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**The Productivity of Health Care and Pharmaceuticals: Quality of Life,
Cause of Death and the Role of Obesity***

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I. Introduction and Summary

In an earlier study, sponsored by Sciences Po de Paris, we used data from the Organization for Economic Cooperation and Development (OECD) to study the effects of per capita consumption of pharmaceuticals and other health care on objective, but crude, measures of health (Frech and Miller 1999; Miller and Frech 2000). Our crude measures of health were life expectancies at birth, at age 40, and at age 60, and infant mortality. We found that increased pharmaceutical consumption is both economically and statistically significantly related to increased life expectancy at the ages of 40 and 60, even when controlling for per capita income and lifestyle factors such as smoking, alcohol consumption, and diet. In contrast, we found no economically or statistically significant effect of non-pharmaceutical health care consumption. Our analysis of infant mortality was not robust. Among other problems, infant mortality is not consistently measured, not even in the rich countries. Our earlier study has raised as many questions as it has answered. In this analysis, we extend that earlier work to answer some of these questions. We also replicate the original work with later data and a slightly improved model that allows us to take account of the large international variation in obesity.

We go beyond simple mortality to include the quality of life as a measure of health. At the time of our earlier work, there were no data on this. But now, thanks to the work of the World Health Organization (WHO), there are consistent data on disability-adjusted life expectancy (DALE) for many countries, including all the OECD countries. In a natural extension of the early work, we examine the production of DALE. We find that pharmaceutical consumption is more powerful in extending DALE than life

expectancy. We also find that the results on life expectancy are even more robust and stronger than in the original research.

We also extend the original work in a different direction by investigating how the effects of pharmaceutical consumption vary by disease (cause of death). Here, we cannot look at quality of life. We find that the productivity of pharmaceutical consumption varies greatly by both cause of death and by age. For individuals under 70 years of age, pharmaceutical consumption is very helpful in circulatory disease, but has little effect on cancer or respiratory disease. At later ages, pharmaceutical consumption is generally productive.

II. Literature Review

There have been many international, cross-country studies of health care. Mostly driven by budgetary concerns, they have largely focused on the determinants of health care expenditures.¹ Less attention has been placed on estimating the determinants of health itself. Scholars from a number of disciplines have done work in this area and the results have generally been mixed. We have already analyzed much of this literature in our earlier work, especially Frech and Miller (1999, pp. 2-21). In this section we summarize the main conclusions from that analysis, then review our own work, and finally review other studies, particularly the newer ones that are most relevant.

Conclusions from our Earlier Literature Review.

¹ See, for example, Joseph Newhouse (1977); David Parkin, Allistar McGuire and Brian Yule (1987); Ulf-G Gerdtham and B. Jonsson (1992) and Ulf-G Gerdtham (1991).

Certain overall results from earlier studies stand out as robust and sensible. In studies including poor countries, public health infrastructure such as potable water supplies and sanitation are the most powerful determinants of health. Expansion of health care services has much less effect. Per capita income and education are also more powerful than medical care. Diet and other lifestyle variables, such as tobacco and alcohol consumption, have also been found to be important. The small impact of medical care has led to the conventional wisdom in health economics that medical care does little to improve life expectancy. But, a few good studies have found medical care to matter, including a closely related one using OECD data (Zweifel and Ferrari 1992).

The closest precursor to our earlier study was by Akira Babazona and Alan Hillman (1994). Unusual for its time, it disaggregated health care to examine effects on perinatal and infant mortality and life expectancy. In contrast to our study, this one found no effect of pharmaceutical consumption on health measures. But, this study was marred by the use of inappropriate functional form and seriously incorrect pharmaceutical prices, as was shown by our own work. Specifically, Babazona and Hillman converted domestic pharmaceutical prices, expressed in domestic currency, to a common currency by using the overall purchasing power parity exchange rate. This would be fine if real pharmaceutical prices were the same in all markets, but, as we have shown, this is far from the case. In a more recent paper, Anders Anell and Michael Willis (2000, p. 772) make the same mistake, justifying it by the argument that there is a world market in pharmaceuticals. While this is true, cross-country differences in price controls and price discrimination produce large differences in real prices.

Our Earlier Work.

Of course, the most relevant literature for our study is our own earlier work. In that analysis, we used OECD life expectancy and infant mortality data on 21 countries as of the early 1990s. We converted pharmaceutical and non-pharmaceutical health care expenditures to U.S. dollars by using purchasing power parity (PPP) exchange rates specifically for pharmaceutical and all health care expenditures.

The pharmaceutical PPP exchange rates are imperfect, but extensive analysis showed them to be by far the best available for more than a handful of countries. This is important because the relative price of drugs is very different across countries and earlier studies had mis-measured pharmaceutical consumption by using the wrong exchange rate.

We measured health by several crude, but objectively observable variables: life expectancy at birth, age 40 and age 60 and by infant mortality. These health measures were from 1993. We used multivariate regressions to estimate production relationships. To do this, we used a log-log functional form that allows for diminishing marginal returns to each input. Aside from the health care variables discussed above, the regressions include gross domestic product (GDP) and several lifestyle variables: tobacco and alcohol consumption and the fat content of diet. These explanatory variable measures were from the 1983 to 1985 time period, thus they were lagged by 8 to 10 years.

The main finding was that pharmaceutical consumption has a surprisingly powerful impact on life expectancy of adults (age 40 and 60). This result was both statistically and economically significant. (Effects were smaller and statistically insignificant for life expectancy at birth.) Quantitatively, the elasticities were 0.02 at age

40 and 0.04 at age 60. Thus, a doubling of pharmaceutical use would increase life expectancy at age 40 by about 2 percent and at age 60 by about 4 percent.

The result can also be expressed in terms of additional dollars spent on pharmaceuticals per life year saved. Doing so highlights differences across countries. In high pharmaceutical consumption countries, the cost of saving a life year by additional consumption is much higher than in low consumption countries. The estimates (1990 dollars, for males) range from \$3,800 in Turkey to \$60,000 in France. In the U.S., a middling country in pharmaceutical consumption, the cost of saving a life year is \$21,000.

Sensitivity analysis revealed that the results were fairly robust. The results were not sensitive to small changes in the specification, such as adding more controlling variables (population over 65, unemployment, educational level) or excluding the lifestyle variables. Further, it was not sensitive to either dropping Turkey (an outlier) or to dropping all non-European observations. Consistent with our finding of large differences in pharmaceutical pricing across countries, the results were sensitive to using a general PPP exchange rate, in place of the pharmaceutical-specific one. Using a general exchange rate, rather than the pharmaceutical one is one of the major weaknesses of the Balazona and Hillman (1994) study. Furthermore, results were sensitive to using a linear functional form that forced constant returns to the inputs. This was also a weakness of the Balazona and Hillman (1994) study.

The analysis of infant mortality, on the other hand, was not successful. Signs and magnitudes of effects were very sensitive to small, and easily defended, variations in specification. Infant mortality seems a poor measure of health. By construction, this

measurement problem also afflicts the data on life expectancy at birth, but not life expectancy at later years.

Our results also indicated that non-pharmaceutical health consumption has no measurable effect on life expectancy at any age. Its effects on infant mortality were unstable. GDP, on the other hand, has positive and statistically significant effects on life expectancy, though it disappears in the Europe-only sample. The effect is larger for life expectancy at older ages. The lifestyle variable with the biggest effect is fat consumption. Interestingly, it is non-monotonic. At low levels, more fat consumption increases life expectancy, but at high levels it reduces it. This is fairly surprising. One might have thought that the OECD countries were all wealthy enough that nutrition, in this basic sense, would not have been an issue.

Recent Aggregate Studies of the Production of Health

Studies within the United States. Frank Lichtenberg (2000a) performed a time-series analysis of the production of health in the U.S. As far as we know, this is unique. He modeled life expectancy at birth over the 1960-1997 time period as a function of lagged health care expenditures and U.S. Federal Drug Administration (FDA) new drug approvals. Health expenditures were disaggregated into public v. private, but not into pharmaceutical v. other expenditures. The analysis was done both separately for whites and blacks and also pooled. Lichtenberg finds large positive and statistically significant effects of both variables. He finds that the long-run elasticity of life expectancy with respect to health care spending is about 0.07, which is more than double our estimates.

Expressed conveniently as the marginal cost of a life year saved by health care expenditures is quite low, about \$11,000.

For a life year saved by pharmaceutical innovation, obtaining a cost per life year saved involves an extra step, putting a dollar cost to a new drug approval. Citing conventional beliefs that it currently costs about \$500 million to get FDA approval for a new drug, he calculates the cost of a life year saved at a very low \$1,345.

Lichtenberg cites other authors for the proposition that the value of a life year saved is about \$150,000. Thus, both more health care spending and more drug approvals apparently score extremely well in terms of average cost/benefit ratio. Extending this reasoning to the margin for pharmaceutical research requires a strong assumption of constant returns to research.

The results are robust to the inclusion of GDP per capita (the effects of which are very imprecisely estimated) and a time trend. While the text indicates that the results include a correction for first-degree autocorrelation of the errors, the equation presents a correction for a moving-average error process.

Lichtenberg interprets the result on new drug approvals as representing (or perhaps embodying) technical progress in health care in general. If one views technical progress as resulting entirely from medical R&D spending, rather than learning-by-doing and exogenous innovation from other scientific research (e.g. basic chemistry), this roughly triples the cost per life year saved. But medical R&D is still a great bargain if one can take the estimates literally.

These results, along with Or's (discussed below), show medical spending to be far more productive than cross-sectional studies show. We are concerned that these results

may be spurious, caused by regressions involving heavily trended, nonstationary variables for which there may be unit root problems (Pyndyck and Rubinfeld 1998, pp. 508-516; Hamilton 1994, pp. 557-562; Kennedy 1998, pp. 268-270). Both GDP and health expenditures per capita have been generally found to have units roots in both country-by-country and panel tests (Hansen and King 1996, 1998; Bloomqvist and Carter 1997; Gerdtham and Lothgren 2000, MacDonald and Hopkins 2002).² We would expect similar findings for life expectancy, since it clearly trends upward.

International Studies. In a study similar to our work in focusing on OECD countries, Zeynep Or (2000, 2001) estimated a pooled, cross-country, time-series model of the production of health. She used premature mortality (Potential Years of Life Lost, PYLL) as the dependent variable, rather than life expectancy.

Or's sample is 21 OECD countries, spanning 1970-1992. The main data source is the OECD, with the PYLL calculated from unpublished mortality statistics from the WHO. Suicide is excluded.

The primary method used was OLS with fixed effects, with dummy variables for each country. The model was log-log, so that coefficients are elasticities. Separate regressions were run for men and women. The model included variables for health care spending, the proportion public, GDP and lifestyle and environmental variables. A novel inclusion is a variable for the proportion of workers in white-collar jobs as a measure of social status and education.

She finds large effects of both medical expenditures and of GDP, especially for women. The elasticity of PYLL with respect to aggregate medical spending for women

² McCoskey and Selden (1998) reach a contrary conclusion

is large at -0.1771, but small at -0.0375 for men. The estimate for women is statistically significant at high levels ($t = -4.5$), while it is statistically weak for men ($t = -1.1$). Direct comparison of our earlier results is difficult, since life expectancies and PYLL are not exactly inverses, even though both are expressed as years. Further, we disaggregate health care. Still, it is safe to say that Or's point estimates for women are far higher than ours, while the estimates for men are comparable (though very imprecise). The proportion of public spending was also important.

The proportion of workers in white-collar jobs was very important, with an elasticity of -0.8098 for women and -0.7441 for men, both highly significant (t statistics greater in absolute value than 10.0). Or interprets this as an environmental variable (in the sense of labor economics, i.e. measuring the social and intellectual environment of an individual). We believe it also likely picks up some aspects of real income that are not measured by GDP (e.g. a pleasant and safe workplace). In a later paper, discussed below, Or (2001) shows that the simple correlation between GDP and proportion white collar is very high, 0.8393. GDP per capita had about half the effect of white-collar work, though still much larger than in our study, and was also highly significant. Lifestyle variables and a variable for air pollution also had the expected effects. Country dummy variables were very important.

In a later paper, Or (2001) expanded her reach. She used a slightly shorter panel and a different econometric technique (Feasible Generalized Least Squares) to account for heteroskedasticity and serial correlation. Also, she considered other dependent variables, life expectancy at 65, PYLL by cause for cancer and heart disease and perinatal and infant mortality. And she entered some variables to account for the type of health

care system (e.g. fee-for-service versus global budgeting for hospitals). Perhaps more importantly, she replaced spending on health care with the doctor/population ratio. The results were generally consistent with her earlier study, but with even larger effects of physicians than the effects of health spending. The elasticities of PYLL with respect to the doctor/population rate are -0.3761 for women and -0.2751 for men. Note that the results for men are much stronger than in her previous study. The effect on infant and perinatal mortality is even higher, as is the effect on PYLL due to heart disease, while the effect on cancer is smaller. Also, all of these results, except for male PYLL due to cancer, are statistically significant at the 1.0 percent level.

The inclusion of the other health measures is helpful for comparisons. The elasticities of life expectancy at 65 with respect to the doctor/population ratio are much lower than the PYLL elasticities, at 0.1005 for women and 0.1007 for men. But, this is still a comparatively large effect. It is a bit more than twice as big as our elasticity for life expectancy at 60 with respect to pharmaceutical consumption. The variables for the type of system were weak and not robust. Sensitivity analyses, comparing this econometric method to fixed effects, show generally similar results.

Or offers several sensible interpretations for the strong showing of doctor/population ratio. We find most interesting and most convincing the idea that higher doctor/population ratios are correlated with more use of high tech medicine. This is similar in spirit to Lichtenberg's (2000a) interpretation of new drug approvals in the U.S. as being the result of general medical research.

