The Productivity of Pharmaceuticals in Improving Health: An Analysis of the OECD Health Data

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SUMMARY

Although a number of studies have been conducted on health production functions, little attention has been given to pharmaceuticals as a separate input into the production of health. Building upon existing published work, this paper uses an alternative specification and more recent data to estimate the effect of pharmaceutical expenditures on levels of health in the member countries of the Organization for Economic Cooperation and Development (OECD). In a sample of developed countries, we found that pharmaceutical consumption, as measured by per capita drug expenditures, has a positive effect on life expectancy at advanced ages. The marginal effect of pharmaceutical consumption is consistent with estimates that have been reported previously but appears to decline with increasing age. Over the past few years, the substantial and disproportionate growth of pharmaceutical expenditures for public and private payers in the U.S. has led to calls for regulatory intervention (e.g., price controls). However, our research suggests that increases in drug spending may yield further increases in life expectancy.

Keywords: Health production, life expectancy, pharmaceuticals, OECD, ecological studies

INTRODUCTION

Auster et al. [1] were the first economists to study the production function for health. Since then, many similar analyses have been conducted using data from the United States or from multiple countries [2-22]. Several of these [3,6-8,11,13,20,21] have used aggregate data from the member countries of the Organization for Economic Cooperation and Development (OECD). Given the large percentage of gross domestic product (GDP) expended on health among more developed nations (e.g., 12.9% in the US, 9.3% in Canada, and 9.3% in France in 1999), one may reasonably infer that these countries collectively place a high priority on the health status of their populations. Therefore, for informed policy purposes, it is extremely important that work on the population health production function be ongoing using the latest data available and the latest sound econometric methods.

It has long been known that there is a strong positive correlation between average income and life expectancy [23]. Many studies of the production function for health have found that wealth, typically measured using per capita GDP, has a negative influence on mortality rates and a positive influence on life expectancy at various ages [3,6,9,17,20,21]. Most studies [1-3,8,9,14,17,19,20,21] have found that the consumption of medical care has no statistically significant effect on measures of a population's health. The evidence pertaining to the effect of lifestyle factors, such as the consumption of tobacco products and alcohol, is more equivocal [3,14,20,21,24]. Few studies [9,13,15,16,20,21] have directly or indirectly dealt with the effect of pharmaceuticals on mortality or life expectancy. Though many of these analyses have been flawed, the consensus among them is that pharmaceutical consumption has a positive impact on health.

In the last few years, a number of important, albeit expensive, new pharmaceutical agents have been brought into the marketplace [25,26]. Based on existing research, one would expect these to have a positive impact on a nation's stock of health. In order to investigate this important issue, we used more recent data than have been used in previous health production function studies, better measures of the variables involved, and improved econometric modeling techniques. We also provide a new and original discussion of the major issues involved when using ecological data to study the production function for health. Accordingly, the first section of this paper discusses our methodology. In a following section, we discuss the results we obtained, including the effects of lifestyle factors, wealth, non-pharmaceutical health care consumption, and pharmaceutical consumption on the health stock. We conclude with sensitivity analyses of our results, a brief discussion of some of the limitations of our work, and our conclusions.

METHODS

Sample

Data were taken from the OECD Health Data 2000 database. OECD Health Data 2000 is an interactive database that contains aggregate data on the health care systems of 29 of the 30 countries that are currently members of the OECD. The database includes over 1,200 indicators spanning the period 1960 to 1999, with official data up to 1998 and selected estimates for 1999. In particular, the data set includes various measures of health status (morbidity and mortality), health care resources and utilization, health expenditures and financing, as well as information relating to population demographics, non-medical determinants of health (alcohol and tobacco consumption), and economic references (GDP and monetary conversion rates).

We chose to work with the OECD Health Data for two reasons. First, these data provided us with a readily identifiable population for the purpose of statistical inference. That is, the population of more developed countries. One may further delimit this population to those countries dedicated to democratic and market economic principles; however, given that most developed nations are committed to such principles, we view this qualifier as being unnecessary. Second, the data set includes a large number of social, health, and economic indicators, which obviated the need to abstract data from multiple sources.

One may question our decision to focus on more developed countries as opposed to less developed nations. We recognize the importance of ascertaining the determinants of population health in developing countries. However, the consumption of pharmaceuticals in developed countries is, in general, much greater than that in developing countries. Given the availability and utilization of pharmaceuticals in more developed countries, we believe it to be important to understand their effects on population health relative to other potentially important factors (e.g., diet, education, wealth). In addition, data regarding drug consumption in developing nations are limited, which precluded a detailed analysis of the effect of drug consumption on population health in these countries.

Variable definitions and descriptive statistics are presented in Table 1. The dependent variables included life expectancies for males and females at ages 40, 60, and 65. Life expectancy data were missing for Ireland in 1997, and we substituted 1995 data for this country in our model. We hypothesized that health care and lifestyle factors would have cumulative effects on health. That is, the consumption of some factor over time by an individual would either have a positive or negative effect on that individual's health. While it is conceivable that the consumption of certain factors (e.g., alcohol, tobacco) by a mother would influence the health

of her offspring, this represents a different model from the one we were interested in estimating. Thus, we chose not to include life expectancy at birth as a dependent variable in our model.

Under the presumption that health care and lifestyle factors would have cumulative effects, we chose to lag the explanatory variables by roughly 15 years. The literature suggested that a lag of 20 years or more would be appropriate for alcohol and tobacco consumption [27-30]. However, there was little empirical evidence regarding the appropriate lag length for indicators of health care consumption. Missing data precluded us from lagging expenditure variables by more than 12 years or lifestyle variables by more than 17 years. A full model of this type would typically require several lags for each independent variable. Because of data and sample size limitations, we included only one lag per variable.

We measured income using per capita GDP in 1985. Pharmaceutical and other health care consumption were measured using the 1985 per capita expenditures for each country. PHARM was computed as total per capita expenditures on pharmaceuticals and other medical non-durables minus per capita expenditures on medical non-durables (when data for the latter were available). Our measure of pharmaceutical consumption included expenditures for outpatient prescription and over-the-counter medications as well as pharmacists' remuneration. HEALTH was computed by subtracting PHARM from total per capita expenditures on health care. Total health care expenditures in 1985 were missing for Greece and were estimated by summing total current expenditures on health and total investments in medical facilities.

The OECD Health Data database includes specific purchasing power parity (PPP) conversion factors for pharmaceutical and health care expenditures in addition to conventional GDP-based conversion factors. Elsewhere, it has been shown that pharmaceutical expenditures converted to U.S. dollars using GDP PPP exchange rates underestimate actual pharmaceutical

expenditures outside the U.S. [20,21]. The OECD pharmaceutical PPP exchange rates yield results that are consistent with those obtained using more accurate conversion factors developed by Szuba [31] and others. Unfortunately, the pharmaceutical- and health-specific exchange rates are available only for a limited number of years (i.e., 1980, 1985, 1990, 1993, and 1996). Thus, their availability influenced the lag we used for measures of income and health care consumption. Income and expenditure variables were converted into U.S. dollars by dividing by the appropriate 1985 PPP conversion factor.

Alcohol consumption was measured in liters consumed per capita by persons age 15 or older in 1980. We substituted 1983 data for Greece since data on alcohol consumption in 1980 were missing for this country. Smoking behavior was controlled for using grams of tobacco consumed per capita by persons age 15 or older in 1980. Data on tobacco consumption in 1980 were missing for Germany, Ireland, and Italy. For these countries, 1979 data were used instead. Data on tobacco consumption were unavailable for Spain in any year. For this country, we substituted the mean value for tobacco consumption in 1980 for the other countries included in our sample.

