectancy increase from 22.5 years to roughly 24.5 years.

These are large increases, and not terribly surprising, since economic growth has been associated with rising life expectancies over the last two centuries. These results are consistent with most previous studies, the most notable exceptions being Auster, Leveson, and Sarachek (1969) and Zweifel and Ferrari (1992).[12] In each of these two studies, increased per capita wealth was associated with higher mortality rates and lower life expectancies, respectively.

The Effect of Health Care Consumption. The most surprising results are those relating pharmaceutical consumption and other health care consumption to life expectancy. First, we find our measure of health care consumption to have virtually no

[12]We also attempted to replicate Zweifel and Ferrari (1992) in detail. See appendix.

effect on life expectancy at birth and to be negatively related to life expectancy at the ages of 40 and 60, although the relationship is not statistically significant in any of the analyses. Further, the negative relationship is also weak in terms of magnitudes. The elasticity is never greater in absolute magnitude than -0.0145. Our data reject any large effect of either sign. As was pointed out in the literature review, this is not the first study to find a weak negative relationship between life expectancy and health care consumption, especially amongst studies of developed countries. For instance, Cochrane, St. Leger and Moore (1978) found a negative relationship, albeit a weak one, between many medical inputs and life expectancy. The result does conflict with Zweifel and Ferrari (1992), however. This could mean that developed countries are on the flat of the curve when it comes to non-pharmaceutical health care consumption or it could simply reflect a bias due to the endogeneity of health care consumption and a nation's health.

The Effect of Pharmaceutical Consumption. Pharmaceutical consumption, on the other hand, appears to be surprisingly productive. It has positive and statistically significant relationships with the life expectancies at the ages of 40 and 60. Doubling pharmaceutical expenditures would increase life expectancy at 40 by roughly 2 percent, and life expectancy at 60 by about 4 percent. 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 years 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 years to 23.5 years. These results are sharply different from those found in the flawed study by Babazono and Hillman (1994), 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 Lichtenberg (1996) and others who have found, in micro studies and in studies of restricted formularies, that pharmaceutical consumption is associated with lower morality rates and better health outcomes.

Sensitivity of the Results. To check for the robustness of the above results we tried several variants. For one the models were re-estimated, excluding Turkey. This was done because Turkey is quite different from the rest of our sample in such key measures as wealth, pharmaceutical consumption, and life expectancy. For instance, male life expectancy at birth in Turkey was 65.4 years in 1993, whereas the next lowest life expectancy was Portugal's at 70.8 years. Since ordinary least squares estimators can be sensitive to outliers, one might fear that the observations for Turkey were driving the results. When Turkey was excluded from the analysis, the results did not change appreciably. For instance, the elasticity of pharmaceutical consumption was 0.016 for life expectancy at age 40 and equal to 0.039 for life expectancy at age 60. These estimates were significant at the same levels as in the inclusive regressions reported in Table 5. Therefore, including Turkey does not drive the results.

We also ran regressions with only the European countries, the results of which are reported in Table 6. The results are very similar to those from our original inclusive regressions. There are some differences, though. First, pharmaceutical consumption has a larger and more statistically significant positive effect for Europe than it had in our original sample, which also included Turkey, North America and Oceana. Second, the effect of wealth is smaller and insignificant. If anything, pharmaceutical consumption is even more productive in producing health in Europe than it is elsewhere.

As an additional test for sensitivity, the lifestyle variables were dropped from the models. Excluding the lifestyle variables, the positive effect of pharmaceutical consumption is much larger for all three life expectancies, especially at the youger ages. The effect of wealth is greatly increased in the regression for life expectancy at birth, but not at later ages. Finally, the effect of other health care consumption does not change in any appreciable way. Apparently, controlling for lifestyle factors is important to avoid overstating the beneficial effect of pharmaceutical consumption on health.

Table 6: Constant Elasticity Regression Results for the European Countries
(t-Statistics in parentheses)