It seems that we can rule out the competing idea that medical equipment is the instrument of medical research. Perhaps surprisingly, the doctor/population ratio among

rich countries is not strongly related to the use of high tech equipment. For example, comparing the U.S. to France on a per capita basis, in 1996, the U.S. had about seven times as many MRI machines. In 1991, the U.S. had about almost four times as many CT scanners, yet the U.S. had fewer doctors (Anell and Willis 2000, p. 773).

We suspect that the doctor/population ratio is highly correlated with pharmaceutical consumption. Many pharmaceuticals embody the results of recent research. Explicit consideration of pharmaceutical consumption would be very interesting, but it cannot be done for more than a very small number of years, due to the lack of data on pharmaceutical purchasing parity exchange rates.

The fact that in both studies, the results were similar using country (but not time) fixed effects suggests that most of the explanatory power is coming from time-series variation. While the econometric problems of this approach are not fully worked out for panel data, this suggests possible unit root problems with trended data, leading to spurious results. As mentioned above, in the discussion of the Lichtenberg study, studies focusing on the determinants of health care spending have generally found units roots in both GDP and health expenditures in both country-by-country and panel data (Hansen and King 1996, 1998; Bloomqvist and Carter 1997; Gerdtham and Lothgren 2000, MacDonald and Hopkins 2002). We would expect the same for life expectancy, since it is strongly trended. Unit roots may lead to spurious regression results (Pyndyck and Rubinfeld 1998, pp. 508-516; Hamilton 1994, pp. 557-562; Kennedy 1998, pp. 268-270). Further, A.G. Bloomqvist and R.A.L. Carter (1997, pp. 221, 225-226) argue that, in this context, OLS is asymptotically biased and inefficient and that the data strongly reject the

pooling assumption, even with country fixed effects included. Thus, the apparently strong results of both Or and Lichtenberg may be statistical artifacts.

Epidemiological Studies of Risk Factors

An alternative way of looking at the production of health, at least for some of the causal factors, is the epidemiological technique of calculating the risk attributable to exposure to certain risk factors. Murray and Lopez have analyzed disability-adjusted life years (DALYs) in this manner (1997c, 1999). Based on this analysis, the largest cause of DALYs in the established market economies is tobacco use (11.7 percent), followed by alcohol use (10.3 percent), occupation (5.0 percent), physical inactivity (3.8 percent) and hypertension (3.9 percent). Interestingly, air pollution is only in eighth rank, responsible for only 0.5 percent of DALYs (Murray and Lopez 1997c, p. 1440).

Note that physical inactivity and hypertension are related to obesity. This is convenient because objective measures of obesity are available, both in time-series and cross-section. This may make it possible to capture much of the variation in these two risk factors by including obesity in a statistical analysis.

The epidemiological method, called attributable burden (Murray and Lopez 1999; 1997c, 1436-1437) is interesting, but crude and subjective. It is based on the opinions of experts in various fields, which are, in turn, at least partly based on epidemiological studies of specific risk factors. The attributable burden is defined as the difference in burden between what is observed and what would occur with some specified reference exposure to the risk factor. Theoretically, the reference exposure could be anything, including zero. Murray and Lopez do not use zero, but rather some lower level that is

viewed as somehow attainable. This is equivalent to assuming that the risk factors affect health in an additively separable fashion and that the production function is a step function, where the extent of exposure doesn't matter, but only whether there is exposure or not. While the method is subjective, it allows analysts to crudely summarize the information in many different small-scale studies and surveys. The results are, at the least, suggestive for those estimating health production relations using ordinary statistical techniques.

Obesity. Obesity is a particularly interesting risk factor, since many countries have recently experienced growth in obesity. Among the rich countries the U.S. has the highest obesity rates, but, whereas a few years ago it was in a class by itself, both the U.K. and Australia are catching up quickly. There is surprisingly large variation in obesity rates, even among the rich countries.

A person is typically considered obese if his or her body mass index (BMI) is 30 or larger. The BMI is simply the ratio of an individual's weight in kilograms to the square of that individual's height in meters. For example, a BMI of 30 corresponds to a person five feet, five inches tall, weighing 180 lbs. (1.65 meters, 81.65 kg), or to a person five feet, 10 inches tall, weighing 207 lbs. (1.77 meters, 93.89 kg). Overweight is similarly defined as a BMI of over 25, corresponding to 173 lbs. for the five foot, ten inch person.

Both the level and the growth in obesity rates are impressive (see Figure 1). For example, in 1999, 26.0 percent of U.S. adults were obese, up from 14.5 percent in 1978-80. In the U.K., the obesity rate was 21.0 percent in 2000, up from only 7.0 percent in

1980. In Australia, it was 20.8 percent in 1999, up from 7.1 percent in 1980. At the other end of the scale (no pun intended), only 2.9 percent of Japanese adults were obese in 2000, up from 2.0 percent (OECD Health Data 2002, website, table 5). Even in the poor countries, obesity is becoming a major problem (Winslow and Landers 2002, Hill and Peters 1998).

Obesity can be viewed as an intermediate output, resulting from a combination of overeating and physical inactivity. Physical inactivity has been studied separately (Murray and Lopez, 1997c, 1999; Manning, Keeler, Newhouse, Sloss and Wassterman 1991, pp. 107-126). J. Michael McGinnis and William Foege (1993) used epidemiological methods to estimate that obesity causes 14 percent of deaths in the U.S., ranking number two in lifestyle or consumer choice variables (behind smoking, but well ahead of alcohol consumption).

In a large-scale, multivariate analysis, Roland Sturm (2002) has examined the effect of obesity on health status and health care use in U.S. data. He finds that obesity leads to far worse performance on two measures of health status, a count of the number of seventeen chronic health problems and a physical health scale. In both cases, obesity leads to far worse outcomes than smoking, problem drinking or overweight. In fact, obesity has the same effects on health as an extra twenty or thirty years of aging. Turning to health care use, the effects of obesity are especially striking. Obesity leads to 36 percent more total health care consumption and a whopping 77 percent more pharmaceutical consumption. Smoking, the next most important lifestyle variable, raises total consumption by 21 percent and pharmaceutical use by 28 percent. Sturm finds

being overweight to be far less serious, with statistically insignificant point estimates of less than one third of the effects of obesity.

The economic theory of obesity has recently gotten attention from Tomas Philpson and Richard Posner (1999). They model obesity as being encouraged by technical change that simultaneously has made the consumption of calories both easier and cheaper, while discouraging physical activity by substituting light for heavy work. They analyze the comparative statics of consumers' choice of body weight. Thus, they explain the increase in obesity in recent years in a static, fully utility-maximizing framework. This is a nice example of how powerful standard economic theory is for comparative statics--explaining behavioral changes in response to change in the constraints.

Though he doesn't apply the idea to obesity, Sam Peltzman (2001) has recently written about health-related behavior that partly offsets improvement in the productivity of health care. He argues that when health risks are reduced, consumers partly offset the health gains by less healthy behavior. He points to accidents, suicides and homicides, but a sedentary lifestyle and overeating may be more important, especially since the most important recent improvements in health care seem to be in the circulatory disease area.

However, other aspects of obesity are more problematic. Consumers themselves often voice discontent with their own obesity. This could be explained as simply a problem in the economics of self-control. Consumers' tastes may change over time in an inconsistent way, such that their planned diet may not be carried out. Robert Strotz (1955-1956) presented a nice early statement of this problem. For more recent consideration of these problems see Becker and Mulligan (1997) or Laibson (1997).

The apparent time-inconsistency of overeating is given a very interesting evolutionary interpretation by Trent Smith (2001). He suggests that the biological mechanisms underlying the sensations of hunger and satiation have evolved to serve humans well when faced with periodic famines. Now that famines are exceedingly rare in the rich countries, the biological mechanisms lead to obesity. Consumers are still behaving rationally, given their sensations of hunger. It is simply that these feelings have been programmed by evolution to lead to excessive fat under the new conditions of long-term plentiful food.

Other Studies of the Productivity of Pharmaceuticals

Frank Lichtenberg (2000b, 2001, forthcoming) has studied the productivity of pharmaceutical innovation (not consumption) in novel ways, exploiting variation across diseases within the U.S. In one of these studies (2000b), He uses data from a large-scale, detailed survey of U.S. consumers that contained three waves of interviews over a short time period. Using the prescription as the unit of analysis, rather than the individual, he examined the effect of using new drugs, rather than older drugs. He found significantly less mortality (by the end of the third wave of the study), controlling for individual characteristics, including details of the diagnosis. He also found that using new drugs is related to lower disability and to lower spending on other types of health care. This analysis is hard compare to our analyses. Think of the mortality result. He shows that using newer drugs leads to lower mortality per prescription (called a Prescribed Medicine Event in the survey). It is hard to know how this translates into population mortality or life expectancy.

In part of the study, Lichtenberg introduces dummy variables for each individual and studies the effect of new drugs on all the measures (except mortality). He finds similar beneficial effects of newer drugs. This is even harder to interpret.

Further, there is a major problem in interpreting the coefficient causally because of omitted variable bias. Suppose that healthier people, within each diagnosis, are more likely to get the newer drug. This could happen if the people with milder forms of the illness have only recently been put on drug therapy. They would have no history of using the older drug, thus they might naturally be given the newer drug. In this situation, all of Lichtenberg's results might obtain, purely because of the correlation of the newness of the drug and the mildness of the illness. On the other hand, people with worse forms of the illness might get the newer drugs if the newer drugs are more powerful and more costly. Lichtenberg acknowledges this problem in the paper, but argues that the literature on small area variations (which shows that the amount of health care varies greatly by geographic area) suggests that it may be acceptable to treat the data as if it were generated by a random assignment of people to new versus old drugs. This strikes us as a big leap.

We find the second study (forthcoming) both easier to interpret and more convincing, though it still can't be directly compared to our work. In this analysis, Lichtenberg relates the change in the potential years of life lost (PYLL) from 1970 to 1991 to the proportion of drugs, which are classified as new (FDA approval after 1970), prescribed for each diagnosis. Each of the 80 observations is an aggregate diagnosis (ICD9 two digit level disease). He finds strong effects, explaining almost half of the variation. Quantitatively, the effects are large. For the quartile with the highest new drug

use, the PYLL declined by 72.7 percent. At the other extreme, the PYLL declined by only 13.0 percent. This indicates high productivity for drug research. Using the conservative value of \$25,000 per life year saved and an average cost of a new drug approval of \$667 million, he finds the social rate of return to pharmaceutical innovation is about 40 percent.³ While not directly comparable to our results on the consumption of pharmaceuticals, it is certainly consistent with our finding that more consumption raises life expectancy. Also note that in the calculation of the social rate of return to pharmaceutical innovation, Lichtenberg ignores the fact that the innovations benefit consumers in the entire world, not merely the U.S. As in the choice of a dollar value per life year, this is a conservative approach. Adjusting for the worldwide health benefits would show even higher benefits to pharmaceutical research and development.

III. Extending Our Earlier Work

As we have already stated, the results of our previous analyses indicate that individuals living in countries with higher per capita pharmaceutical consumption can expect to live longer lives. These results left us with more questions to consider.

First, does pharmaceutical or other health care consumption have a bigger effect on the quality of life than on the length of life? Second, are the relationships the same across all causes of death, or does it differ across different causes of death?

³ Kevin Murphy and Robert Topel (forthcoming) have recently argued that the value of a life year gained is much higher, at \$150,000-\$200,000 (in the U.S.). Cutler and McClellan (2001) have recently used a value of \$100,000 per disability-free life year.

New Measures of Health

Quality of Life. Mortality-based measures miss completely the morbidity dimension of health that is, we believe, more sensitive to pharmaceutical consumption and other health care than mortality.⁴ Of course, there are a number of direct morbidity measures (e.g. work days lost, subjective evaluations of health). Many of these measures are so partial and context-specific that they can't even be meaningfully compared across countries, let alone be used in aggregate production of health analysis.

Therefore, in order to study the relationship between pharmaceutical consumption and quality of life we consider another class of health measures. The basic idea behind these measures is to adjust years lost from premature mortality or life expectancy for the amount of time spent in imperfect health. When the focus is on years lost or gained, the measure is called quality-adjusted life years (QALYs). QALYs are created by multiplying the number of life years by a weight reflecting the quality of life (the opposite of morbidity) (Johannesson 1996, pp. 117-218).

There are many approaches to finding the weights to employ. Ideally, the weights can be obtained using special types of surveys, posing hypothetical choices to actual consumers. Sometimes, the weights come from non-choice type surveys or from opinions of researchers (e.g. physicians). All the methods are problematical. QALY estimation requires sophisticated multi-dimensional measurement and weighing of quality of life (Cameron et. al. 1997). This is particularly difficult to interpret across cultures and across long periods of time. At best, QALYs can be viewed as an approximation to the number of healthy years.

⁴ For an argument that ignoring quality of life understates health benefits by 30 percent see David Cutler and Elizabeth Richardson (1997, p. 262).

Another measure focuses on disability as a way of reducing the dimensionality of the quality measures. The result, though it is logically a special case of a QALY, has been given its own name. It is called disability-adjusted life years (DALYs). DALYs are defined as the disability-adjusted life years lost, when compared to a reference ideal situation of no disability and living to some age limit, interpreted as a full potential life span. Thus, they are similar to the Potential Years of Life Lost (PYLL) measure of premature mortality, which is discussed below, except that the premature mortality adjusted for disability.

When applied to life expectancy, this approach yields the disability-adjusted life expectancy (DALE). Perhaps most important, this approach has been applied in a consistent manner to most of the countries in the world as part of the World Health Organization's Global Burden of Disease Project. These measures are designed for aggregate comparisons of the burden of disease and to be used in studies of resource allocation in health care. Both the method of construction and the purpose of the measures are described and defended by Christopher Murray and Arnab Acharya (1997). Thus, the DALE is a reasonable measure that is available across countries.

The weights used in the construction of the DALEs are based on the altruistic or social values of a reference group of (mostly physician) health care providers convened in Geneva. (I.e. the measures are not based on valuations by consumers themselves.)⁵ Weights differ by age, with higher weights for young adults (Murray and Acharya 1997, pp. 712-719). Further, future ill health is discounted at 3 percent (though there is an alternative measure of DALYs using zero percent discounting and equal age weights).