We also sought to control for the effects of dietary factors. As a measure of positive dietary intake, we included fruit and vegetable consumption in kilograms per capita in 1980. Frech and Miller had reported animal fat to be an important predictor of life expectancy. The measure of fat consumption they used is no longer collected by the OECD and was not available in the Health Data 2000 database. Therefore, we elected to use butter consumption in kilograms per capita in 1980 as an alternate measure of animal fat intake. This included quantities of butter used in food preparations or mixed with other fats to obtain particular types of margarine or cooking fats. Certain studies [20,21,24] have suggested that the relationship between fat intake

and life expectancy is parabolic (i.e., low levels of fat consumption yield increased life expectancy, whereas higher levels of consumption yield reduced life expectancy). We could have specified a curvilinear relationship between life expectancy and butter consumption by including a second-order term for the latter in our model. However, we chose to exclude the second-order term since collinearity with the first-order term led to instability in the model's parameter estimates.

Because of missing data, we restricted our analysis to 19 of the 30 OECD countries. We excluded Switzerland from our sample due to the limited availability of pharmaceutical and health-specific PPP exchange rates as well as tobacco and alcohol consumption data. We also excluded Turkey from our sample since it is relatively underdeveloped when compared with the other member countries of the OECD.

Model Specification

We elected to use a log-linear functional form in modeling the data. There are several reasons for this. First, it allowed us to interpret our parameter estimates as elasticities. Second, it allowed for diminishing marginal returns to the independent variables. In a log-linear model, the elasticity is held constant, while the absolute value of the marginal effect for each explanatory variable is forced to fall at higher and higher values of the variable. The continuous independent variables were centered prior to estimation to reduce multicollinearity. A dummy variable for Spain was added to the model to control for the imputation of missing tobacco consumption data for this country.

Initially, ordinary least squares (OLS) was used to estimate separate models for the six groupings $j = 1,\ldots,6$ defined by age and sex. The age-sex groupings were ages 40, 60, and 65

for both males and females. Country $i = 1,\ldots,19$ was then the unit of observation in the following life expectancy regression:

$$\ln \mathrm{LE}_{ij} = \beta_{0j} + \beta_{1j} \ln \mathrm{GDP}_i + \beta_{2j} \ln \mathrm{PHARM}_i + \beta_{3j} \ln \mathrm{HEALTH}_i + \beta_{4j} \ln \mathrm{ALCOHOL}_i + \qquad (1)$$

$$\beta_{5j} \ln \mathrm{TOB}_i + \beta_{6j} \ln \mathrm{BUTTER}_i + \beta_{7j} \ln \mathrm{VEG}_i + \beta_{8j} \mathrm{SPAIN}_i + \varepsilon_{ij}$$

Tests were performed using the OLS residuals to ascertain whether life expectancy data for the six strata could be pooled. We performed a test to determine whether the intercept varied among the six age-sex categories followed by a test for homogeneity of regression or parallelism. These tests are often ascribed to Chow [32]; however, they were described earlier in a number of other sources [33-36]. To maintain an overall two-tailed alpha level of 0.05, the first test was performed with an alpha of 0.025, while the second was performed with an alpha of 0.05. As would be expected, there was a significant difference among the six age-sex categories in the intercept term $(F_{5,107} = 7{,}053.06, p < 0.0001)$. However, the other parameters did not appear to vary significantly among the strata $(F_{40,60} = 1.13, p = 0.33)$.

Conventional tests for poolability assume spherical disturbances [37, p. 53]. In the presence of non-spherical disturbances, these tests are not robust. For example, when estimating an error components model, they may exhibit a high frequency of Type I error when the variance components are large. According to Baltagi [37], conventional tests for poolability should only be used after the disturbances have been transformed so that they are spherical. Baltagi describes a method for transforming the disturbances that follows from the work of Roy [38] and Zellner [39]. Using the methods described by Baltagi [37, pp. 53-55], we performed the Roy-Zellner analogs of the intercept and parallelism tests. These allowed for a one-way error components model in which country was treated as a random effect. The data were transformed using

consistent estimates of the covariance matrices for the restricted and unrestricted models; thus, the test statistics followed an approximate *F* distribution. The results were similar to those described in the preceding paragraph. While there was a significant difference among the six age-sex categories in the intercept term ($\hat{F}_{5,107} = 1{,}709.16, p < 0.0001$), the parameter vectors (excluding the intercept) did not vary significantly among the strata ($\hat{F}_{40,60} = 1.29, p = 0.19$).

Given the preceding results, we decided to pool life expectancy data for the six age-sex categories, adding dummy variables for age and sex to the model to control for differences in the intercept term. We chose to estimate a mixed model that treated country as a random effect. Residual maximum likelihood (REML) was used to estimate the model, and the White estimator [40] was used to adjust for potential heteroscedasticity. A sequential modeling approach was used to identify significant interactions between the dummy variables for age and sex and the other independent variables. All terms that were significant at the $p = 0.10$ level were included in the final model. Equation (2) depicts the final model specification, where the $\beta's$ are fixed effects, the u_i are random country effects from a $N(0, \sigma_u^2)$ distribution, and the ε_{ij} are independently identically distributed errors (at the level of age-sex category within country) from a $N(0, \sigma_\varepsilon^2)$ distribution and are independent of the u_i.

$$\ln \mathrm{LE}_{ij} = \beta_0 + \beta_1 \ln \mathrm{GDP}_i + \beta_2 \ln \mathrm{PHARM}_i + \beta_3 \ln \mathrm{HEALTH}_i + \beta_4 \ln \mathrm{ALCOHOL}_i + \tag{2}$$

$$\beta_5 \ln \mathrm{TOB}_i + \beta_6 \ln \mathrm{BUTTER}_i + \beta_7 \ln \mathrm{VEG}_i + \beta_8 \mathrm{SPAIN}_i + \beta_9 \mathrm{MALE}_{ij} +$$

$$\beta_{10} \mathrm{AGE60}_{ij} + \beta_{11} \mathrm{AGE65}_{ij} + \beta_{12} (\mathrm{MALE}_{ij})(\mathrm{AGE60}_{ij}) + \beta_{13} (\mathrm{MALE}_{ij})(\mathrm{AGE65}_{ij}) +$$

$$\beta_{14} (\mathrm{AGE60}_{ij})(\ln \mathrm{GDP}_i) + \beta_{15} (\mathrm{AGE65}_{ij})(\ln \mathrm{GDP}_i) + \beta_{16} (\mathrm{AGE60}_{ij})(\ln \mathrm{PHARM}_i) +$$

$$\beta_{17} (\mathrm{AGE65}_{ij})(\ln \mathrm{PHARM}_i) + \beta_{18} (\mathrm{MALE}_{ij})(\ln \mathrm{ALCOHOL}_i) +$$

$$\beta_{19} (\mathrm{MALE}_{ij})(\ln \mathrm{VEG}_i) + \beta_{20} (\mathrm{AGE60}_{ij})(\ln \mathrm{VEG}_i) + \beta_{21} (\mathrm{AGE65}_{ij})(\ln \mathrm{VEG}_i) +$$

$$\beta_{22} (\mathrm{AGE60}_{ij})(\mathrm{SPAIN}_i) + \beta_{23} (\mathrm{AGE65}_{ij})(\mathrm{SPAIN}_i) + u_i + \varepsilon_{ij}$$

A number of tests were performed to assess the model's goodness of fit. The Shapiro-Wilk test [41] was used to evaluate the normality of the error distribution. Multicollinearity was assessed using Belsley's condition index [42]. Ramsey's regression specification error test (RESET test) [43] was used to test for omitted variables and/or incorrect functional form. We added second- through fourth-order polynomials of the fitted values to the model and tested their joint significance. The Breusch-Pagan Lagrange multiplier test [44] and Hausman test [45] were performed to evaluate the efficiency and consistency, respectively, of the random effects model. Finally, a robust test for first-order serial correlation [46] was performed.