	Dependent Variable: Life Expectancy		
Regressor	At Birth	Age 40	AGE 60
CONSTANT	-0.8722	-4.2760*	-5.9190
	(-0.9360)	(-1.9060)	(-1.5420)
FEMALE	0.0460**	0.0808**	0.1393**
	(3.9620)	(3.4870)	(3.7130)
ln(PHPC)	0.0090*	0.0230**	0.0501**
	(1.9110)	(2.3370)	(3.2100)
ln(GDPPC)	0.0094	0.0228	0.0353**
	(0.4920)	(0.5080)	(0.5160)
ln(HEPC)	0.0020	-0.0059	-0.0083
	(0.2600)	(-0.3360)	(-0.3070)
ln(SMOKE)	-0.0104	-0.0096	0.0055
	(-1.4910)	(-0.6950)	(0.2840)
ln(ALCOHOL)	-0.0090**	-0.0118	-0.0108
	(-2.2710)	(-1.3220)	(-0.6410)
ln(ALCOHOL) X FEMALE	0.0132**	0.0238**	0.0324*
	(2.7100)	(2.3160)	(1.9120)
ln(ANFAT)	1.5010**	2.2700**	2.4860*
	(4.9460)	(3.1270)	(2.0250)
ln(ANFAT) - Squared	-0.1107**	-0.1687**	-0.1858*
	(-4.9520)	(-3.1480)	(-2.0440)
R-squared	0.9574	0.9386	0.9350
Adj. R-squared	0.9400	0.9135	0.9084
Shapiro-Wilkes p-value	0.6668	0.8728	0.3420
Sample Size	32	32	32

* Significant at the 0.10 level based on a two-tailed test.
** Significant at the 0.05 level based on a two-tailed test

IV.4 Empirical Results for Infant Mortality

Table 7 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 comtemporaneous levels of the regressors. In the second regression it is explained by values of the regressors which have been lagged by five to seven years (as in the life expectancy regressions). We do this for two 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.

The Effect of Lifestyle. Lifestyle factors make a big difference for infant mortality. In both regressions reported in Table 7 one can reject the hypothesis that all four of the lifestyle coefficients are zero. The nutrition variable, 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 effects 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 beneficial effect of animal fat consumption, at least at levels below 1000 calories per day, is again consistent with the idea of the epidemiological transition. Those diseases which are most likely to kill infants are those which a rich diet is most helpful. The bad effects of a diet that is too rich would most likely be due to issues pertaining to maternal behavior at the prenatal stage. Extremely rich diets are probably correlated with other behavioral characteristics which we have not directly controlled for, such as maternal obesity or diabetes.

The results for smoking are more problematic. In the contemporaneous regression, female smoking appears to have a very small negative and statistically

insignificant effect on infant mortality. The result is quite imprecise. For instance, at the 95 percent level, we couldnot reject a positive elasticity of nearly 0.20. Still, this result contradicts most previous studies, including the one by Cochrane, St. Leger and Moore (1978). 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 comforting, 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 led to poorer fetal health, thus more miscarriages and stillbirths. But, perhaps there is not much effect on infant mortality for those fetuses that survive to a live birth. The results on alcohol consumption are not mixed. In both regressions alcohol consumption is negatively, although statistically insignificantly, associated with infant mortality.

The Effect of Wealth. The results on the effects of wealth on infant mortality are mixed. Gross domestic product 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. The results in the contemporaneous model indicate that a doubling of gross domestic product per capita would cut infant mortality by nearly 50 percent. The average country would see a fall in infant mortality from roughly ten infant deaths to only five infant deaths per 1000 live births. The results in the lagged regression indicate that for the average country, a doubling of gross domestic product would slightly increase infant mortality, raising it by roughly 3 percent. Again, note that this effect is imprecisely measured. Our data can not reject a negative effect as large as -0.16 at the 95 percent level.

The Effect of Health Care Consumption. Again the results are mixed on the effect of non-pharmaceutical health care. In the contemporaneous regression the effect is positive and insignificant, whereas in the lagged regression the effect is negative and

Table 7: Constant Elasticity Regression Results for Infant Mortality in 1990 (t-statistics in parentheses)

	Specification	
Regressor	Contemporaneous	Lagged Five Years
CONSTANT	53.721**	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
Adj. R-squared	0.9132	0.9475
Shapiro-Wilkes p-value	0.3814	0.1493
Sample Size	21	21

* Significant at the 0.10 level based on a two-tailed test.
** Significant at the 0.05 level based on a two-tailed test.

significant. The result from the contemporaneous regression is very much like the one found by Cochrane, St. Leger and Moore (1978), who found that the number of doctors per capita in general and pediatricians per capita in particular had a positive effect on infant mortality. The result from the lagged regression is similar to the one found by some others, particularly Hadley (1982). The result from the lagged regression indicates that doubling health care consumption would lower infant mortality in the average country from roughly 10 deaths per 1000 live births to roughly eight deaths per 1000 live births.

The 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. The point estimate would seem to indicate that after controlling for wealth, other health care 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.

A small effect of pharmaceutical consumption on infant mortality makes sense in light of the results from the regressions for life expectancy. It appears that pharmaceutical consumption has great potential for extending life in one's later years, but that it can do little to improve the likelihood of survival for the young. For the young, nutrition plays a much larger role. Also, one should keep in mind that the pharmaceutical consumption variable is the average pharmaceutical expenditures for the entire population. Data on the pharmaceutical consumption of infants or women during pregnancy are not available.