⁵ The use of medical experts has been criticized as unrepresentative of actual consumers by David Cutler and Elizabeth Richardson (1997, pp. 251-252).

When the two measures are applied to calculate the burden of diseases, they are highly correlated across diseases (Murray and Acharya 1997, pp. 719-726).

Cause-Specific Mortality. The World Health Organization (WHO) regularly collects mortality data from over 100 countries. Mortality rates are available by cause and are disaggregated by age and gender. For instance, the data set contains information on lung cancer mortality among French men age 65 to 74 and on ischemic heart disease mortality for Swedish women age 55 to 64.

Another measure of mortality, which is calculated from these age-gender specific mortality rates, is known as potential years of life lost (PYLL). In effect, PYLL is a weighted mortality rate, with the weights equal to the difference between a fixed measure of potential life span (which varies by study, mostly between 65 and 85 years) and the time of actual death. Note that extending life after the fixed potential life span is implicitly given a value of zero using this measure. If one had data on a panel of countries over time, PYLL would be expressed algebraically as a rate per 100,000 population as

$$(1) \quad PYLL = \sum_{a=0}^{l-1} (l-a)(d_{at} / p_{at})(P_a / P_n) * 100000,$$

where:

a = age,

l = the age limit,

d_{at} = the number of deaths at age a ,

p_{at} = the number of persons aged a in country i at time t ,

P_a = the number of persons aged a in the country,

P_n = the total number of persons aged 0 to $l-1$ in the

country.

PYLL has the important advantage of being well defined for different causes of death, while life expectancy only makes sense on an overall basis. Using WHO mortality data, the OECD routinely calculates PYLL measures for a number of different causes of death, using the age of 70 as its age limit.

Because there are many causes of death, we have decided to focus on those causes that are most prevalent in the 21 OECD countries that we had included in our previous analysis. Data limitations have forced us to leave Turkey out of our present analyses, leaving a sample of 20 countries.⁶ In Table 1, we present the average mortality rates (per 100,000 population) among the 20 countries for 14 different causes of death. We find, not surprisingly, that circulatory disease is the leading cause of death in these countries, accounting for 40 percent of all deaths in 1994. Cancer is the second leading cause of death, accounting for 26 percent, and respiratory disease is a distant third, accounting for 8 percent of all deaths. Taken together, these three causes accounted for about three-quarters of all deaths in 1994. Because of their prevalence, we use these cause-specific mortalities in our present study.

IV. Modeling the Production of Health

As in our previous work, we have based our analysis on the standard household production model of health. In this model, the level of an individual's health is determined by his or her consumption of medical care goods and services as well as

⁶ Neither the WHO nor OECD could provide cause-specific mortality data for Turkey.

environmental and life-style factors. Aggregating up to the national level yields the following model to explain the variation in health levels across countries:

$$(2) \quad H_i = \alpha + \beta MC_i + \gamma X_i + \varepsilon_i,$$

where H_i is the measure of the average health of the citizens of country i , MC_i is a vector of the average consumption of various types of medical care by the citizens of country i , X_i is a vector of life-style or environmental variables for country i , and ε_i is a random error term.

Health Indicators. As we stated in the last section, we use a new set of health indicators in this study. The data for these health indicators came from both the WHO and OECD. We estimated models for disability-adjusted life expectancy (DALE) at birth and at age 60. We obtained the DALE measures from the WHO (2000). The DALEs come from the 1998-99 time period. In addition to the DALE models, we also estimated models for life expectancy at birth and at age 40 and 60, using life expectancy data from 1997 to 1999. We did this for two reasons. First, we wanted to be able compare the impacts of medical care inputs and other factors on the life expectancy and DALE measures. Second, in this work, the data are newer than in our previous study. Also, we use a slightly different model, so that these results are a check on the robustness of our previous results.

These new measures include circulatory disease, cancer, and respiratory disease. We estimated potential years of life lost (PYLL) models for each of these three leading causes of death. The PYLLs were obtained from the OECD. As we mentioned earlier, in calculating PYLLs the OECD considers deaths before the age of 70 to be “preventable,”

and therefore sets 70 as the potential life span.⁷ This is a fairly short life span, so that the effects of pharmaceuticals and other health care that is focused on older consumers will be missed. Because of this limitation, we also examine separate models for cause-specific mortality rates at particular ages: age 35 to 54; age 55 to 64; age 65 to 74; and age 75 and up. All of these cause-specific mortality measures come from the 1994-96 time period. These mortality rates were obtained from the WHO. Not surprisingly, the results of the models for the PYLLs were very similar to the results for the age 35-54 and age 55-64 mortality rates. Therefore, for the sake of brevity, we report the results for the PYLLs, capturing effects at younger ages and the mortalities at ages 65-74 and 75 and over, capturing effects at older ages.

Note that interpreting the age-specific mortality for these later years is biased downward for inputs that improve health. The population base for the later years' mortality includes people who were healthy enough to have survived to these later ages. For example, suppose that high health care consumption leads to many survivors aged 65-74. But, some of the survivors are in fragile health and, absent the aggressive health care, would not have survived. This could lead to higher mortality at the 65-74 ages as a result of successful health care. Of course, the problem is even worse for the last age category, ages 75 or more.

Medical Care Inputs. We list all explanatory variables in Table 2. As in our previous study, we focus here on two medical care inputs: consumption of pharmaceuticals and consumption of other medical care. The data on pharmaceutical and other medical care consumption come from the OECD (2000). Based on the results of our

⁷ In other words it sets l equal to 70 in equation (1).

previous work, we create a measure of pharmaceutical consumption by converting 1990 per capita expenditures on pharmaceuticals to US dollars using pharmaceutical purchasing power parity (PPP) exchange rates provided by the OECD. We create a measure of other medical care consumption in 2 steps. First, we convert 1990 per capita expenditures on medical care to US dollars using medical care PPP exchange rates. We then subtract our pharmaceutical consumption measure from this figure.

Other Explanatory Variables. We also include four measures of living standards and lifestyle factors. First, we include each nation's 1990 per capita gross domestic product (GDP) to 1990 U.S. dollars using each nation's 1990 GDP purchasing power parity exchange rate. We also control for cigarette smoking by including the percentages of females and males aged fifteen years or older that smoke as of the period around 1990. As we noted in our previous work, we prefer measuring smoking in this way because most health researchers believe that the adverse effects of smoking begin at low levels of consumption. The effect of switching from ten cigarettes a day to two packs a day is small, while the effect of switching from a non-smoker to smoking ten cigarettes a day is large. The percentage of the population who smokes captures this inherent non-linearity better than a measure that simply measures the average tobacco consumption in grams per person per day.

Another lifestyle factor we control for is alcohol consumption. Alcohol consumption is measured as per capita consumption in liters. Data on the percentage of adults who consume alcohol do not exist for enough of the countries in our sample. (For alcohol, unlike smoking, there is not a clear a priori reason to prefer a percentage

measure.) Finally, we control for differences in female and male mortality rates across countries.

In our earlier work we controlled for richness of diet, and tried to proxy for obesity, by including a measure of animal fat calories consumed per capita per day. At the time of our earlier study, data on obesity levels were quite sparse. Now, enough such data exist so that we can use this measure. The only drawback is that we had to drop two additional countries from our sample, Germany and Greece. Still, the models that included obesity levels generally performed better than the models that included the animal fat calorie measures. Our measure is quite standard. As discussed above, it is the percentage of the population that is obese, defined as a body mass index (BMI) of 30 or more.

The Model Specification and Estimation. We use regression analysis to determine the effect of each of the explanatory variables on each of the health indicators. We lag the explanatory variables by roughly 5 to 10 years because we believe that lifestyle factors and medical care consumption have a cumulative rather than an instantaneous effect on health. A full model would require several lags of each explanatory variable. Due to data and sample size limitations this is impossible. The implicit assumption we are making here is that cross-national variations in the values of the explanatory variables as of 1990 reflect their historical cross-national variations.

Also, as we did in our previous work, we use a log-log, or constant elasticity, functional form. There are two advantages to this specification. First, a coefficient from a log-log regression is interpreted as an elasticity: the percentage change in the dependent

variable associated with a 1 percent change in the value of an explanatory variable. Second, a model for the production of health should allow for diminishing returns to all of the independent variables. In the log-log model, the elasticity is held constant and the absolute value of the marginal effect of each explanatory variable is forced to fall at higher and higher values of the explanatory variable. The data to which one applies such a model determines the rate at which the marginal effect decreases.

Finally, in our regression analyses, we pool our data across sexes and include an indicator variable, *FEMALE*, equal to one for observations on female health outcomes and equal to zero for observations on male health outcomes. We do this because, as a rule, the effects of the various explanatory variables do not differ significantly by sex except for alcohol consumption. We include an interaction term between the gender indicator variable and alcohol consumption, to capture this. It should be noted that *SMOKE* (see Table 2) is equal to the percentage of females who smoke for those observations where *FEMALE* equals one and equal to the percentage of males who smoke for those observations where *FEMALE* equals zero. Likewise, *OBESITY* is equal to the percentage of females who are obese (BMI exceeds 30) for those observations where *FEMALE* equals one and equal to the percentage of males who are obese for those observations where *FEMALE* equals zero.

One would expect mild heteroskedasticity in this data, i.e. that the error terms are not identically distributed. Further, because we have pooled observations on male and female health outcomes, there are two observations for each of the 18 countries in our sample. It is possible, even likely, that the within-country observations are not independent because of unobserved country effects.

These problems do not create bias or inconsistency in the estimated beta coefficients, but they can lead to problems in the estimated standard errors. We correct for these problems by estimating the standard errors using a version of the robust heteroscedasticity-consistent covariance estimator, which was introduced by Huber (1967) and further developed by White (1980). Rogers (1993) notes that one can use a version of this estimator when relaxing the assumptions of both identically and independently distributed error terms. In our case, we need only assume that the observations are independent across countries.

V. Results

Descriptive Statistics

See Tables 3, 4, and 5 for descriptive statistics. The full dataset is reproduced in Appendix A. Descriptive statistics in Tables 3 and 4 indicate that there is much variation in cause-specific mortality rate for both men and women among the 18 OECD countries we used in our final analyses. Another thing to note is that although circulatory disease is the leading cause of death in these countries, cancer is actually a greater cause of premature mortality, especially among women. Cancer is the cause of over 1,100 PYLLs (before the age of 70) per 100,000 women whereas circulatory disease is the cause of only about 469 PYLLs per 100,000 women. The difference is smaller among men but cancer is the leading cause of premature mortality among men as well. The respiratory disease mortality rates are the smallest, but they also exhibit the greatest variation as measured by their coefficients of variation. Of course, the male mortality rates are mostly

higher than the female mortality rates. Another finding from Tables 4 and 5 is that DALEs exhibit slightly greater variation than do the life expectancies.

The descriptive statistics in Table 5 indicate that there is a good deal of variation in the explanatory variables as well. Pharmaceutical consumption per capita, for example, varies by a factor of over six, from \$105.20 in Ireland to \$664.60 in France. Gross domestic product varies by a factor of over two, from \$9,598 in Portugal to \$22,266 in the United States. Other health care consumption varies by a factor of almost four, from \$714.30 in Portugal to \$2,515.00 in the United States.

Lifestyles also vary widely in our sample. Men in Spain are twice as likely to smoke as are men in Sweden and women in Denmark are seven times more likely to smoke than are women in Portugal. The French consume more than three times the alcohol per capita than do the Norwegians.

Obesity, once again, is particularly interesting. The U.S. has the highest obesity rates by far, with 25.1 percent of women being obese, more than double the mean of 10.7 percent and 67 percent higher than the next highest, the U.K. The story is similar for men. In the U.S., 19.9 percent of adult men are obese, again more than double the mean of 9.49 percent and 50 percent higher than the next highest, Canada. Several European countries have far lower obesity rates; the obesity rates for Swedish men and for Swiss women are only 5.4 and 4.7 percent, respectively. Note that our data are from various years in the early 1990s. As discussed above, recent trends indicate rapid increases in obesity rates worldwide, with the U.K. and Australia, in particular, catching up by 2000.

Table 6 presents simple cross-correlations among the explanatory variables. Most of the correlations are not significantly different from zero, although the results indicate

that countries with higher pharmaceutical consumption appear to have lower tobacco use rates among females and higher rates of alcohol consumption. Richer countries tend to spend more on non-pharmaceutical health care goods and services, but, surprisingly, not on pharmaceuticals. Also, it is interesting to note that male and female obesity rates are highly correlated whereas male and female tobacco use rates are not. This indicates that certain bad health habits, such as over-eating and a sedentary lifestyle, may be culturally ingrained, whereas others like smoking are not. It is also interesting to note that the male smoking rate is positively correlated with the rate of alcohol consumption.

Empirical Results for Disability-Adjusted and Unadjusted Life Expectancy

The Effect of Lifestyle. The results for disability adjusted and unadjusted life expectancy are presented in Table 7. The new results for life expectancy are useful to compare with the DALE results as well as with the results of our earlier study. The lifestyle effect with the biggest impact on both life expectancy and on DALE is obesity. The countries with higher levels of obesity tend to have both shorter life expectancies and shorter DALEs. The results indicate that lowering obesity levels by ten percent, from the OECD averages of 10 percent to about 9 percent, would increase disability adjusted life expectancy at birth by about 0.2 percent and at age 60 by about 0.5 percent. This would raise the average DALE at birth by about 52 days for women and about 48 days for men. The average DALE at age 60 would increase by 34 days for women and by 27 days for men.

Note also that the obesity elasticities are higher for the DALEs than for the normal life expectancies. Because the mean value of DALE is lower than unadjusted life

expectancy, this finding does not necessarily imply that marginal effects, measured in days of life, are bigger in the DALE models, but we have found it to be the case here. The 10 percent decrease in obesity rates would increase female life expectancy at birth by 44 days and male life expectancy at birth by 41 days. It would also increase life expectancy at age 60 by 15 days for women and by 12 days for men.