All statistical analyses were performed using SAS Release 8.02 (SAS Institute, Inc., Cary, NC) and Stata 8.0 (Stata Corporation, College Station, TX).

Analysis of Marginal Effects

We computed the marginal effect of pharmaceutical consumption on life expectancy at a given age in two ways. First, as shown in equation (3), we calculated the average number of

days of life expectancy gained in 1997 per additional per capita dollar spent on pharmaceuticals in 1985.

$$\mathrm{ME}_{ij}^{\mathrm{PHARM}} = (\hat{\beta}_2 + \hat{\beta}_{16}\mathrm{AGE60}_{ij} + \hat{\beta}_{17}\mathrm{AGE65}_{ij})\left(\frac{365 \times \mathrm{LE}_{ij}}{\mathrm{PHARM}_i}\right) \quad (3)$$

Second, as shown in equation (4), we calculated the average number of years of life expectancy gained in 1997 per additional 1% GDP share spent on pharmaceuticals in 1985.

$$\mathrm{ME}_{ij}^{\mathrm{PHARM}} = (\hat{\beta}_2 + \hat{\beta}_{16}\mathrm{AGE60}_{ij} + \hat{\beta}_{17}\mathrm{AGE65}_{ij})\left(\frac{\mathrm{LE}_{ij} \times \mathrm{GDP}_i}{100 \times \mathrm{PHARM}_i}\right) \quad (4)$$

In each case, $\hat{\beta}_2$, $\hat{\beta}_{16}$, and $\hat{\beta}_{17}$ represent estimates of the parameters specified in equation (2). Given that we specified a log-linear functional form and included a main effect for GDP in equation (2), our use of the same estimates in equations (3) and (4) is valid.

We were interested in testing the significance of the difference between the largest and smallest marginal effects within each age-sex category. This necessitated generating an estimate of the variability associated with the difference. The distribution of differences between the extreme values of an order statistic is unknown. However, the bootstrap can be used to generate a confidence interval to summarize the uncertainty in a parameter estimate. Carpenter et al. [47] discuss parametric and nonparametric residual bootstrap methods that can be used to derive confidence intervals for the parameter estimates of a mixed model. These methods are implemented in the current version of MLwiN (Centre for Multilevel Modelling, Institute of Education, London, United Kingdom). We applied Carpenter et al.'s methods to derive 95% confidence intervals for the difference between the smallest and largest marginal effects in four of the six age-sex categories (i.e., males and females at ages 60 and 65).

The parametric bootstrap proceeded as follows. The parameters β, σ_ε^2, and σ_u^2 were estimated by REML using the model specified in equation (2). We simulated $\varepsilon_{ij}^* \sim N(0, \hat{\sigma}_\varepsilon^2)$ for $i = 1,\ldots,19$ and $j = 1,\ldots,6$. Further, we simulated $u_i^* \sim N(0, \hat{\sigma}_u^2)$ for $i = 1,\ldots,19$. Next, we calculated the bootstrap data from

$$\ln \mathrm{LE}_{ij}^* = \hat{\beta}_0 + \hat{\beta}_1 \ln \mathrm{GDP}_i + \hat{\beta}_2 \ln \mathrm{PHARM}_i + \ldots + u_i^* + \varepsilon_{ij}^* \tag{5}$$

We then refitted the model to the bootstrap data and calculated the marginal effect of pharmaceutical consumption on life expectancy for each country within a given age-sex category. We then computed the difference between the smallest and largest marginal effects within each age-sex grouping. The entire process was repeated many times to generate an empirical distribution of 10,000 estimates for each of the four marginal effect differences. The 95% confidence interval for each difference was derived using the bias-corrected and accelerated (BCa) method [48,49]. Jackknifing was employed to derive the acceleration term, which involved estimating the parameters specified in model (2) while excluding one country at a time.

While the parametric bootstrap can be useful, it does not free inference from the assumption that the residuals have a normal distribution. Parametric bootstrap confidence intervals will not adequately reflect any non-normality in the data. Ergo, we thought it worthwhile to apply the nonparametric bootstrap in addition to the parametric method. Using REML, we obtained parameter estimates for model (2) from the data and calculated the residuals, $\{\hat{\varepsilon}_{ij}\}_{i=1,\ldots,19;\, j=1,\ldots,6}$ and $\{\hat{u}_i\}_{i=1,\ldots,19}$. The latter were the empirical best linear unbiased predictors. The two sets of residuals were centered and then rescaled using methods described by Carpenter et al. [47] to have variances equal to those estimated by model (2). We then

sampled independently and with replacement from the two sets, obtaining two new sets $\{\varepsilon_{ij}^*\}_{i=1,\ldots,19;\, j=1,\ldots,6}$ and $\{u_i^*\}_{i=1,\ldots,19}$. Using these, we calculated the bootstrap data from equation (5), refitted the model to the bootstrap data, and calculated the difference between the smallest and largest marginal effects within each age-sex category. The process of sampling from the residuals and estimating differences between marginal effects was repeated 10,000 times. Ninety-five percent BCa confidence intervals were then computed for the differences.

Sensitivity Analyses

Though not entirely arbitrary, we recognize that some researchers may not agree with the lag structure used in this research. We also recognize that some may criticize our decision to exclude Switzerland or the monetary conversion rates we used. Because of these concerns, we elected to perform sensitivity analyses around several of the assumptions made in our model.

First, we evaluated the impact of excluding Spain on the base model estimates. When excluding Spain, we also excluded the dummy variable for Spain and its associated interactions. Second, we evaluated the impact of using GDP PPP or market exchange rates instead of the OECD PPP exchange rates on the base model estimates. While doing so, we also evaluated the impact of including Switzerland on our results. Recall that the primary reason for Switzerland's exclusion was the lack of OECD PPP exchange rates in 1985. Third, we evaluated the effects of different lag structures on our results. The measure of tobacco consumption we used in our base model was not available for all countries (e.g., Germany, Italy, the U.S.) after 1980. Thus, when performing sensitivity analyses around the lag structure of our model, tobacco consumption was measured in expenditures (U.S. dollars) per capita. Three scenarios were considered: (1) 1985 economic data (GDP, PHARM, HEALTH) and 1980 lifestyle data (ETOH, TOB, BUTTER,

VEG) to provide a comparison with our base model, (2) 1985 economic and lifestyle data, and (3) 1990 economic and lifestyle data. In each of the three scenarios, Spain was included in the sample, Switzerland was excluded, and economic data were converted into U.S. dollars using OECD PPP exchange rates.