Sensitivity of the Results. The sensitivity of our results were checked for in the same manner as they were for the life expectancy regression results. Excluding Turkey from the analysis did not change the results in the lagged regression in any appreciable way. In the contemporaneous regression, no individual variables were statistically

significant, after dropping Turkey, not even the richness of diet variables. Further dropping North America and Oceana, leaving only the 16 European countries, changed the results somewhat in the contemporaneous regression. For one, the effect of smoking became positive and marginally significant (at the 25 percent level). The effect of pharmaceutical consumption became more positive with an elasticity of 0.08, but again was estimated very imprecisely. The effect of wealth became more negative with an elasticity of -0.8441 but less precisely measured. In the lagged regression, the effects of wealth and alcohol consumption both went from being positive and insignificant to negative and insignificant.

As an additional test for sensitivity, we dropped the lifestyle variables from our analysis. Here the results on pharmaceutical consumption are quite different. The effect of pharmaceutical consumption is negative in each regression and is even significant in the lagged regression, where the elasticity is -0.32. The effect of wealth is large, negative (the elasticity is -1.5), and significant in the contemporaneous regression (no doubt, capturing a good deal of the effect of nutrition). It is also negative, though much smaller and insignificant, in the lagged regression. Finally, the effect of health care consumption changes but not in a significant way. It remains positive and insignificant in the contemporaneous regression and negative and significant in the lagged regression.

Overall, the infant mortality model was much less successful than the life expectancy models. Most results are not robust to slightly different, and perfectly defensible, specifications. The infant mortality results must be counted as suggestive, at best.

V. Conclusion

The purpose of this study has been to study the production of health with special emphasis on the productivities of pharmaceutical consumption and other health care.

Whereas pharmaceutical consumption has been shown to have a positive effect on health in micro studies and in studies of restricted formularies, such an effect has not previously been found in studies of international aggregate data. But it has hardly been studied. Most cross-national work, driven by an obsession over cost containment, has concentrated on expenditures rather than health.

Results have also been mixed on the effects of wealth and general health expenditures on health outcomes in the few international studies of the production of health. Most such studies have found that lifestyle and environmental factors have a much greater impact than health care, especially in the developing world, but also amongst developed countries.

Many of these studies have been flawed, however. Some have used poor measures of health care inputs, transforming national expenditures to common units using inappropriate price measures. Others have used measures such as number of hospital beds or hospital lengths of stay and compared them across countries although such comparisons are most likely inappropriate. Hospitals serve different functions in different countries, so comparing hospital related statistics across countries is not very enlightening Finally, few studies have taken account of the fact that the production of health exhibits diminishing returns and thus few have used functional forms that allow for such diminishing returns.

In this study we have estimated a production function for health which is a function of pharmaceutical consumption, other health care consumption, gross domestic product, and three lifestyle measures: alcohol consumption, the percentage of the population which smokes, and the richness of diet. Purchasing power parity exchange rates for pharmaceuticals and health care were used to transform pharmaceutical expenditures and health care expenditures to U. S. dollars. The dependent variables were life expectancies at birth, at age 40, and at age 60, and infant mortality.

In a sample of twenty one OECD countries, it was 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. 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 quite robust. In a sample comprised of only the sixteen European countries for which complete data were available, these elasticities were higher (0.023 for age 40 and 0.050 for age 60) and pharmaceutical consumption even had a small positive significant effect on life expectancy at birth. A significant effect of pharmaceutical consumption on infant mortality was not found, although it appears that, controlling for lifestyle factors, increased pharmaceutical consumption may be related to slightly increased infant mortality. Unfortunately, the infant mortality model is not robust to small changes, which does not inspire much confidence.

Gross domestic product was found to have a positive and significant effect on life expectancies at the ages of 40 and 60, although this effect was not present in the European-only sample. The results from the infant mortality regressions were mixed. We found no effect of non-pharmaceutical health care consumption in any of the life expectancy regressions. However, we did find that it had a negative effect on infant mortality in one specification.

The lifestyle variable with the biggest effect on health is dietary richness, measured by the consumption of animal fat. Increased richness of diet improves mortality up to a point but the impact becomes negative as a diet becomes very rich. This result is consistent with the idea of the epidemiological transition: the idea that at low nutritional levels, enriching a diet allows one to better fight off infections, but that at high nutritional levels, enriching a diet leads to a greater incidence of degenerative diseases such as cancer and heart disease. This result is slightly surprising. One might have thought that the OECD countries were wealthy enough that nutrition, in this basic sense, would not be an issue.

We believe that this study will add to the debate over how OECD governments should allocate ever more scarce medical care resources. It improves on much of the existing literature in that it uses better measures of pharmaceutical and other health care 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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