The other lifestyle variables do not matter much in explaining either life expectancy or the DALE measure. We estimate fairly similar effects of alcohol consumption on life expectancies as we did in the earlier study, but none of them is precisely estimated, with standard errors that exceed the elasticity estimates. The results are similar for the DALEs. We also find similar results for smoking as we did in the previous study. The elasticity estimates are small and swamped by the standard errors. In the life expectancy regressions, we also find similar results for the effect of wealth (gross domestic product) but, unlike in the earlier study, none of the point estimates are precise enough for us to make much of them. Also, we still have the same problem of co linearity between per capita GDP and non-pharmaceutical health care consumption. Interestingly the point estimates for gross domestic product are smaller in the comparable DALE models.

The Effect of Non-Pharmaceutical Medical Care Consumption. As we found in our earlier study, non-pharmaceutical medical care consumption does not have a statistically significant effect on life expectancies at even the 10 percent level of significance. While the point estimates are larger in the newer study, the estimates are

very imprecise. In the DALE models, the point estimates are slightly higher, but the estimates are still too imprecisely measured to say anything definite.

These point estimates are sensitive to the inclusion or exclusion of per capita GDP, which is not surprising given the very high correlation between these two measures. For instance, when we exclude per capita GDP, the measured effect of non-pharmaceutical health care consumption jumps from an elasticity of 0.044 to 0.065 and becomes statistically significant at the 10 percent level. Because of this, one should use caution in interpreting the results for both per capita GDP and non-pharmaceutical health care consumption.

The Effect of Pharmaceutical Consumption. In contrast to this, pharmaceutical consumption appears to be productive, and the effect is robust. As we did in our earlier study (with different data and a slightly different specification), we find that pharmaceutical consumption has no discernible effect on life expectancy at birth, but it does have a positive and statistically significant relationship with life expectancy at the ages of 40 and 60. Increasing per capita pharmaceutical expenditures by 10 percent would increase life expectancy at age 40 by 0.3 percent, and life expectancy at age 60 by 0.6 percent. This would increase life expectancy at age 40 by 46 days for women and by 40 days for men. Life expectancy at age 60 would increase by 51 days for women and by 42 days for men.

These results on life expectancy are consistent with our earlier work, though slightly stronger. In our earlier work, a 10 percent increase in pharmaceutical consumption led to an increase in life expectancy of about 2 percent at age 40 and about

4 percent at age 60. Further, the newer results are more precise (Frech and Miller 1999, p. 42).

The results are even more striking for disability-adjusted life expectancy. A 10 percent increase in pharmaceutical consumption would increase the DALE at *birth* by 0.2 percent. This would increase the DALE at birth by 50 days for women and by 47 days for men. This same 10 percent increase in drug consumption would increase the DALE at age 60 by nearly 0.9 percent. The DALE at age 60 would increase by 62 days for women and by 51 days for men. These results, taken together, indicate that pharmaceutical consumption not only prolongs life, but also improves the quality of that life.

In Tables 8 and 9 we present marginal effects of pharmaceutical consumption on life expectancies and DALEs – measured in days per additional 1990 US dollar spent on pharmaceuticals – for each country. We find that countries like France, which consume the most pharmaceuticals, stand to gain the least from increased drug consumption, whereas those countries that consume fewer drugs stand to gain more. For instance, increasing pharmaceutical consumption by one dollar would increase the DALE at age 60 in Ireland by 5.2 days for women and by 4.3 days for men. In France, such an increase would only improve the DALE at age 60 by 1.1 days for women and by 0.8 days for men. The results are similar for all five life expectancy measures. Note that the marginal effect generally increases with age. As an example, on average across all countries, an additional unit of pharmaceutical consumption increases the DALE for women by about 2.1 days at birth and by about 2.6 days at age 60.

In Tables 10 and 11, we present estimates of the lifetime per capita pharmaceutical expenditures necessary to increase each of our life expectancy and DALE

measures by one year. See Figure 2 for selected countries. The estimates are fairly conservative because they are based on the assumption that pharmaceutical expenditures are constant over the entire lifetimes of the individuals. The results tell the same story as those reported in Tables 8 and 9. The highest expenditures are necessary in France and Italy – where the marginal effects are smallest – and the lowest expenditures are necessary in Ireland and Denmark – where the marginal effects are largest.

These estimates for life expectancy can be compared to our earlier work. They show the same pattern as in our earlier work. The main difference is that the newer work indicates slightly lower costs per life year saved. To give an example, for 40 year-old females in the U.S., the earlier work showed that the cost of additional year of life expectancy was \$21,165 (Frech and Miller, 1999, p. 51). In the newer work, the estimate is \$15,952. Note that all of our estimates are well below current estimates for the value of a life year, in the neighborhood of \$150,000 in the U.S., as discussed above. Next, we turn to a finer level of detail--the determinants of life years lost and age-specific mortality by cause of death.

Empirical Results for Circulatory Disease Mortality

The Effect of Lifestyle. Table 12 presents our results for circulatory disease mortality. Not surprisingly, the lifestyle variable with the greatest effect this type of mortality is obesity. The results indicate that countries with greater obesity levels also have significantly higher levels of circulatory disease mortality, at least up to the age of 74. Obesity seems to harm health much more for younger people. Lowering obesity rates by 10 percent, from the sample average of 10 percent to an average of 9 percent,

would decrease the potential years of life lost before 70 by nearly 4 percent; by nearly 18 years per 100,000 women and by 46 years per 100,000 men.

Lowering obesity rates by 10 percent would also lower the circulatory disease mortality rate among 65 to 74 year-olds by about 1.6 percent, lowering the average death rates by about 10 deaths per 100,000 women and by about 21 deaths per 100,000 men in this age group. The results indicate obesity has little effect on circulatory disease mortality for those aged 75 and older. As discussed earlier, the effects on mortality of the elderly is biased downward, especially where there is a large effect at the younger ages.

The point estimates also indicate that alcoholic consumption may have a small negative effect on circulatory disease mortality and that this effect does not vary across men and women. The negative effect is statistically significant in the age 65-74 mortality equation, yielding an elasticity of -0.17 , but imprecisely estimated in the PYLL and age 75 and over mortality equations. In our earlier work, we found that alcohol consumption actually led to an increase in over-all mortality (decreased life expectancy), which we found surprising given epidemiological research showing that moderate drinking substantially reduces the risk of heart disease. These results here begin to provide an answer to this puzzle, as it appears that alcohol consumption does reduce mortality due to heart and circulatory disease.

The effects of smoking are very imprecisely estimated in our models. The only model where we find anything near a significant result is in the model for circulatory disease for those over 74 years of age. Here we find a significant negative effect of smoking, which is puzzling. Still, this is our worst-performing model for circulatory disease mortality with an R-square statistic of only 0.55—the other models boast R-

squares of over 0.90—so that may explain this result. The effects of wealth are also quite imprecisely estimated.

The Effect of Non-Pharmaceutical Medical Care Consumption. Our results indicate that non-pharmaceutical medical care consumption has no statistically significant effect on premature circulatory disease mortality (PYLL), even at the 10 percent level. It is almost significant at the 10 percent level for the oldest, and fairly large, with an elasticity of -0.35 . Again, the point estimates effects are very sensitive to the inclusion or exclusion of per capita GDP. As we stated earlier, caution is called for in interpreting the results for both per capita GDP and non-pharmaceutical health care consumption.

The Effect of Pharmaceutical Consumption. Pharmaceutical consumption appears productive for circulatory disease. This is consistent with the widely held view that medical advances have been especially successful in treating this class of disease.⁸ Pharmaceutical consumption is negatively associated with both premature circulatory disease mortality and mortality among the elderly. Increasing per capita pharmaceutical consumption by 10 percent, from about \$238 to about \$262, would decrease the potential years of life lost before 70 by nearly 2 percent: by about 9 years per 100,000 women and by about 23 years per 100,000 men. Such an increase in per capita pharmaceutical consumption would also lower the circulatory disease mortality rate among 65 to 74 year-olds by about 3.6 percent, lowering the average death rates by about 23 deaths per 100,000 women and by about 47 deaths per 100,000 men in this age group. The measured effect is a little lower for those 75 and older, but very precisely estimated. The point

estimate indicates a 10 percent increase in pharmaceutical consumption would lower mortality rates in this age group by about 1.5 percent, lowering the average death rates by about 64 deaths per 100,000 women and by about 75 deaths per 100,000 men.

In Figures 3 and 4 we present marginal effects of pharmaceutical consumption on circulatory disease mortality in Ireland, New Zealand, the U.S., and France. In Figure 3, we focus on premature mortality, where the marginal effect is measured as the decrease in the PYLL measure per additional dollar spent on pharmaceuticals in 1990. Paralleling our results for overall health, those countries that spend the most on pharmaceuticals stand to gain the least by increasing drug consumption while those countries that spend the least stand to gain bigger decreases in premature mortality. For instance, in France an additional dollar spent per capita on pharmaceuticals would decrease the PYLL measures by only 0.10 years per 100,000 women and by only 0.24 years per 100,000 men. In contrast to this, in Ireland an additional dollar spent on pharmaceuticals would decrease the PYLL measures by about one year per 100,000 women and by nearly three years per 100,000 men.

In Figure 4 we focus on mortality rates for those in the 65-74 and 75-and-older age groups. Here the marginal effects are measured as the decreases in the mortality rates (per 100,000 individuals) per additional dollar spent on pharmaceuticals in 1990. Generally, the same pattern follows here as in the case of premature mortality, with the higher drug consumption countries like France standing to gain less in marginal terms than low-drug-consumption countries. We see here that the marginal effect of drug consumption on circulatory disease mortality is universally higher for men and also

⁸ See, e.g. Cutler and McClellan (2001).

higher for those in the aged 75-and-over age group. Note the differences in the estimated effect across countries are larger for circulatory disease than for overall health.

Empirical Results for Cancer Mortality

The Effect of Lifestyle. We present our results for cancer mortality in Table 13. Unlike in the case of circulatory disease, each of the lifestyle variables was significant in at least two out of the three models. We find smoking, for instance, to have a tremendous effect on cancer mortality at all ages. Lowering the smoking rate by 10 percent, from the sample averages of 25.2 percent for females and 35.2 percent for males to 22.7 and 31.7 respectively, would decrease the potential years of life lost before 70 by 2.5 percent.

This same decrease in smoking rates would decrease cancer mortality among those aged 65 to 74 by 2.5 percent and the cancer mortality rate among those in the 75-and-older age group by about 1 percent. These results are not surprising given the epidemiological evidence tying tobacco use to many forms of cancer.

We also find alcohol consumption to be associated with higher rates of cancer mortality, at least among men. Decreasing alcohol consumption by 10 percent would decrease the potential years of life lost before 70 by about 2.4 percent and decrease cancer mortality among those aged 65 to 74 by 1.9 percent. The interactions between alcohol consumption and the female indicator variable indicate that alcohol consumption has no effect on either premature mortality or mortality between the ages of 65 and 74 for females. Among both men and women, this drop in alcohol consumption would lower the cancer mortality rate among those in the 75-and-older age group by about 1.4 percent.

Again, these results are not terribly surprising since alcohol consumption is also known to be a contributing factor to certain types of cancer.

As we found in the circulatory disease models, we find that obesity leads to higher cancer mortality rates, at least up to the age of 74. For example, lowering obesity rates by 10 percent, from the sample average of 10 percent to an average of 9 percent, would decrease the potential years of life lost before 70 by about 1.6 percent. This same decrease would also decrease the cancer mortality rate among individuals in the 65 to 74 age group by roughly 1.5 percent.

We also find that cancer mortality rates are smaller in richer countries, when controlling for the other lifestyle factors. The results indicate that increasing per capita wealth by 10 percent would decrease the potential years of life lost before 70 by about 6.2 percent. The magnitude and precision is generally the same for cancer mortality among individuals aged 75-and-older. Unfortunately, confidence in the coefficient on GDPPC is undermined by high correlation between it and non-pharmaceutical consumption, to which we now turn.

The Effect of Non-Pharmaceutical Medical Care Consumption. Here the results of our models are puzzling, since we find a statistically significant wrong-sign relationship between non-pharmaceutical medical care consumption and cancer mortality. At the same time, this result is very sensitive to the inclusion or exclusion of per capita GDP. The explanation for this odd result is the high degree of co linearity between the per capita GDP and medical care consumption measures. Implausibly large opposite sign

coefficients sometimes occur when there is co linearity among variables.⁹ When we dropped non-pharmaceutical medical care consumption from the model, this large negative effect of GDP almost vanished. When we dropped per capita GDP from the model, the positive effect of per capita medical consumption did vanish. This is an indication that due to co linearity it is impossible to disentangle the effects of per capita wealth and per capita non-pharmaceutical medical consumption.

The Effect of Pharmaceutical Consumption. The results indicate that pharmaceutical consumption has no statistically significant on premature cancer mortality. The point estimate has the wrong sign, but is estimated imprecisely. The standard error is about the same value as the coefficient. At the same time, the results indicate that pharmaceutical consumption does reduce cancer mortality among their elderly population. Increasing per capita pharmaceutical consumption by 10 percent would also lower the cancer mortality rate among 65 to 74 year-olds by a little over 1 percent, lowering the average death rates by about 7 deaths per 100,000 women and by about 12 deaths per 100,000 men in this age group. The measured effect is about the same for those 75 and older, and very precisely estimated. The point estimate indicates a 10 percent increase in pharmaceutical consumption would lower the average death rates by about 13 deaths per 100,000 women and by about 24 deaths per 100,000 men.