RESULTS AND DISCUSSION

Base Model

In general, the model appeared to provide a good fit for the observed data. The Breusch-Pagan test rejected OLS in favor of random effects ($\chi_1^2 = 90.05, p < 0.0001$), while the Hausman test failed to reject the null hypothesis that the individual effects were uncorrelated with the other regressors. Thus, the random effects model appeared to be both efficient and consistent. The combined residuals were normally distributed ($z = 0.38, p = 0.35$), and serial correlation did not appear to be a problem ($\chi_1^2 = 0.53, p = 0.47$). Multicollinearity appeared to be much less of an issue in our model than in some previous research. The condition index for our model was 9.44, which did not exceed the commonly accepted threshold of 20-30 [42,50]. Finally, the RESET test failed to reject the null hypothesis ($\chi_3^2 = 2.70, p = 0.44$), suggesting that the model's functional form was correctly specified.

Table 2 presents the results for our base model. The first five independent variables following the CONSTANT are dummy variables. As such, their coefficients cannot be directly interpreted as elasticities; they must be converted into "pseudo" elasticities using the formula $E = e^{\beta} - 1$ [51]. Thus, for example, the elasticity coefficient for MALE would be $E = 2.7183^{-0.1319} - 1 = -0.1236$. This elasticity would be interpreted as follows: being male (i.e., a change in the dummy variable MALE from zero to one) is associated with a 12.4% reduction in

life expectancy relative to being female. As would be expected, both male sex and advanced age were associated with reduced life expectancy. The elasticity for the interaction of MALE and AGE60 was –0.5412 (e.g., $E = 2.7183^{-0.1319-0.5805-0.0667} - 1 = -0.5412$). Similarly, the elasticity for the interaction of MALE and AGE65 was –0.6303, which implies that the life expectancy of a male at age 65 is 63% lower than the life expectancy of a female at age 40.

In the following four subsections of the paper, we discuss the base model's results with respect to the other independent variables presented in Table 2.

Lifestyle Effects

We found evidence that lifestyle factors, such as the consumption of alcohol, tobacco, butter, and fruits and vegetables, have important effects on life expectancy after controlling for the effects of wealth and health care consumption. Our findings are generally consistent with those of many clinical and epidemiological studies [52-62] that have reported such lifestyle factors to be important predictors of mortality.

Though alcohol consumption did not have a statistically significant effect on female life expectancy, its effect on the life expectancy of males was both significant and negative. Our results indicate that a doubling of alcohol consumption per capita would be associated with an approximate 3.3% decrease in male life expectancy. This finding most likely reflects a difference in alcohol intake between males and females and is consistent with the findings of Frech and Miller [20,21] and Cochrane et al. [3]. Although moderate drinking (i.e., no more than one drink a day for most women and no more than two drinks a day for most men) has been associated with psychological [63] and cardiovascular [64-67] benefits, it also increases risks for hemmorhagic stroke [68], motor vehicle accidents [69], adverse medication reactions [70,71],

and certain types of cancer [72,73]. Further, various researchers have suggested that moderate drinking is not cardioprotective, arguing that higher mortality among abstainers results from including among them people who have stopped drinking due to ill health. At the ecological level, it is likely that the small health benefits provided by moderate drinking are outweighed by the risks associated with alcohol consumption.

As would be expected, tobacco consumption had a statistically significant negative effect on life expectancy. Our results indicate that a doubling in tobacco consumption per capita would be associated with an approximate 10.2% reduction in population life expectancy. This is consistent with the findings of Cochrane et al. [3] and Wolfe and Gabay [8]. Cochrane et al. also controlled for the effects of per capita GDP, alcohol consumption, and poor diet in their models; however, they did not account for the effect of pharmaceutical consumption. Our results differ from those of Frech and Miller [20,21] who reported that smoking had no significant effect on life expectancy. Frech and Miller's estimate of the effect of smoking on life expectancy was imprecise due to the measure of smoking that was used. In addition, our proxy for smoking has the advantage of being an intensity measure. Similar to micro-level measures such as pack-years or the number of cigarettes consumed daily, the number of grams of tobacco consumed annually per capita better captures the dose-response effect of smoking on health than simple exposure measures like the percentage of the population that smokes.

Our proxy for animal fat intake behaved much like the other lifestyle factors included in the model. The parameter estimate for butter consumption per capita was statistically significant and carried a positive sign. There are several possible explanations for the apparent effect of butter consumption on life expectancy. First, it is possible that the positive effect was the result of vitamin fortification. In many developed countries, milk products, such as butter and

margarine, are fortified with vitamins A and D. It is conceivable that this would have a positive effect on a population's health. Second, one might hypothesize that the positive effect of butter consumption was due to the use of butter as a spread for vegetables. Although we investigated this hypothesis by testing for an interaction between butter consumption and vegetable consumption, we found no evidence to support it. Third, the positive effect of butter consumption could have been due to omitted variable bias. Since we explicitly controlled for the effect of wealth, it seems unlikely that our measure of fat intake simply captured an omitted income effect (i.e., that people in wealthier countries consumed fattier diets).

Our findings with respect to butter consumption are consistent with those of a number of other studies. Wolfe and Gabay [8] studied the relationship between negative changes in lifestyle and health status in a sample of OECD countries. Although negative changes in lifestyle were associated with declines in health status, butter consumption was negatively related to the former, suggesting a positive association with health status. Gage and O'Connor [24] reported that increases in the dietary contribution of fats relative to proteins were associated with increased life expectancy. However, the effect was moderated by diet quality such that in the presence of a high-quality diet, the effect of a high fat-to-protein ratio on life expectancy was reversed. Frech and Miller [20,21] also reported a curvilinear relationship between life expectancy and dietary fat intake such that low levels of consumption had a strong positive effect on life expectancy, while higher levels of consumption were associated with reduced life expectancy. We chose to exclude a second-order term for butter consumption from our model due to extreme multicollinearity. When adding the square of the logarithm of BUTTER to our model, the estimate for the first-order term was still positive and significant; however, the estimate for the second-order term bore a positive sign and was not statistically significant.

Other researchers have failed to detect an independent association between dietary fat intake and life expectancy or mortality. Hertz et al. [14] used exploratory stepwise regression methods to estimate the effects of dietary and other factors on life expectancy at birth in 44 countries. Total fat calorie consumption and the proportion of fat calorie consumption not explained by total calorie consumption were included as regressors in the final model. However, parameter estimates for these variables were not statistically significant. Similarly, Cochrane et al. [3] found that total calorie, protein, and fat consumption contributed little in explaining mortality when controlling for per capita income. More recent studies have suggested that the effect of dietary fat intake on mortality is confounded by age and pre-existing risk for heart disease. A consensus among researchers has not yet been reached as to whether the consumption of saturated fats above recommended levels by individuals who are not already at high risk for heart disease increases the likelihood of untimely death [74-77].

Finally, as expected, per capita fruit and vegetable consumption had a statistically significant positive effect on life expectancy. The effect of fruit and vegetable consumption was larger for males than for females and was larger for life expectancy at age 65 than at age 40. As with alcohol consumption, these findings likely reflect differences in intake among groups. In many developed countries, fruit and vegetable consumption appears to increase with increasing age among adults [78-82]. Further, although women tend to report eating fruits and vegetables with greater frequency than men, actual intake tends to be higher for the latter when more objective measures (e.g., average number of grams consumed daily) are used [80]. Since we measured intake using the number of kilograms consumed annually, it is not surprising that the effect of fruit and vegetable consumption on life expectancy was greater for males than females.