In Figure 5 we present marginal effects of pharmaceutical consumption on cancer mortality among the elderly in Ireland, New Zealand, the U.S., and France. Generally, the same pattern follows here as in the case circulatory disease mortality among this population, with the higher drug consumption countries like France gaining less than

⁹ See Greene (1993) pages 267-270.

low-drug-consumption countries from increasing pharmaceutical spending. For instance, among the 65 to 74 year olds in France, increasing per capita pharmaceutical consumption by one dollar would lower male cancer mortality by only 0.37 deaths per 100,000 and lower female cancer mortality by only 0.18 deaths per 100,000. In contrast, the corresponding decreases in male and female mortality in Ireland are 1.27 and 0.81 deaths per 100,000. We also see here that the marginal effect of drug consumption on cancer mortality is universally higher for men and also higher for those in the aged 75-and-over age group. Comparing these marginal effects of drug consumption with those we found for circulatory disease in Figure 4, we find that the marginal effect on circulatory disease mortality is generally much higher than the one on cancer mortality.

Empirical Results for Respiratory Disease Mortality

The Effect of Lifestyle. Table 14 presents our results for respiratory disease mortality. Not surprisingly, obesity has a very significant effect at all ages. For instance, lowering obesity rates by 10 percent, from the sample average of 10 percent to an average of 9 percent, would decrease the potential years of life lost before 70 by about 1.4 percent; by nearly 2 years per 100,000 women and by nearly 3 years per 100,000 men.

The effect is much bigger for mortality among those aged 65 to 74. Lowering obesity rates by 10 percent would also lower the respiratory disease mortality rate in this age group by nearly 7 percent, lowering the average death rates by about 9 deaths per 100,000 women and by about 20 deaths per 100,000 men in this age group. The effect is also large for those aged 75 and older. A 10 percent decrease in obesity rates would lower respiratory disease mortality by about 3.7 percent, lowering the average death rates by

about 30 deaths per 100,000 women and by about 52 deaths per 100,000 men in this age group.

Alcohol consumption has virtually no effect on premature respiratory disease mortality before age 70. The absolute value of the coefficient in the model for mortality at ages greater than 74 is rather large, but it is very imprecisely estimated. There is no evidence that this effect varies across genders, as the interaction term is never significant.

Not surprisingly, we find that smoking raises respiratory disease mortality rates at all ages. A 10 percent decrease in the rate of smoking, from the sample averages of 25.2 percent for females and 35.2 percent for males to 22.7 and 31.7 respectively, would decrease the potential years of life lost before 70 by 0.6 percent. This same decrease in smoking rates would have a much bigger effect on respiratory disease mortality among those aged 65 to 74, decreasing it by roughly 6.5 percent. It would also decrease the mortality rate among those in the 75-and-older age group by about 3.2 percent. These results are not surprising given the epidemiological evidence tying tobacco use to many forms of respiratory disease, especially emphysema.

The results on wealth are somewhat puzzling. Taken at face value, the results suggest that increased wealth is associated with higher premature mortality, but lower mortality among those in the 75-and-older age group. The result suggests that increasing a nation's wealth by 10 percent would decrease its mortality in this age group by 15 percent! Again, though, this result is very sensitive to the inclusion or exclusion of non-pharmaceutical medical care consumption. When this health care consumption measure is excluded from the model, the effect of per capita GDP is cut in half.

The Effect of Non-pharmaceutical Medical Care Consumption. Our results indicate that non-pharmaceutical medical care consumption lowers premature respiratory disease mortality (PYLL), but that its effects those aged 65 or older is imprecisely estimated. The results indicate that increasing non-pharmaceutical medical care consumption by 10 percent would lower premature mortality by about 2 percent. And, again, the results are not robust to the exclusion of per capita GDP. For instance, when we exclude per capita GDP from the model for mortality at ages 75 and older, we find a strong negative and significant effect on mortality. Some linear combination of wealth and health care consumption is lowering mortality for these older individuals, but disentangling the effects of the two measures is thwarted by co linearity problems.

The Effect of Pharmaceutical Consumption. The results indicate that pharmaceutical consumption has little or no effect on premature respiratory disease mortality. At the same time, they indicate that it reduces respiratory disease mortality among their elderly population. Increasing per capita pharmaceutical consumption by 10 percent would lower the mortality rate among 65 to 74 year-olds by about 3.3 percent, lowering the average death rates by about 4.5 deaths per 100,000 women and by about 10 deaths per 100,000 men in this age group. The effect is not significant for those 75 and older, since it is not very precisely estimated.

In Figure 6 we present marginal effects of pharmaceutical consumption on respiratory disease mortality among those aged 65 and 74 in Ireland, New Zealand, the U.S., and France. The same pattern follows here as in the cases of circulatory disease and cancer mortality among this population, with the higher drug consumption countries like

France standing to gain less in marginal terms than low-drug-consumption countries. We also see here that the marginal effect of drug consumption on respiratory disease mortality, as for the other causes of death, is universally higher for men. Comparing across causes of death, we find that the marginal effect on circulatory disease mortality is generally much higher than the ones on either cancer or respiratory disease mortality.

Sensitivity Testing

We already discussed some of our sensitivity testing in the preceding discussion of our results pertaining to per capita GDP and non-pharmaceutical health care consumption. Due to extreme co linearity between these two measures in our data, we found that the estimated effects of each of these measures are very sensitive to the inclusion or exclusion of the other measure. Given the instability of the results for these variables, caution should be taken in interpreting them.

In addition to this, we tried several other variants of the models. For instance, as we did in our previous study (Frech and Miller, 1999), we dropped the lifestyle variables from the models. Just like the results of the earlier study, the effects of pharmaceutical consumption are typically robust to this change. A minor exception to this is in the respiratory disease model, where dropping the lifestyle variables leads to a stronger negative effect of pharmaceutical consumption on mortality.

We also included variables for means years of education, unemployment, and nitrous oxide emissions. Of these variables education was significant only in the circulatory disease PYLL model, and the nitrous oxide emissions measure was significant in two of the respiratory disease models. Education was negatively related to premature

circulatory mortality and the emissions measure was positively related to respiratory disease mortality among the elderly. Adding these variables did not lead to changes in the results for the other variables in the models.

Following the example of Or (2001), we also replaced the non-pharmaceutical health care consumption measure with a per capita physician measure. This generally had very little impact on the models. The only case where the measure was significant was in the model for respiratory disease mortality for those aged 75 and over, where it had a strong negative impact on mortality. The estimated effects of the other variables, especially pharmaceutical consumption, were not overly sensitive to this change. Interestingly, when pharmaceutical consumption is dropped from this class of models, the effect of the per capita physician measure becomes quite pronounced in many cases. This raises the possibility that omitted variable bias may partially explain Or's strong results for the productivity of physicians.

VI. Conclusions and Summary

In this study, we have extended our earlier work in two ways. First, we have gone beyond mortality to include quality of life as a measure of health. Thanks to the work of the World Health Organization (WHO), there are now consistent international data on disability-adjusted life expectancy (DALE). In a natural extension of the early work, we examine the effects of pharmaceutical consumption on DALE at birth and at the age of 60, and compare these effects with those on life expectancy. The availability of new data also allowed us to take account of obesity (replacing animal fat consumption in the models). Studying effects with newer data and slightly different specification provides

an additional test of the robustness of our earlier work. Second, we have investigated how the effects of pharmaceutical consumption vary by cause of death.

We find that pharmaceutical consumption is very productive in increasing disability-adjusted life expectancy (DALE) both at birth and at age 60. In addition to this, we find the effect on DALE to be much larger than the effect on life expectancy. The effect on DALE at birth is more than twice as large. The effect on DALE at 60 is about 50 percent larger. A 10 percent increase in pharmaceutical consumption would increase DALE by about 9 percent, versus 6 percent for life expectancy. This supports the argument of Cutler and Richardson (1997, p. 262), mentioned above, that much of the benefit of modern health care is on quality of life. While many of the health measures are different between this study and our previous one (Frech and Miller 1999, Miller and Frech 2000), the results on life expectancy can be compared easily with our earlier study, because the specification is identical, except for replacing animal fat consumption with obesity and more recent data. In this current study, the effects of pharmaceutical consumption are both larger and estimated more precisely

Obesity, the only other variable that was consistently powerful in these models, had a large negative effect on life expectancy and DALE. The estimated effect of a 10 percent increase in obesity ranged from about 1.5 to 4.9 percent.

Looking at specific disease classes, we find that the effect of pharmaceutical consumption varies by cause of death and by age. It is most productive in reducing mortality due to circulatory disease, even among the non-elderly. A 10-percent increase in pharmaceutical consumption would decrease premature circulatory disease mortality by almost 2 percent, mortality for those aged 65 to 74 by about 3.6 percent, and mortality

for those aged 75 and older by 1.5 percent. After pharmaceutical consumption, obesity is the next most important factor in predicting circulatory disease mortality.

Pharmaceutical consumption has less effect on cancer and respiratory disease. It appears to be unrelated to premature mortality due to either cancer or respiratory disease. Still, it is productive in lowering cancer mortality among the elderly, and in lowering respiratory disease mortality among a particular age group, those aged 65 to 74.

Obesity, tobacco use, and alcohol consumption are all positively related to cancer mortality, although the effect of alcohol is only present among males. For cancer, the effects of tobacco and alcohol use tend to be greater than the effect of obesity.

It is difficult to interpret the results for other health care consumption and GDP, for any health measure, because of high co linearity between these two variables. Because of this, the estimated independent effects of each variable are sensitive to the inclusion or exclusion of the other. Given the instability of the results for these two variables, caution should be used in interpreting them.

There are some lessons to take from this analysis. First, the new work confirms our earlier results. Again, we consistently find statistically significant and economically important effects of pharmaceutical consumption on health. Second, the effect of pharmaceutical consumption on mortality varies across different causes of death, with its greatest impact on circulatory disease mortality at all ages. But it has a significant impact on cancer and respiratory disease mortality among the elderly. Third, pharmaceutical consumption does more than just extend life; it also improves the quality of life. Fourth, obesity is has strong negative effects on health.

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Table 1: Leading Causes of Death in 20 OECD Countries

Cause of Death	OECD Mean	Percentage of Total Mortality
All Causes	739.3	100.0%
Circulatory Diseases	294.7	39.9%
Cancers	195.1	26.4%
Respiratory Diseases	59.8	8.1%
Digestive System Diseases	30.6	4.1%
Endocrine and Metabolic Disorders	29.8	4.0%
Nervous System Diseases	14.2	1.9%
Mental Conditions	11.8	1.6%
Genito-urinary Conditions	10.8	1.5%
Infectious Diseases	8.0	1.1%
Congenital Anomalies	4.4	0.6%
Musculoskeletal Conditions	3.0	0.4%
Diseases of the Blood	2.6	0.4%
Diseases of the Skin	1.1	0.1%
Other causes	73.5	9.9%

Source: Authors' calculations.

Table 2: Definitions of Explanatory Variables

FEMALE	An indicator variable equal to 1 if the observation is for a female outcomes measure.
GDPPC	Gross domestic product per capita in 1990, converted to US dollars using the GDP purchasing power parity exchange rate.
PHPC	Pharmaceutical expenditures per capita in 1990, converted to US dollars using the purchasing power parity exchange rate for pharmaceuticals.
HEPC	Other health expenditures per capita in 1990, converted to US dollars using the purchasing power parity exchange rate for health care.
SMOKE	If female=1, the percentage of females age 15 and over who smoke; If female=0, the percentage of males age 15 and over who smoke.
ALCOHOL	Alcohol consumption circa 1990 measured as liters consumed per capita.
ALCOHOL *FEMALE	ALCOHOL interacted with FEMALE.
OBESITY	If female=1, the percentage of females with BMI > 30; if female=0, the percentage of males with BMI > 30.

Table 3: Descriptive Statistics for Outcomes Measures, Females

Outcomes Measure	Average	Standard Error	<i>Minimum</i>	Maximum
<hr/>				
Life Expectancy				
at Birth	80.22	1.22	77.8	81.9
at 40	41.51	1.17	39.0	43.2
at 60	23.23	1.00	21.4	24.9
Disability Adjusted Life Expectancy				
at Birth	74.06	1.59	71.2	76.9
at 60	19.04	1.34	16.6	21.7
Cancer Mortality				
PYLL	1102.24	170.53	825.0	1484.1
Age 65-74	611.70	117.06	432.5	872.9
Age > 74	1189.10	130.54	981.5	1465.3
Circulatory Disease Mortality				
PYLL	457.87	116.85	273.0	741.5
Age 65-74	638.31	153.91	331.3	939.1
Age > 74	4191.79	684.22	3139.4	5971.6
Respiratory Disease Mortality				
PYLL	119.61	47.05	62.0	210.7
Age 65-74	133.27	76.72	51.9	284.3
Age > 74	813.73	367.29	377.6	1754.5

Source: Authors' calculations.

Table 4: Descriptive Statistics for Outcomes Measures, Males

Outcomes Measure	Standard Error		Minimum	Maximum
	Average			
Life Expectancy				
at Birth	73.95	1.23	71.0	75.9
at 40	36.19	0.99	34.6	37.6
at 60	18.90	0.82	17.4	20.0
Disability Adjusted Life Expectancy				
at Birth	68.81	1.45	65.9	71.2
at 60	15.48	1.00	13.9	16.8
Cancer Mortality				
PYLL	1308.50	204.79	946.7	1764.2
Age 65-74	1113.23	122.06	854.9	1306.0
Age > 74	2289.34	247.44	1896.1	2857.4
Circulatory Disease Mortality				
PYLL	1195.17	260.68	760.0	1639.7
Age 65-74	1320.73	291.26	801.3	1978.2
Age > 74	4859.27	741.08	3558.8	6498.1
Respiratory Disease Mortality				
PYLL	192.87	71.28	112.4	333.5
Age 65-74	287.06	99.44	150.8	532.2
Age > 74	1393.63	454.90	659.4	2583.4

Source: Authors' calculations

Table 5: Descriptive Statistics for the Explanatory Variables

Variable	Mean	Standard Error	Minimum	Maximum
GDPPC (\$)	16291.1	3188.7	9598	22266
PHPC (\$)	238.3	132.3	105.2	664.6
HEPC (\$)	1741.1	474.4	714.3	2515.0
SMOKE (%)				
Female	25.2	7.6	5.8	42.0
Male	35.2	6.8	25.7	51.5
ALCOHOL (liters)	10.8	2.6	5.0	16.6
OBESITY (%)				
Female	10.1	4.8	4.7	25.1
Male	9.5	3.7	5.4	19.9

Source: Authors' calculations

Table 6: Simple Correlations Among the Explanatory Variables

	GDPPC	PHPC	HEPC	Female SMOKE	Male SMOKE	ALCOHOL	Male OBESITY	Female OBESITY
GDPPC	1.000							
PHPC	0.0929	1.000						
HEPC	0.9274**	0.1746	1.000					
Female SMOKE	0.3467	-0.4944**	0.3733	1.000				
Male SMOKE	-0.1304	0.121	-0.0545	0.2064	1.000			
ALCOHOL	-0.1134	0.5125**	-0.132	-0.1913	0.4089*	1.000		
Male OBESITY	0.1031	-0.1293	0.0553	-0.0946	-0.2971	-0.0293	1.000	
Female OBESITY	0.1082	-0.0864	0.0641	-0.2106	-0.3361	-0.0201	0.9206**	1.000

** Correlation is significantly different from zero at the 0.05 level.

* Correlation is significantly different from zero at the 0.10 level.

Source: Author's calculations

Table 7: Life Expectancy Regressions (Standard Errors in Parentheses)

Variable	Life Exp. at Birth	Life Exp. at 40	Life Exp. at 60	DALE at Birth	DALE at 60
FEMALE	0.0479 (0.0288)	0.0867 (0.0534)	0.1693** (0.0688)	0.0337 (0.0438)	0.1943 (0.1163)
GDPPC	-0.0058 (0.0259)	0.0455 (0.0506)	0.1033 (0.0705)	-0.0058 (0.0373)	0.0322 (0.1290)
PHPC	0.0086 (0.0068)	0.0302** (0.0113)	0.0607** (0.0163)	0.0186** (0.0079)	0.0896** (0.0234)
HEPC	0.0228 (0.0210)	-0.0087 (0.0347)	-0.0263 (0.0484)	0.0250 (0.0292)	0.0444 (0.0937)
SMOKE	-0.0040 (0.0109)	-0.0045 (0.0173)	0.0064 (0.0233)	-0.0071 (0.0123)	0.0078 (0.0344)
ALCOHOL	-0.0107 (0.0120)	-0.0194 (0.0215)	-0.0137 (0.0268)	-0.0118 (0.0175)	-0.0102 (0.0442)
ALCOHOL * FEMALE	0.0139 (0.0135)	0.0210 (0.0250)	0.0171 (0.0314)	0.0161 (0.0197)	0.0073 (0.0515)
OBESITY	-0.0153** (0.0055)	-0.0191* (0.0098)	-0.0176 (0.0136)	-0.0192** (0.0065)	-0.0485** (0.0163)
CONSTANT	4.2170** (0.1428)	3.1540** (0.2729)	1.8549** (0.3819)	4.0971** (0.1908)	1.7176** (0.6407)
R-SQUARED	0.928	0.922	0.938	0.872	0.883

** Coefficient is significant at the 0.05 level.

* Coefficient is significant at the 0.10 level.

Source: Authors' calculations.

Table 8: Marginal Effect of Pharmaceutical Consumption on Life Expectancy Measures, Females (Days per additional 1990 US Dollar spent)

Country	Life Expectancies			DALEs	
	At Birth	At Forty	At Sixty	At Birth	At Sixty
Australia	1.29	2.36	2.67	2.61	3.36
Austria	1.27	2.30	2.57	2.56	3.10
Belgium	0.83	1.52	1.73	1.66	2.11
Canada	1.18	2.17	2.50	2.33	2.87
Denmark	2.16	3.81	4.20	4.30	4.98
Finland	1.32	2.39	2.66	2.62	3.17
France	0.39	0.72	0.83	0.79	1.07
Ireland	2.35	4.17	4.53	4.63	5.16
Italy	0.57	1.04	1.17	1.14	1.45
Netherlands	1.94	3.47	3.88	3.88	4.95
New Zealand	1.39	2.53	2.84	2.70	3.10
Norway	1.49	2.71	3.03	2.98	3.79
Portugal	0.99	1.80	1.98	2.00	2.35
Spain	0.89	1.65	1.87	1.79	2.29
Sweden	1.13	2.07	2.34	2.25	2.84
Switzerland	1.35	2.49	2.85	2.69	3.54
UK	1.36	2.43	2.70	2.72	3.31
USA	1.04	1.87	2.11	2.05	2.51
Average	1.06	1.92	2.16	2.11	2.61

Source: Authors' calculations

Table 9: Marginal Effect of Pharmaceutical Consumption on Life Expectancy Measures, Males (Days per additional 1990 US Dollar spent)

Country	Life Expectancies			DALEs	
	At Birth	At Forty	At Sixty	At Birth	At Sixty
Australia	1.20	2.09	2.20	2.45	2.80
Austria	1.17	1.99	2.10	2.37	2.52
Belgium	0.76	1.31	1.38	1.53	1.70
Canada	1.10	1.92	2.04	2.20	2.43
Denmark	2.02	3.38	3.46	4.04	4.12
Finland	1.20	2.01	2.10	2.39	2.49
France	0.35	0.60	0.66	0.71	0.83
Ireland	2.18	3.67	3.67	4.36	4.32
Italy	0.52	0.91	0.95	1.06	1.18
Netherlands	1.80	3.02	3.08	3.63	3.87
New Zealand	1.30	2.26	2.36	2.54	2.63
Norway	1.38	2.37	2.46	2.74	2.90
Portugal	0.90	1.55	1.62	1.81	1.86
Spain	0.81	1.42	1.53	1.65	1.91
Sweden	1.05	1.84	1.94	2.14	2.43
Switzerland	1.24	2.18	2.33	2.48	2.75
UK	1.27	2.16	2.22	2.58	2.79
USA	0.95	1.64	1.76	1.91	2.04
Average	0.97	1.67	1.76	1.96	2.12

Source: Authors' Calculations

Table 10: Lifetime Cost of Extending Life (or Disability Adjusted Life) by 1 Year, Females (in 1990 US Dollars)

Country	Life Expectancies			DALEs	
	At Birth	At Forty	At Sixty	At Birth	At Sixty
Australia	23,118	12,835	11,562	10,698	8,810
Austria	23,243	13,043	11,916	10,757	9,391
Belgium	35,844	19,923	17,871	16,588	13,973
Canada	25,384	14,029	12,471	11,750	10,174
Denmark	13,290	7,664	7,158	6,151	5,726
Finland	22,446	12,580	11,508	10,389	9,144
France	78,219	42,890	37,769	36,194	28,266
Ireland	12,384	7,070	6,648	5,733	5,487
Italy	52,722	29,205	26,369	24,399	20,321
Netherlands	15,326	8,621	7,883	7,093	5,952
New Zealand	21,112	11,860	10,788	9,775	9,182
Norway	20,030	11,161	10,142	9,271	7,779
Portugal	29,056	16,481	15,335	13,447	12,243
Spain	33,829	18,591	16,621	15,656	12,942
Sweden	26,585	14,710	13,217	12,305	10,365
Switzerland	22,419	12,320	10,950	10,376	8,420
UK	21,631	12,242	11,269	10,011	8,775
USA	28,259	15,952	14,486	13,080	11,558
Average	28,054	15,684	14,234	12,984	11,180

Source: Authors' calculations

Table 11: Lifetime Cost of Extending Life (or Disability Adjusted Life) by 1 Year, Males, in 1990 US Dollars

Country	Life Expectancies			DALEs	
	At Birth	At Forty	At Sixty	At Birth	At Sixty
Australia	23,140	13,670	13,356	10,707	10,150
Austria	23,269	14,045	13,863	10,769	11,046
Belgium	35,884	21,532	21,205	16,607	16,517
Canada	25,409	14,948	14,443	11,760	11,583
Denmark	13,302	8,164	8,302	6,157	6,670
Finland	22,475	13,752	13,727	10,403	11,080
France	78,321	46,860	44,850	36,245	34,348
Ireland	12,397	7,562	7,807	5,738	6,325
Italy	52,777	31,354	30,821	24,424	23,821
Netherlands	15,341	9,262	9,373	7,100	7,208
New Zealand	21,131	12,571	12,388	9,784	10,479
Norway	20,051	11,946	11,851	9,281	9,571
Portugal	29,094	17,796	17,840	13,467	14,752
Spain	33,869	20,091	19,323	15,674	14,855
Sweden	26,609	15,634	15,184	12,313	11,673
Switzerland	22,443	13,185	12,709	10,388	10,231
UK	21,651	13,012	13,061	10,019	10,017
USA	28,292	17,099	16,581	13,094	13,571
Average	28,084	16,830	16,597	12,998	13,140

Source: Authors' calculations

Table 12: Circulatory Disease Mortality Regressions (Standard Errors in Parentheses)

Variable	PYLL	Mortality Age 65-74	Mortality Age >74
FEMALE	-0.9830** (0.2961)	-0.7115** (0.2729)	-0.3772 (0.2421)
GDPPC	-0.1628 (0.4160)	0.0018 (0.4177)	0.3649 (0.2895)
PHPC	-0.1912** (0.0582)	-0.3597** (0.0680)	-0.1542** (0.0597)
HEPC	0.0134 (0.2583)	-0.1034 (0.2787)	-0.3479 (0.2109)
SMOKE	-0.0596 (0.0770)	-0.1123 (0.0928)	-0.1718** (0.0754)
ALCOHOL	-0.1270 (0.1162)	-0.1703* (0.0925)	-0.0580 (0.1015)
ALCOHOL *FEMALE	-0.0088 (0.1353)	-0.0301 (0.1245)	0.0707 (0.1126)
OBESITY	0.3861** (0.0822)	0.1608** (0.0670)	-0.0692 (0.0475)
CONSTANT	9.2313** (2.1978)	10.2879** (2.1271)	9.2515** (1.4069)
R-SQUARED	0.932	0.907	0.553

** Coefficient is significant at the 0.05 level.

* Coefficient is significant at the 0.10 level.

Source: Authors' calculations

Table 13: Cancer Mortality Regressions (Standard Errors in Parentheses)

Variable	PYLL	Mortality Age 65-74	Mortality Age >74
FEMALE	0.6594* (0.3355)	0.0188 (0.2670)	-0.5671** (0.2086)
GDPPC	-0.6205** (0.1983)	-0.2832 (0.2459)	-0.6373** (0.1922)
PHPC	0.0528 (0.0548)	-0.1106* (0.0598)	-0.1052** (0.0328)
HEPC	0.2457** (0.1211)	0.2246 (0.1716)	0.4688** (0.1374)
SMOKE	0.2549** (0.1011)	0.2444** (0.0810)	0.1060** (0.0487)
ALCOHOL	0.2370** (0.1123)	0.1933** (0.0845)	0.1443* (0.0730)
ALCOHOL *FEMALE	-0.3153** (0.1548)	-0.2307* (0.1251)	-0.0213 (0.0913)
OBESITY	0.1568** (0.0363)	0.1465** (0.0330)	0.0485 (0.0341)
CONSTANT	9.2624** (1.1648)	7.0373** (1.3556)	10.1653** (0.9838)
R-SQUARED	0.682	0.926	0.964

** Coefficient is significant at the 0.05 level.

* Coefficient is significant at the 0.10 level.

Source: Authors' calculations

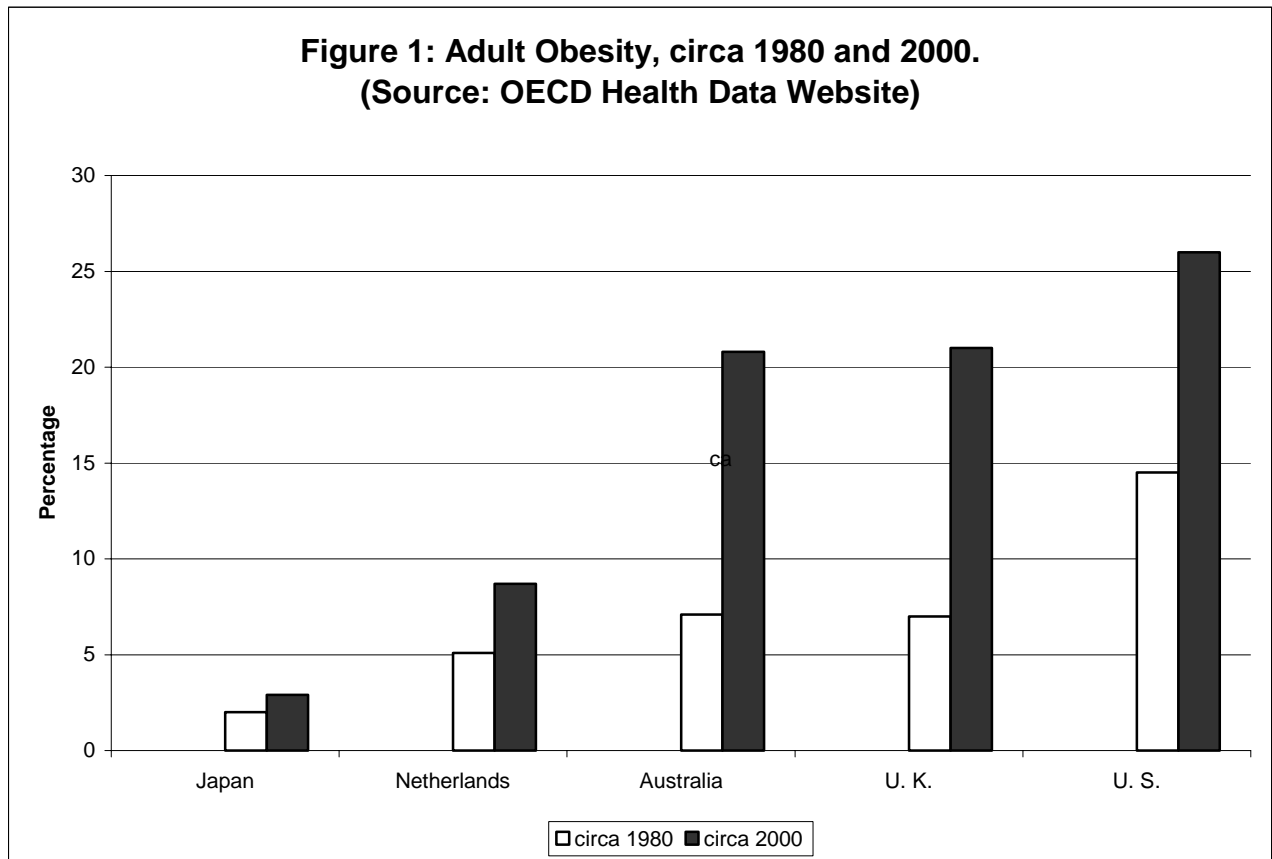
Table 14: Respiratory Disease Mortality Regressions (Standard Errors in Parentheses)