Wealth Effects

Per capita GDP was found to be an important predictor of life expectancy at ages 60 and 65. The marginal effect appeared to increase with increasing age, with elasticities of 0.03 for life expectancy at age 60 and 0.05 for life expectancy at age 65 (Table 2). Based on these estimates, doubling per capita GDP would have the effect of increasing life expectancy at age 65 by roughly 5.5%. In terms of remaining years of life, a 65-year-old male could expect to see his remaining life expectancy increase from an average of 15.4 years to 16.2 years. Similarly, a 65-year-old female could expect her life expectancy to increase from an average of 19 years to 20 years. These effects are consistent with the observation that economic development over the past several decades has led to increasing life expectancy. Our results correspond to those of Frech and Miller [20,21], though our estimate for the effect of per capita GDP on life expectancy at age 60 is somewhat smaller than theirs.

Several studies [5,12,14] have found wealth to be unassociated with mortality or life expectancy after controlling for other explanatory variables. Hadley [5] investigated the relationship between Medicare expenditures and mortality rates using 1980 U.S. Census data. Average family income was not associated with reduced mortality. However, the investigator failed to use a functional form in his models that would account for a log-linear relationship between the two variables. In addition, the results of this study are not directly comparable with our own since Hadley limited his analysis to elderly Medicare beneficiaries. Using a sample of 22 developing countries, Anand and Ravallion [12] reported that when controlling for a measure of poverty, the relationship between per capita GDP and life expectancy was not statistically significant. There is a growing body of evidence [9,12,83-91] that the unequal distribution of income within a country is more important than mean or median income when predicting health,

particularly in developed countries. We tested the validity of the relative income hypothesis in a subset of the 19 OECD countries included in our sample. Using 1994 estimates for economic references and 1997 estimates for indices of health status, we observed that per capita GDP was more strongly associated with life expectancy at all ages than the percentage of the population earning less than 50% of median income. This finding is consistent with recent evidence suggesting that data aggregation may reduce the correlation between health status and relative income [92]. While it may have been appropriate for us to include a measure of poverty or income inequality (such as the Gini coefficient) in our models, the limited availability of these data prevented us from doing so.

At least two studies [1,11] have reported the independent effect of income on health to be negative. Zweifel and Ferrari [11] derived a parameter estimate of -0.88 for 1970 per capita GDP when estimating life expectancy at ages 40 and 65 in 1980. The results of this study are not directly comparable with our own since Zweifel and Ferrari specified a functional form for their model that did not account for nonlinear relationships among the variables. Auster et al. [1] reported a positive association between income and mortality while controlling for the effects of medical care and education. Their findings with respect to income differ from those of Cochrane et al. [3]. The latter also controlled for education and the consumption of medical care but reported that per capita gross national product was negatively associated with mortality.

Anand and Ravallion [12] have argued that the positive effect of wealth on life expectancy reported by many researchers may be due to omitted variable bias. Income does not contribute directly to health but acts through other factors such as education, housing, and food intake [22]. Data for school expectancy are available for only a few countries in the OECD Health Data database. In addition, the data set includes no measures of the availability of

adequate housing. We believe that GDP provided a good proxy for the effects of these agents in our investigation. Still, as the OECD Health Data are updated and more data become available, it may be possible to model the specific effects of these factors.

Non-Pharmaceutical Health Care Consumption Effects

As shown in Table 2, the consumption of health care other than pharmaceuticals had no discernable effect on life expectancy. The parameter estimate for non-drug health care was positive though not statistically significant. Other studies [1-3,8,9,14,17,19,20,21] have also reported health care consumption to be unrelated to life expectancy or mortality rates, although most of these did not make the distinction between drug and non-drug health care. Frech and Miller [20,21] reported that the consumption of medical care other than pharmaceuticals had no significant effect on life expectancy at ages 40 and 60, though their parameter estimates were negative in sign. The results of a study conducted by Wnuk-Lipinski and Illsley [10], though mixed, suggest the absence of a strong relationship between health system indicators (e.g., number of physicians, nurses, hospital beds) and mortality rates.

A relatively small number of studies [4-6,11,12,18,22] have reported the effect of medical care on health to be positive. None of these distinguished between drug and non-drug health care. The results of some studies suggest that certain subpopulations may benefit from medical care services. Based on the work of Hadley [4,5], the elderly would appear to be included among these. Also, Bidani and Ravallion [18] found that differences in public health spending tend to matter more to the poor than to others. While Zweifel and Ferrari [11] reported a positive association between life expectancy and lagged health care expenditures, differences in specification between their model and ours limit the comparability of parameter estimates. In

a recent study, Evans et al. [22] used a fixed-effects translog model to estimate the effects of per capita health expenditures and years of schooling on average life expectancy in 191 industrialized and developing countries. In addition to failing to account for the independent effect of pharmaceuticals, the researchers excluded other potentially relevant variables (e.g., tobacco and alcohol consumption) from their model.

Our findings suggest that developed countries lie on the upper portion of the health care consumption curve where marginal rates of return are negligible. However, the results may also be explained by what Zweifel and Ferrari [11] have called the Sisyphus syndrome. The allocation of medical resources to health should promote increased life expectancy, but as longevity increases so do outlays on medical care. At advanced ages, there is a strong demand for private, and especially public, health care services. Given limited data for public expenditures, we did not make the distinction between private and public health care consumption in our study.

Pharmaceutical Consumption Effects

Pharmaceutical consumption had a positive effect on life expectancy (Table 2). As with per capita GDP, the elasticities for pharmaceutical consumption increased with increasing age. Doubling either the proportion of GDP allocated to pharmaceuticals or expenditures on pharmaceuticals per capita would increase life expectancy at ages 60 and 65 by 2.8% and 3.1%, respectively. Increasing pharmaceutical consumption twofold, a typical 65-year-old male could expect to see his remaining life expectancy increase from 15.4 years to 15.9 years. Similarly, a 65-year-old female could expect her life expectancy to increase from 19 years to 19.6 years.

Our findings are consistent with those of Frech and Miller [20,21], Lichtenberg [15,16], and others who have found that pharmaceutical consumption is associated with increased life expectancy, reduced mortality rates, and improved outcomes. Our results differ from those of Babazono and Hillman [13] who reported that per capita pharmaceutical expenditures have no effect on male or female life expectancy. As noted by other researchers [20], this study has several flaws. In particular, pharmaceutical expenditures were converted to U.S. dollars using GDP PPP exchange rates. In addition, stepwise regression methods were used, which can lead to misleading statistical inferences as well as potentially biased estimates [50,51].

Table 3 presents the marginal effects of drug consumption on total population life expectancy at ages 60 and 65 for males and females. The first four columns in the table report the marginal effects of pharmaceutical consumption in additional days of life expected in 1997 per additional U.S. dollar spent on pharmaceuticals in 1985. Countries with large per capita expenditures for pharmaceuticals in 1985 would stand to gain the least from marginal increases in drug consumption, whereas countries with small per capita drug expenditures in 1985 would realize the greatest life expectancy benefits. For example, a $1 per capita increase in drug spending in France would have the smallest effect. Average life expectancy at age 60 would be increased by 0.55 days for males and 0.69 days for females. Likewise, a $1 per capita increase in drug spending in France would be associated with an increase in life expectancy at age 65 of 0.50 days for males and 0.64 days for females. On the other hand, an increase in drug spending would have the greatest effect in Ireland, where average life expectancy at age 60 would be increased by 2.62 days for males and 3.23 days for females. Similarly, a $1 per capita increase in drug spending in Ireland would be associated with an increased life expectancy of 2.29 days for males and 2.91 days for females. These findings are not surprising considering that countries

like Ireland and Canada have relatively low per capita pharmaceutical expenditures, while per capita drug spending is much higher in countries like France and Italy.