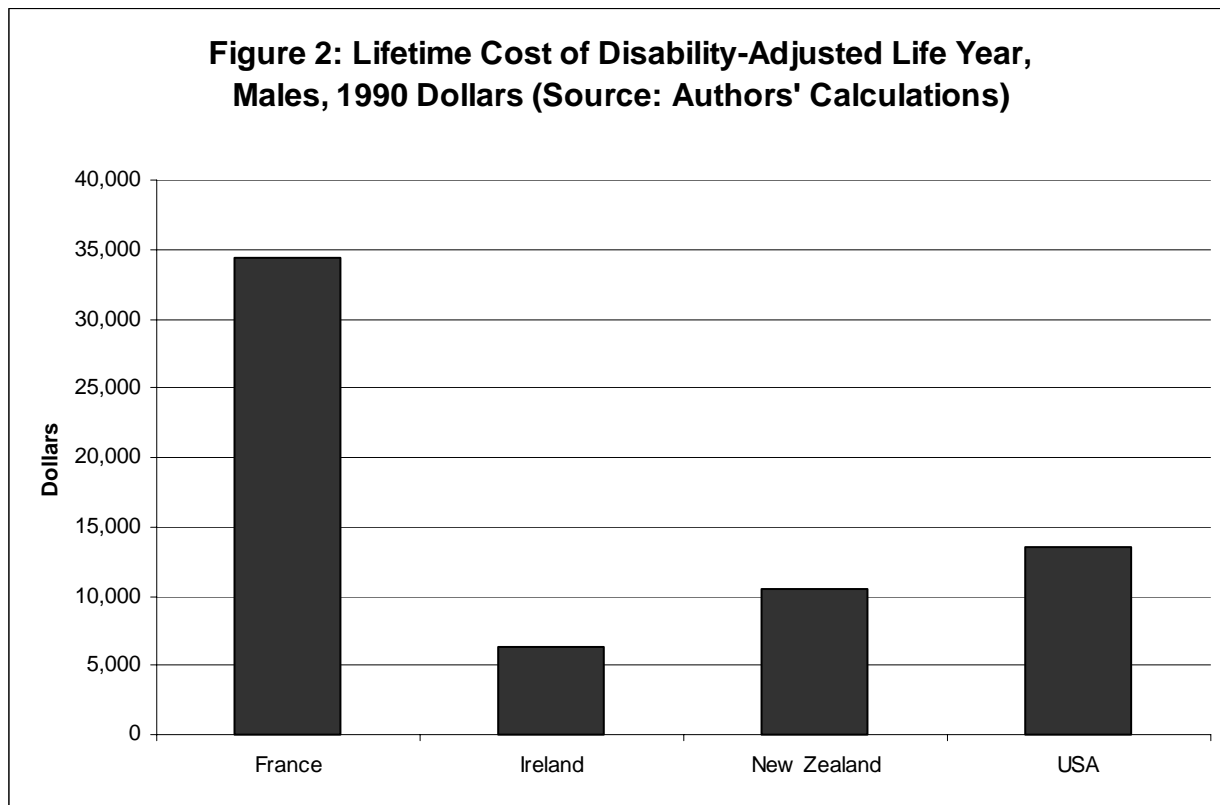
Variable	PYLL	Mortality Age 65-74	Mortality Age >74
FEMALE	0.0504 (0.1030)	0.3112 (0.6537)	-0.1676 (0.6295)
GDPPC	0.1563** (0.0732)	-0.5702 (0.6142)	-1.5299** (0.6335)
PHPC	0.0075 (0.0188)	-0.3352** (0.1362)	-0.1531 (0.1277)
HEPC	-0.2041** (0.0480)	-0.0693 (0.4524)	0.4982 (0.4881)
SMOKE	0.0626** (0.0284)	0.6467** (0.2176)	0.3224** (0.1619)
ALCOHOL	0.0072 (0.0332)	-0.1003 (0.2050)	-0.3383 (0.2229)
ALCOHOL *FEMALE	-0.0555 (0.0484)	-0.4060 (0.3082)	-0.1261 (0.2863)
OBESITY	0.1403** (0.0158)	0.6856** (0.0934)	0.3723** (0.1013)
CONSTANT	1.0621** (0.3985)	9.8795** (3.1533)	17.9606** (3.2147)
R-SQUARED	0.836	0.857	0.714

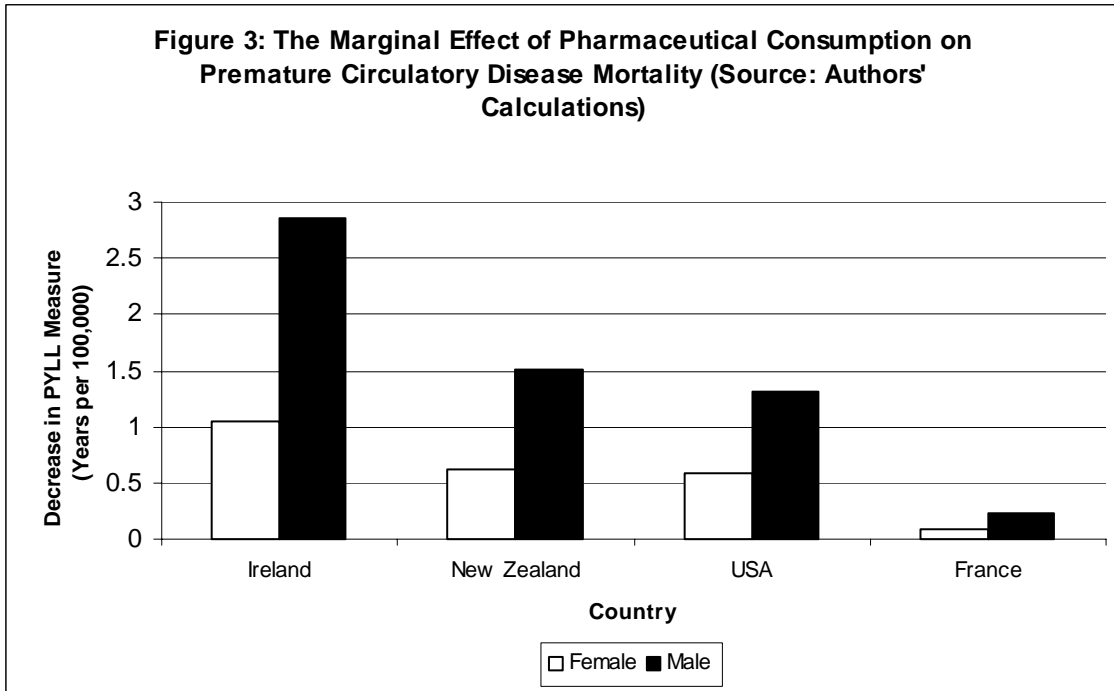
** Coefficient is significant at the 0.05 level.

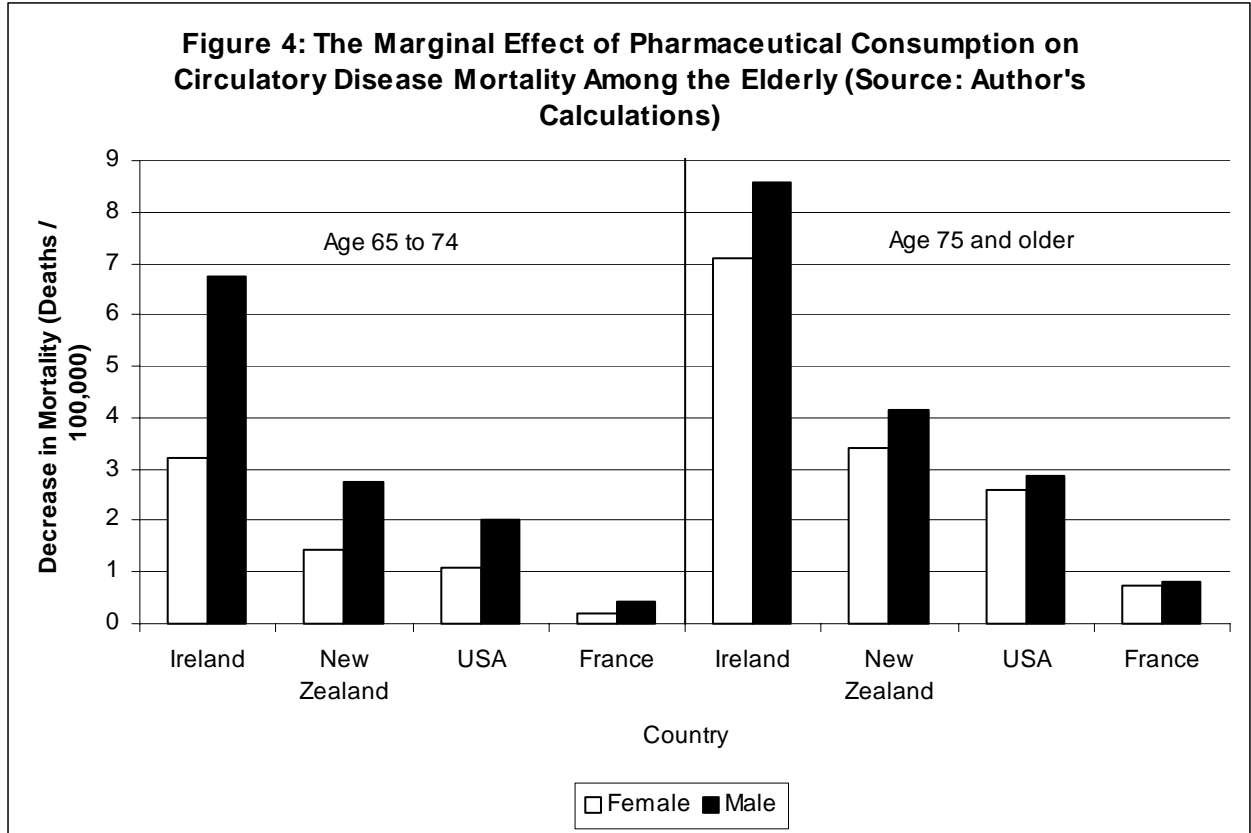
Source: Authors' calculations

Figure 1: Adult Obesity, circa 1980 and 2000.
(Source: OECD Health Data Website)









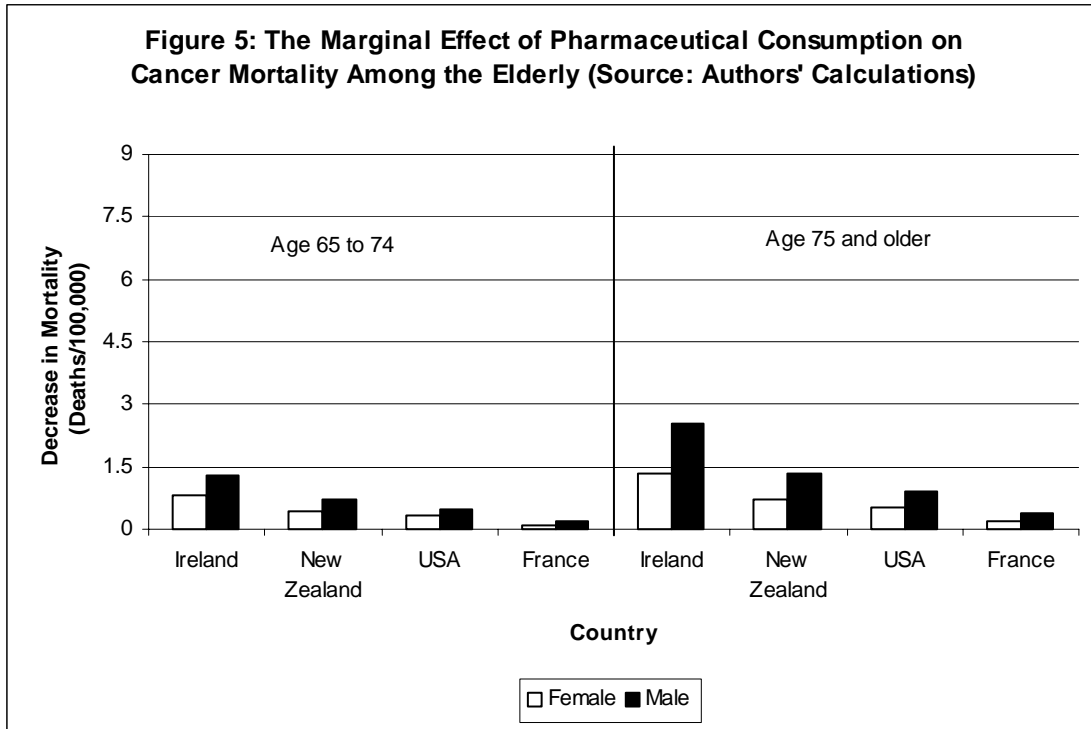
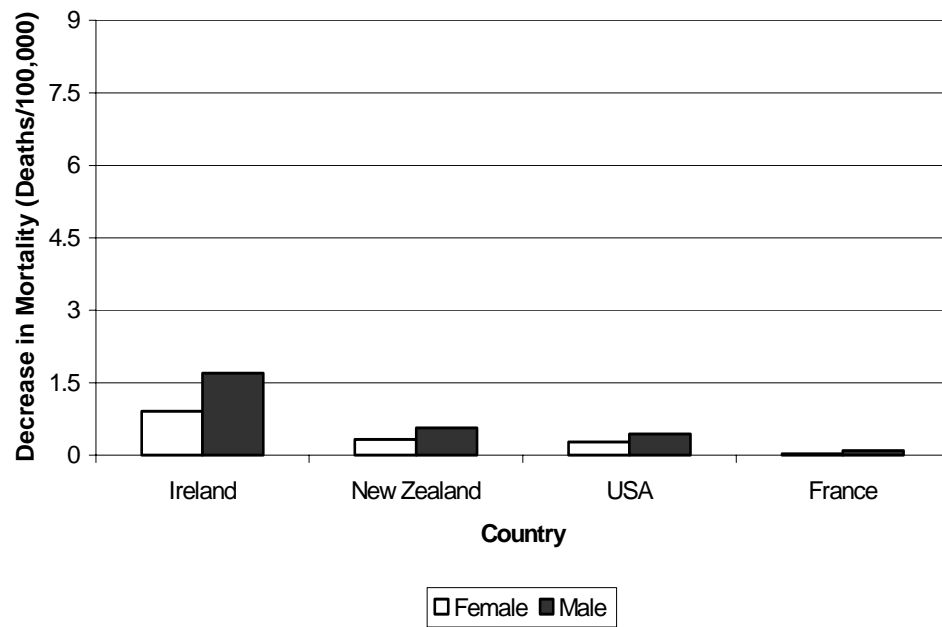


Figure 6: The Marginal Effect of Pharmaceutical Consumption on Respiratory Disease Mortality, Ages 65 to 74



Appendix A: The Data Used in the Analyses

In Tables A-1 through A-5, we list the data that we used in our analyses. Table A-1 lists the life expectancy and DALE measures for each of the countries in our sample. The DALE measures were collected by the World Health Organization (2000) and reflect 1999 levels. The life expectancy measures were compiled by the OECD (2000) and reflect 1995 levels.

Tables A-2 through A-4 present the circulatory disease, cancer, and respiratory disease mortality measures for each of our countries. The potential years of life lost (PYLL) measures were compiled by the OECD and we obtained the age-specific mortality rates from the WHO website. The PYLL measures all reflect 1994 levels. The age-specific mortality rates generally reflect 1995 levels with the following exceptions. For Austria, Canada, Finland, Portugal, Sweden, and the United Kingdom, the mortality rates reflect 1996 levels. The mortality rate for Belgium is for 1994.

Table A-5 presents the explanatory variable measures for the countries in our sample. The per capita measures for GDP, pharmaceutical expenditures, and other health expenditures are all from 1990 as is the measure for alcohol consumption. The male and female smoking data reflect 1990 levels with the following exceptions. The smoking data for Australia and Spain are from 1989. The smoking data for Austria are for 1991, and the smoking data for Portugal are a linear extrapolation of 1987 and 1995 levels.

Finally, the obesity data are mostly from the early to middle 1990s. For Australia, Finland, and Sweden they are from 1990. For Austria, the U.K., and the U.S., they are from 1991. For France and Switzerland they are from 1992. For New Zealand and Spain

they are from 1993. For Canada, Denmark, and Italy they are from 1994. For Norway and Portugal they are from 1995. For Belgium and the Netherlands they are from 1997. For Ireland they are from 1999.

Table A-1: DALEs and Life Expectancies for the countries in our sample.

Country	Females					Males				
	<u>DALE</u>		<u>Life Expectancy</u>			<u>DALE</u>		<u>Life Expectancy</u>		
	at birth	at sixty	at birth	at forty	at sixty	at birth	at sixty	at birth	at forty	at sixty
Australia	75.5	20.2	80.8	42.1	23.7	70.8	16.8	75.0	37.2	19.5
Austria	74.4	18.7	80.1	41.2	22.9	68.8	15.2	73.5	35.7	18.7
Belgium	74.6	19.6	80.2	42.0	23.8	68.7	15.8	73.6	36.1	18.9
Canada	74.0	18.9	81.3	42.5	24.3	70.0	16.0	75.3	37.5	19.9
Denmark	71.5	17.2	77.8	39.0	21.4	67.2	14.2	72.6	34.6	17.6
Finland	73.7	18.5	80.2	41.3	22.9	67.2	14.5	72.8	34.8	18.1
France	76.9	21.7	81.9	43.2	24.9	69.3	16.8	73.9	36.3	19.7
Ireland	71.7	16.6	78.6	39.8	21.5	67.5	13.9	73.0	35.0	17.4
Italy	75.4	19.9	81.0	42.3	23.7	70.0	16.2	74.6	36.8	19.2
Netherlands	74.4	19.7	80.4	41.0	22.8	69.6	15.4	74.6	35.7	18.1
New Zealand	71.2	17.0	79.5	41.1	23.0	67.1	14.4	74.2	36.7	19.1
Norway	74.6	19.7	80.8	41.8	23.3	68.8	15.1	74.8	36.6	18.9
Portugal	72.7	17.7	78.2	40.3	22.0	65.9	14.0	71.0	34.8	18.0
Spain	75.7	20.1	81.6	43.0	24.3	69.8	16.8	74.4	36.9	19.8
Sweden	74.9	19.6	81.3	42.4	23.9	71.2	16.8	75.9	37.6	19.8
Switzerland	75.5	20.6	81.7	43.0	24.5	69.5	16.0	75.3	37.6	20.0
UK	73.7	18.6	79.4	40.5	22.4	69.7	15.7	74.1	36.0	18.4
USA	72.6	18.4	79.2	40.7	22.9	67.5	15.0	72.5	35.6	19.1

Sources: DALEs from WHO (2000), life expectancies from OECD (2000).

Table A-2: Circulatory Disease Mortality Measures for the Countries in Our Sample.

Country	Females			Males		
	PYLL	Mortality Age 65-74	Mortality Age 75 +	PYLL	Mortality Age 65-74	Mortality Age 75 +
Australia	383.7	562.0	3,981.8	998.9	1,138.5	4,417.9
Austria	527.1	789.1	5,971.6	1,393.4	1,529.6	6,498.1
Belgium	431.9	606.4	4,067.2	1,028.6	1,156.1	4,568.4
Canada	409.5	515.9	3,272.5	1,044.3	1,096.2	3,950.3
Denmark	470.4	757.2	4,457.5	1,166.2	1,544.9	5,371.5
Finland	467.5	705.1	4,411.8	1,635.2	1,700.1	5,192.8
France	281.7	331.3	3,139.4	828.8	801.3	3,558.8
Ireland	578.8	939.1	4,832.2	1,567.8	1,978.2	5,837.9
Italy	421.8	542.6	4,445.0	1,036.9	1,091.0	4,860.5
Netherlands	448.6	602.4	3,467.4	1,116.1	1,309.7	4,396.0
New Zealand	583.2	711.6	3,985.4	1,409.7	1,377.2	4,819.6
Norway	399.5	663.7	3,952.3	1,136.7	1,483.8	5,174.4
Portugal	525.6	750.0	5,348.1	1,201.9	1,324.5	5,745.4
Spain	367.3	453.3	3,849.5	1,068.4	914.7	3,873.9
Sweden	353.8	578.1	4,250.1	1,051.2	1,326.6	5,333.4
Switzerland	273.0	427.3	3,994.8	760.0	1,059.3	4,612.1
UK	576.8	826.7	4,001.3	1,429.2	1,594.5	4,797.8
USA	741.5	727.7	4,024.4	1,639.7	1,347.0	4,458.0

Sources: PYLL from OECD (2000) and mortality rates from WHO website (www-nt.who.int/whosis/statistics).

Table A-3: Cancer Mortality Measures for the Countries in Our Sample.

Country	Females			Males		
	PYLL	Mortality Age 65-74	Mortality Age 75 +	PYLL	Mortality Age 65-74	Mortality Age 75 +
Australia	1,057.2	597.6	1,130.7	1,260.1	1,070.3	2,165.4
Austria	1,093.1	590.2	1,272.0	1,326.5	1,058.2	2,218.8
Belgium	1,117.7	585.3	1,325.4	1,492.1	1,306.0	2,857.4
Canada	1,138.5	672.3	1,226.4	1,196.7	1,090.7	2,175.4
Denmark	1,484.1	872.9	1,465.3	1,383.0	1,291.3	2,531.1
Finland	836.9	515.3	1,045.3	1,011.9	983.6	2,239.7
France	937.9	477.2	1,124.8	1,764.2	1,196.5	2,317.8
Ireland	1,269.3	771.2	1,357.3	1,281.0	1,208.0	2,528.8
Italy	1,039.3	530.3	1,161.5	1,494.7	1,224.0	2,203.7
Netherlands	1,192.1	628.2	1,285.2	1,298.8	1,259.8	2,767.6
New Zealand	1,352.3	719.2	1,222.8	1,274.2	1,145.3	2,306.4
Norway	1,139.6	595.5	1,163.0	1,113.4	1,008.2	2,281.4
Portugal	1,054.4	458.4	990.4	1,448.9	969.1	2,013.4
Spain	937.9	432.5	981.5	1,593.3	1,095.4	2,187.4
Sweden	974.2	599.0	1,069.2	946.7	854.9	1,896.1
Switzerland	825.0	526.0	1,110.5	1,076.0	1,018.2	2,149.8
UK	1,238.2	748.8	1,309.2	1,264.0	1,168.7	2,336.4
USA	1,152.7	690.7	1,163.3	1,327.5	1,089.9	2,031.5

Sources: PYLL from OECD (2000) and mortality rates from WHO website (www-nt.who.int/whosis/statistics).

Table A-4: Respiratory Disease Mortality Measures for the Countries in Our Sample.

Country	Females			Males		
	PYLL	Mortality Age 65-74	Mortality Age 75 +	PYLL	Mortality Age 65-74	Mortality Age 75 +
Australia	142.9	136.8	472.1	176.8	256.7	950.9
Austria	89.2	56.9	377.6	123.6	174.7	659.4
Belgium	112.4	104.9	666.2	234.0	404.8	1,637.8
Canada	94.2	127.1	715.9	161.6	237.0	1,257.8
Denmark	162.3	271.7	802.3	168.3	378.1	1,334.0
Finland	81.4	80.0	817.4	172.5	279.2	1,534.8
France	77.3	59.6	653.2	169.5	183.6	1,064.9
Ireland	153.2	284.3	1,754.5	255.1	532.2	2,583.4
Italy	82.4	51.9	440.5	136.3	192.7	1,028.3
Netherlands	85.9	114.9	817.3	113.8	300.4	1,667.1
New Zealand	164.1	175.0	1,042.0	215.6	303.3	1,558.3
Norway	91.9	148.3	1,059.5	116.8	252.2	1,506.4
Portugal	170.1	104.2	794.3	333.5	309.6	1,407.0
Spain	91.3	73.2	682.5	254.2	306.6	1,496.7
Sweden	77.1	94.2	703.8	112.4	150.8	1,096.5
Switzerland	62.0	52.7	444.9	127.0	176.8	936.0
UK	204.6	266.2	1,605.1	304.2	414.0	2,148.6
USA	210.7	197.0	798.0	296.4	314.4	1,217.5

Sources: PYLL from OECD (2000) and mortality rates from WHO website (www-nt.who.int/whosis/statistics).

Table A-5: Explanatory Variable Measures for the Countries in Our Sample

Country	GDPPC	PHPC	HEPC	Female SMOKE	Male SMOKE	ALCOHOL	Female OBESE	Male OBESE
Australia	\$16,743.99	\$196.39	\$1,592.34	27.0	30.2	10.5	9.1	8.2
Austria	\$16,783.00	\$197.43	\$1,772.83	20.3	35.5	12.6	9.0	8.3
Belgium	\$16,746.00	\$304.47	\$2,022.40	26.0	38.0	12.4	12.0	13.9
Canada	\$18,555.01	\$215.65	\$2,170.54	26.7	29.8	9.2	13.1	13.3
Denmark	\$17,096.01	\$112.85	\$1,839.86	42.0	47.0	11.7	7.0	8.2
Finland	\$16,441.99	\$190.66	\$1,640.89	20.0	32.4	9.5	8.8	7.9
France	\$17,655.00	\$664.57	\$2,155.95	19.2	37.8	16.6	6.8	6.1
Ireland	\$11,388.00	\$105.17	\$1,029.43	29.0	31.0	10.5	9.0	12.0
Italy	\$16,257.00	\$447.88	\$1,692.37	17.8	37.8	10.9	6.7	7.2
Netherlands	\$15,921.00	\$130.19	\$2,103.28	31.5	42.8	9.9	8.8	6.3
New Zealand	\$13,344.00	\$179.31	\$1,271.20	27.3	27.8	13.2	12.6	9.5
Norway	\$17,514.00	\$170.16	\$2,011.25	33.0	36.0	5.0	5.0	6.0
Portugal	\$9,598.00	\$246.73	\$714.33	5.8	32.4	10.1	12.6	10.3
Spain	\$11,734.00	\$287.41	\$1,072.32	21.4	51.5	13.5	10.4	9.4
Sweden	\$17,654.01	\$225.86	\$2,075.16	26.2	25.7	6.4	5.6	5.4
Switzerland	\$21,488.01	\$190.48	\$2,135.27	29.0	39.0	12.9	4.7	6.0
UK	\$16,055.00	\$183.72	\$1,525.71	29.0	31.0	9.7	15.0	13.0
USA	\$22,266.01	\$240.00	\$2,515.00	22.8	28.4	9.5	25.1	19.9

Source: OECD (2000)