The estimated confidence intervals indicate that significant differences exist among the marginal effects in each age-sex category. Since the confidence intervals were derived from the empirical distributions of marginal effect differences, an alternative approach for evaluating significance would be to define a minimum meaningful difference between marginal effects and observe whether or not this is included in a given interval. For example, we could say with 95% confidence that the maximum difference between marginal effects in each age-sex category is greater than 0.25 days of life expectancy gained in 1997 per U.S. dollar spent on pharmaceuticals in 1985. For each age-sex category, the nonparametric and parametric bootstrap methods yielded similar confidence limits, providing further evidence that our data met the assumptions of the random effects model.

The second four columns in Table 3 depict the marginal effects of pharmaceutical consumption in additional years of life expected in 1997 per additional 1% of GDP share spent on pharmaceuticals in 1985. Countries that allocated a relatively small percentage of GDP to pharmaceuticals in 1985 (e.g., Austria, Canada, Denmark, the Netherlands) would stand to gain the most in life expectancy by marginally increasing their drug consumption. Conversely, countries that spent a relatively large proportion of their GDP on pharmaceuticals in 1985 (e.g., France, Greece, Portugal, Italy) would stand to gain the least. In Canada, where roughly 0.89% of GDP was spent on pharmaceuticals in 1985, a 1% increase in drug spending would yield an increase in life expectancy at age 65 of 0.61 years for males and 0.76 years for females. Conversely, a 1% increase in GDP share spent on pharmaceuticals in France, where 3.1% of GDP was allocated to drugs in 1985, would yield an increase in average life expectancy at age 65

of only about a fifth of a year for either sex. The bootstrap confidence intervals suggest that significant differences exist among the marginal effects in each age-sex category. As in the preceding case, the parametric and nonparametric methods yielded similar confidence limits.

Using the metric of days of life expectancy gained per capita dollar spent on pharmaceuticals, it appears that the U.S. would fare about as well as the "average" country in our sample. In the U.S., an additional per capita dollar spent on drugs in 1985 would yield an increase in life expectancy at age 65 in 1997 of 1.25 days for males and 1.51 days for females. The average increase in life expectancy at age 65 would be 1.25 days for males and 1.55 days for females. Conversely, it appears that the U.S. would fare better than many other countries by increasing the proportion of GDP spent on pharmaceuticals. In 1985, roughly 0.9% of GDP was allocated to pharmaceuticals in the U.S. A 1% increase in GDP spending on drugs in the U.S. would yield an increase in life expectancy at age 65 of 0.58 years for males and 0.70 years for females. The average increase in life expectancy at age 65 would be 0.39 years for males and 0.49 for females. Thus, the important metric for the U.S. appears to be the proportion of GDP allocated toward pharmaceuticals.

Sensitivity Analyses

Table 4 shows the sensitivity of parameter estimates for economic variables to the exclusion of Spain, the inclusion of Switzerland, the use of alternative exchange rates, and changes in the model lag structure. Table 5 shows the sensitivity of parameter estimates for lifestyle variables to these same factors. Excluding Spain from the sample had no appreciable effect on any of our findings. When using the GDP PPP exchange rates, the estimate for the main effect of GDP was larger than that in our base model, while the estimates for

pharmaceutical and non-drug health care consumption were somewhat attenuated. However, the significance of the parameter estimates was not greatly changed. When using market exchange rates, there were no significant GDP effects, and estimates for the interaction of GDP with age were negligible. In addition, the estimated effects for pharmaceutical and non-drug health care consumption were reduced in magnitude. When using either of these alternative exchange rates, the addition of Switzerland to the sample did not yield large changes in the parameter estimates or their statistical significance. Based on these findings, our interpretation is that the GDP PPP and market exchange rates are inferior to the OECD PPP exchange rates used in our base model.

Because there is no published research on lag structure in this area of health economics, and because it is problematic to deduce lag structure on an *a priori* basis, we also varied the lag structure in the base model to ascertain whether this would influence our results. As shown at the bottom of Table 4 and Table 5, we varied the economic data for the years 1985 and 1990 and the lifestyle data for the years 1980, 1985, and 1990. In the model in which economic data were measured in 1985, lifestyle data were measured in 1980, and tobacco consumption was measured in U.S. dollars per capita, our estimate for the effect of tobacco consumption was -0.07. This was somewhat smaller than the estimate for tobacco consumption in our base model. The estimate for the main effect of GDP was larger than that in our base model though still not statistically significant. Parameter estimates for interactions involving GDP and age were attenuated, while estimates for interactions involving pharmaceutical consumption and age were increased in magnitude. Further, the estimate for the main effect of vegetable consumption was reduced, while estimates for the interaction of vegetable consumption with age were somewhat larger than those in our base model. Aside from these differences, the results were fairly consistent with those presented in Table 2.

Increasing recency in the measurement of explanatory variables was associated with reductions in both the magnitude and statistical significance of parameter estimates for lifestyle factors. This finding indicates that our choice of a cumulative effects model was appropriate for tobacco, alcohol, vegetable, and butter consumption. Parameter estimates for interactions involving pharmaceutical consumption and age did not change appreciably as we moved closer to the year in which life expectancy was measured. However, the main effect for pharmaceutical consumption was both positive and significant when 1990 economic data were used. This suggests that a contemporaneous effects model might have provided an even better fit for the pharmaceutical expenditures data. One explanation for this finding is that a broader range of more effective drugs is available today than was available in previous decades. Interestingly, the parameter estimates for interactions involving GDP and age grew in magnitude as we moved closer to the year in which life expectancy was measured. It appears that variance in life expectancy that had formerly been explained by the lifestyle variables was absorbed by GDP.

Issues Related to Data Aggregation

It would have been highly desirable to use individual-level data in our analyses. Of course, individual-level data were not available for the full set of 19 countries included in our sample. There are several problems associated with the use of aggregate data, including small sample size, a limited range of variation in variables, and heightened sensitivity to outliers compared to individual-level data. Our use of aggregated data for each country theoretically led to a loss of efficiency. In addition, when using aggregate data, heteroscedasticity may result if each grouping does not contain the same number of observations. However, aggregation can cancel out errors in measurement or misspecifications of micro-relationships.

There are several specific issues related to our use of aggregate data that should be addressed. First, we did not explicitly account for population migration between countries. There are hardly any data on the mobility of populations between OECD nations, and no data of this type are available in the OECD Health Data 2000 database. Even if we did have an indicator of population migration, we suspect that its marginal effect would be small. During the years analyzed, it is reasonable to presume that the population growth within each country occurred mainly as a result of increases in the birth rate as opposed to migration between countries. Second, no adjustment was made for underlying differences between individuals in factors that might influence health status (e.g., chronic diseases). To the extent that these factors were distributed similarly in each country, we feel that an analysis using mean life expectancy data was valid. Third, our use of aggregate data masked differences between countries in the relative price and availability of pharmaceuticals. We attempted to adjust for cross-national price differences using the OECD's pharmaceutical PPP exchange rates. While this method was imperfect, it is widely recognized that there is no single, correct measure of international price differences for pharmaceuticals [93]. Sensitivity analyses were performed to evaluate the potential impact of using other exchange rates for pharmaceutical expenditures.

CONCLUSIONS

In a sample of more developed countries, we found that drug consumption, as measured by per capita pharmaceutical expenditures, has a positive effect on population life expectancy at various ages. The marginal effect of pharmaceutical consumption is somewhat smaller than has been previously reported and appears to decline with increasing age. The consumption of health care other than pharmaceuticals was found to have a negligible effect on life expectancy. We

observed that wealth, as measured by per capita GDP, has a large, positive effect on life expectancy. In addition, a variety of lifestyle factors were found to have important effects in producing health after controlling for the effects of wealth and pharmaceutical consumption. Our results are broadly consistent with those of Frech and Miller [20,21] who conducted a similar analysis in a sample of 21 OECD countries.

Upwardly spiraling pharmaceutical expenditures have given rise to concern among policy makers and consumers in the U.S. and other developed countries. However, our research suggests that increases in pharmaceutical spending may yield significant health benefits in many countries, particularly with respect to life expectancy. In order to have a positive impact on health, increased expenditures must be associated with an increased volume or outlays on novel therapeutic agents that demonstrate greater efficacy than existing medications. Increases in expenditures due to increases in the prices of existing medications or on new market entries that provide no clinical advantage over less-costly existing therapies will provide no additional health benefits.

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Table 1. Variable definitions and descriptive statistics.

CONTINUOUS VARIABLES

Variable	Definition	Mean	SD	Minimum	Maximum
LEM40	Number of years of life expectancy for males at age 40, 1997	36.55	1.00	34.70	38.10
LEM60	Number of years of life expectancy for males at age 60, 1997	19.17	0.82	17.40	20.10
LEM65	Number of years of life expectancy for males at age 65, 1997	15.46	0.76	13.70	16.30
LEF40	Number of years of life expectancy for females at age 40, 1997	41.71	1.11	39.50	43.50
LEF60	Number of years of life expectancy for females at age 60, 1997	23.38	0.98	21.50	25.20
LEF65	Number of years of life expectancy for females at age 65, 1997	19.19	0.91	17.40	20.80
GDP	Gross domestic product per capita, 1985 U.S. dollars	11,719.11	2,751.13	6,105.00	16,976.00
PHARM	Pharmaceutical expenditures per capita, 1985 U.S. dollars	171.26	72.20	73.21	400.34
HEALTH	Health expenditures (not including pharmaceuticals) per capita, 1985 U.S. dollars	1,100.26	448.04	309.71	1,960.05
ETOH	Liters of ethyl alcohol consumed annually per capita by persons age 15 or older, 1980	11.99	3.82	5.30	20.60
TOB	Grams of tobacco consumed annually per capita by persons age 15 or older, 1980	2,727.33	530.18	1,492.00	3,588.00
BUTTER	Kilograms of butter consumed annually per capita, 1980	6.01	4.05	0.50	13.90
VEG	Kilograms of fruits and vegetables consumed annually per capita, 1980	187.01	66.20	70.90	362.20

DISCRETE VARIABLES

Variable	Definition
MALE	Dummy variable taking on value of 1 if dependent variable was life expectancy for males and 0 otherwise
AGE60	Dummy variable taking on value of 1 if dependent variable was life expectancy at age 60 and 0 otherwise
AGE65	Dummy variable taking on value of 1 if dependent variable was life expectancy at age 65 and 0 otherwise
SPAIN	Dummy variable taking on value of 1 if country was Spain and 0 otherwise

Note: Descriptive statistics apply to the sample of 19 countries. Independent variables included in the sensitivity analysis of model lag structure were measured in 1980, 1985, or 1990.
SD = standard deviation.

Table 2. Regression parameter estimates for base model.

Independent Variable		Independent Variable		Independent Variable	
CONSTANT	3.7277[a] (0.0026)	AGE60 × ln(GDP)	0.0286[a] (0.0064)	MALE × ln(ETOH)	-0.0336[a] (0.0129)
MALE	-0.1319[a,c] (0.0037)	AGE65 × ln(GDP)	0.0546[a] (0.0087)	ln(TOB)	-0.1019[a] (0.0264)
AGE60	-0.5805[a,c] (0.0019)	ln(PHARM)	0.0027 (0.0138)	ln(BUTTER)	0.0189[a] (0.0041)
AGE65	-0.7786[a,c] (0.0025)	AGE60 × ln(PHARM)	0.0275[a] (0.0061)	ln(VEG)	0.0943[a] (0.0233)
MALE × AGE60	-0.0667[a,c] (0.0030)	AGE65 × ln(PHARM)	0.0308[a] (0.0079)	MALE × ln(VEG)	0.0301[a] (0.0123)
MALE × AGE65	-0.0847[a,c] (0.0046)	ln(HEALTH)	0.0230 (0.0173)	AGE60 × ln(VEG)	0.0106 (0.0080)
ln(GDP)	-0.0073 (0.0262)	ln(ETOH)	0.0041 (0.0112)	AGE65 × ln(VEG)	0.0183[b] (0.0095)

Sample size: 114 observations.
Note: Robust standard errors given in parentheses.
a Significantly different from 0, $p < 0.05$, two-tailed.
b Significantly different from 0, $p < 0.10$, two-tailed.
c To be interpreted as an elasticity, this must be converted using the formula: $E = e^{\beta} - 1$ [51].

Table 3. Marginal effect of pharmaceutical consumption on life expectancy at various ages.

	Days per U.S. Dollar				Years per 1% GDP Share			
	Males		Females		Males		Females	
Country	Age 60	Age 65	Age 60	Age 65	Age 60	Age 65	Age 60	Age 65
Australia	1.42	1.28	1.72	1.57	0.52	0.47	0.63	0.57
Austria	1.85	1.66	2.27	2.06	0.62	0.56	0.76	0.69
Belgium	0.89	0.80	1.12	1.02	0.30	0.27	0.38	0.34
Canada	1.71	1.55	2.07	1.91	0.68	0.61	0.82	0.76
Denmark	1.64	1.47	1.98	1.81	0.60	0.54	0.73	0.67
Finland	1.54	1.37	1.91	1.73	0.50	0.45	0.62	0.56
France	0.55	0.50	0.69	0.64	0.20	0.18	0.25	0.23
Germany	1.08	0.97	1.32	1.20	0.39	0.35	0.49	0.44
Greece	1.26	1.13	1.45	1.31	0.25	0.22	0.28	0.26
Ireland	2.62	2.29	3.23	2.91	0.54	0.47	0.66	0.60
Italy	0.80	0.71	0.99	0.91	0.26	0.23	0.32	0.29
Netherlands	1.92	1.70	2.40	2.18	0.62	0.55	0.77	0.70
New Zealand	1.42	1.28	1.70	1.57	0.44	0.39	0.52	0.48
Norway	1.41	1.25	1.72	1.57	0.53	0.47	0.65	0.59
Portugal	1.39	1.24	1.71	1.53	0.23	0.21	0.29	0.26
Spain	1.28	1.15	1.58	1.44	0.28	0.25	0.35	0.32
Sweden	1.31	1.17	1.57	1.44	0.47	0.42	0.56	0.51
U.K.	1.00	0.88	1.20	1.09	0.31	0.27	0.37	0.34
U.S.	1.38	1.25	1.64	1.51	0.64	0.58	0.76	0.70
Average (SD)	1.39 (0.46)	1.25 (0.40)	1.70 (0.57)	1.55 (0.51)	0.44 (0.16)	0.39 (0.14)	0.54 (0.19)	0.49 (0.18)
Difference[a]	2.07	1.79	2.54	2.27	0.48	0.44	0.57	0.53
95% CI (LL, UL)								
Nonparametric	0.30, 4.59	0.33, 3.70	0.36, 5.64	0.42, 4.70	0.07, 1.07	0.08, 0.90	0.08, 1.27	0.10, 1.09
Parametric	0.29, 4.51	0.28, 3.63	0.35, 5.55	0.36, 4.61	0.07, 1.05	0.07, 0.88	0.08, 1.25	0.08, 1.07

SD = standard deviation; CI = confidence interval; LL = lower limit; UL = upper limit.
a Defined as the difference between the maximum and minimum marginal effects within a given age-sex category.

Table 4. Economic variables: sensitivity of regression results to changes in the measurement of model parameters.

Observation Changes and Parameter Changes	ln(GDP)	AGE60 × ln(GDP)	AGE65 × ln(GDP)	ln(PHARM)	AGE60 × ln(PHARM)	AGE65 × ln(PHARM)	ln(HEALTH)	Sample Size
Base Model:								
Including Spain	-0.0073	0.0286[a]	0.0546[a]	0.0027	0.0275[a]	0.0308[a]	0.0230	114
	(0.0262)	(0.0064)	(0.0087)	(0.0138)	(0.0061)	(0.0079)	(0.0173)	
Excluding Spain	-0.0073	0.0286[a]	0.0546[a]	0.0027	0.0275[a]	0.0308[a]	0.0230	108
	(0.0262)	(0.0064)	(0.0087)	(0.0138)	(0.0061)	(0.0079)	(0.0173)	
GDP PPP Exchange Rates:								
Excluding Switzerland	0.0404	0.0261[a]	0.0513[a]	-0.0093	0.0238[b]	0.0276[b]	-0.0010	114
	(0.0469)	(0.0089)	(0.0100)	(0.0188)	(0.0127)	(0.0155)	(0.0248)	
Including Switzerland	0.0404	0.0293[a]	0.0530	-0.0109	0.0226[b]	0.0269[b]	-0.0008	120
	(0.0458)	(0.0085)	(0.0094)	(0.0185)	(0.0127)	(0.0155)	(0.0241)	
Market Exchange Rates:								
Excluding Switzerland	0.0344	-0.0015	0.0073	-0.0117	0.0308[a]	0.0376[b]	-0.0045	114
	(0.0594)	(0.0089)	(0.0113)	(0.0192)	(0.0152)	(0.0196)	(0.0380)	
Including Switzerland	0.0363	0.0010	0.0092	-0.0134	0.0295[a]	0.0366[b]	-0.0043	120
	(0.0583)	(0.0089)	(0.0111)	(0.0191)	(0.0151)	(0.0195	(0.0372)	
Lag Structure:								
1985 Economic Data / 1980 Lifestyle Data	0.0588	0.0228[a]	0.0459[a]	-0.0002	0.0282[a]	0.0319[a]	-0.0109	114
	(0.0452)	(0.0073)	(0.0105)	(0.0130)	(0.0063)	(0.0082)	(0.0210)	
1985 Economic Data / 1985 Lifestyle Data	0.0555	0.0244[a]	0.0486[a]	0.0070	0.0267[a]	0.0293[a]	-0.0165	114
	(0.0553)	(0.0078)	(0.0109)	(0.0103)	(0.0065)	(0.0083)	(0.0243)	
1990 Economic Data / 1990 Lifestyle Data	-0.0579	0.0298[a]	0.0581[a]	0.0326[a]	0.0273[a]	0.0307[a]	0.0577	114
	(0.0919)	(0.0111)	(0.0158)	(0.0135)	(0.0042)	(0.0055)	(0.0747)	

Note: Robust standard errors given in parentheses.
a Significantly different from 0, $p < 0.05$, two-tailed.
b Significantly different from 0, $p < 0.10$, two-tailed.

Table 5. Lifestyle variables: sensitivity of regression results to changes in the measurement of model parameters.

Observation Changes and Parameter Changes	ln(ETOH)	MALE × ln(ETOH)	ln(TOB)	ln(BUTTER)	ln(VEG)	MALE × ln(VEG)	AGE60 × ln(VEG)	AGE65 × ln(VEG)	Sample Size
Base Model:									
Including Spain	0.0041	-0.0336[a]	-0.1019[a]	0.0189[a]	0.0943[a]	0.0301[a]	0.0106	0.0183[b]	114
	(0.0112)	(0.0129)	(0.0264)	(0.0041)	(0.0233)	(0.0123)	(0.0080)	(0.0095)	
Excluding Spain	0.0030	-0.0312[a]	-0.1019[a]	0.0189[a]	0.0937[a]	0.0314[a]	0.0106	0.0183[b]	108
	(0.0112)	(0.0135)	(0.0265)	(0.0041)	(0.0233)	(0.0123)	(0.0080)	(0.0096)	
GDP PPP Exchange Rates:									
Excluding Switzerland	0.0067	-0.0336[a]	-0.1155[a]	0.0186[a]	0.0892[a]	0.0301[a]	0.0119	0.0195[b]	114
	(0.0191)	(0.0129)	(0.0273)	(0.0076)	(0.0266)	(0.0123)	(0.0095)	(0.0110)	
Including Switzerland	0.0082	-0.0336[a]	-0.1176[a]	0.0188[a]	0.0914[a]	0.0301[a]	0.0136	0.0204[b]	120
	(0.0189)	(0.0129)	(0.0266)	(0.0076)	(0.0261)	(0.0120)	(0.0094)	(0.0108)	
Market Exchange Rates:									
Excluding Switzerland	0.0204	-0.0336[a]	-0.1179[a]	0.0158	0.0858[a]	0.0301[a]	0.0145[b]	0.0235[a]	114
	(0.0262)	(0.0129)	(0.0303)	(0.0110)	(0.0338)	(0.0123)	(0.0080)	(0.0091)	
Including Switzerland	0.0222	-0.0336[a]	-0.1202[a]	0.0160	0.0880[a]	0.0301[a]	0.0167[a]	0.0252[a]	120
	(0.0258)	(0.0129)	(0.0298)	(0.0110)	(0.0331)	(0.0120)	(0.0080)	(0.0089)	
Lag Structure:									
1985 Economic Data / 1980 Lifestyle Data	-0.0039	-0.0336[a]	-0.0724[a,c]	0.0171[a]	0.0311[b]	0.0301[a]	0.0138	0.0230[a]	114
	(0.0169)	(0.0129)	(0.0144)	(0.0076)	(0.0180)	(0.0123)	(0.0085)	(0.0108)	
1985 Economic Data / 1985 Lifestyle Data	-0.0150	-0.0318[a]	-0.0554[a,c]	0.0104	0.0231	0.0323[a]	0.0149[b]	0.0250[a]	114
	(0.0213)	(0.0139)	(0.0139)	(0.0132)	(0.0309)	(0.0137)	(0.0077)	(0.0101)	
1990 Economic Data / 1990 Lifestyle Data	-0.0161	-0.0291[a]	-0.0038[c]	0.0007	0.0115	0.0222	0.0092	0.0191[a]	114
	(0.0283)	(0.0143)	(0.0269)	(0.0168)	(0.0260)	(0.0157)	(0.0069)	(0.0095)	

Note: Robust standard errors given in parentheses.
a Significantly different from 0, $p < 0.05$, two-tailed.
b Significantly different from 0, $p < 0.10$, two-tailed.
c Tobacco consumption measured in U.S. dollars per capita.