The Determinants of Life Expectancy: An Analysis of the OECD Health Data

James W. Shaw,* William C. Horrace,† and Ronald J. Vogel‡

This study considers an aggregate life expectancy production function for a sample of developed countries. We find that pharmaceutical consumption has a positive effect on life expectancy at middle and advanced ages but is sensitive to the age distribution of a given country. Thus, ignoring age distribution in a regression of life expectancy on pharmaceutical consumption creates an omitted-variable bias in the pharmacetical coefficient. We find that doubling annual pharmaceutical expenditures adds about one year of life expectancy for males at age 40 and slightly less than a year of life expectancy for females at age 65. We also present results for lifestyle inputs into the production of life expectancy. For example, decreasing tobacco consumption by about two cigarettes per day or increasing fruit and vegetable consumption by 30% (one-third pound per day) increases life expectancy approximately one year for 40-year-old females.

JEL Classification: I12

1. Introduction

This study is concerned with understanding the determinants of life expectancy in developed countries. The level (and variability) of life expectancy has important implications for individual and aggregate human behavior; it affects fertility behavior, economic growth, human capital investment, intergenerational transfers, and incentives for pension benefit claims (Zhang, Zhang, and Lee 2001; Coile et al. 2002). From the social planners perspective, it has implications for public finance. For example, Gradstein and Kaganovich (2004) conclude that increasing longevity results in increasing public funding of education and economic growth. Cremer, Lozachmeur, and Pestieau (2004, p. 2260) argue that early retirement “puts pressure on the financing of healthcare and pension schemes [and this pressure] is made worse by growing longevity.” While typically assumed strictly exogenous for the purpose of policy analysis, it has been argued that life expectancy (or more broadly “health”) is predetermined by behavioral and policy variables in what can be loosely described as a production function for health. Estimating this function is the goal of this study.

Auster, Leveson, and Sarachek (1969) were the first economists to study a population production function for health: a regression of state-level mortality rates on medical care and environmental variables. Today their research motivations and questions remain compelling. Indeed, given the size

* Tobacco Control Research Branch, Behavioral Research Program, National Cancer Institute, Bethesda, MD 20892, USA; E-mail shawjm@mail.nih.gov.
† Department of Economics, Syracuse University, 426 Eggers Hall, Syracuse, NY 13244, USA; E-mail whorrace@maxwell.syr.edu; corresponding author.
‡ Center for Health Outcomes and PharmacoEconomic Research, College of Pharmacy, University of Arizona, Tucson, AZ 85721, USA; E-mail vogel@pharmacy.arizona.edu.

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and rapid growth of health care–related industries and recent public interest in containing medical and insurance costs, it could be argued that understanding the socioeconomic determinants of societal health is more important today than ever. Moreover, issues concerning the government’s role in sponsoring basic medical and pharmaceutical research; in regulating drug, alcohol, and tobacco consumption; and in promoting healthy lifestyles are all particularly newsworthy. The research questions related to public health are obvious. If societal health can be measured as life expectancy or mortality rates, what are the various socioeconomic factors that increase or decrease it? Can the marginal effects of these factors be disentangled? If so, which of these factors produces the largest health benefits (or costs) to society? These questions are as important now as when first posed by Auster, Leveson, and Sarachek in 1969.

Since Auster, Leveson, and Sarachek, several economic studies have attempted to answer these questions using data from the United States or multiple countries. Many of these have used aggregate data from the member countries of the OECD to explain cross-country mortality rates or life expectancies. While the empirical results are mixed, the general consensus is that population life expectancy (or mortality) is a function of environmental measures (e.g., wealth, education, safety regulation, infrastructure), lifestyle measures (e.g., tobacco or alcohol consumption), and health care consumption measures (e.g., medical or pharmaceutical expenditures). However, the appropriate econometric methodology for disentangling these effects and its meaning for the relative importance (statistical or economic) of the estimated effects is more contentious.

These methodological issues are most vividly illustrated in the few studies that have focused on pharmaceutical expenditures as a separate input to life expectancy. These include Peltzman (1987), Babazono and Hillman (1994), Lichtenberg (1996, 1998), Frech and Miller (1999), and Miller and Frech (2000). For example, Peltzman (1987) examined the effects of wealth and prescription drug laws on infectious disease mortality and on poisoning mortality across middle-income countries in a generalized least squares (GLS) framework. He found that wealth variables significantly decreased both disease and poisoning mortality rates, while prescription drug laws had a significant and positive effect on poisoning mortality only. The implication of the latter result was that mandatory prescription drug enforcement may lead to more frequent accidental poisonings (or deaths due to the overconsumption of pharmaceuticals, as interpreted by Peltzman). Peltzman also considered a GLS regression of life expectancy at birth on wealth and government health expenditures and found only wealth to be a significant determinant. His life expectancy variable was an average for the entire population (including males and females) of each country and was only for a single age stratum (at birth) in each country. He also ignored lifestyle variables in his regressions.

To fully appreciate the vast differences in methodological approaches in pharmaceutical studies, compare Peltzman’s analysis to that of Frech and Miller (1999), a subset of whose findings were also published in Miller and Frech (2000). Frech and Miller partitioned OECD data into age strata (life expectancy at birth, age 40 years, and age 60 years) and estimated separate life expectancy regressions for each stratum (pooling data for males and females). The determinants in each stratum regression were wealth, some lifestyle variables (alcohol, tobacco, and animal fat consumption), and pharmaceutical and nonpharmaceutical medical expenditures. Using country-level OECD data, they

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1 All the studies discussed and compared herein analyzed health data at some aggregated macroeconomic level (such as state- or country-wide). While there are clinical and epidemiological studies that have examined health outcomes at the individual level, these rarely yield macroeconomic policy implications, which are a major focus of the current study.

found that pharmaceutical expenditures had a significant and positive effect on life expectancy and that this increased with age. They also found that tobacco consumption (as measured using the concatenated percentages of males and females who smoked in each country) was not a significant determinant of life expectancy at any age. Frech and Miller’s study is among the better investigations that have sought to estimate a population health production function since properly constructed measures of health care and pharmaceutical expenditures were used, and the effects of environmental and lifestyle factors were also taken into account.3

This study considers a life expectancy production function similar to that of Frech and Miller but with some methodological innovations that have meaningful effects on the magnitude of our results. First, our data distinguish life expectancies by age (40, 60, and 65 years) and by gender. However, specification tests indicate that pooling the life expectancy data across gender and age strata cannot be rejected. Therefore, we pool the data across the distribution of ages to produce a larger effective sample size and include interactions with age and gender variables to disentangle marginal effects for different age–gender strata. Second, this study uses a nonparametric jackknife technique to quantify the sampling variability of coefficient estimates. None of the previously mentioned studies employed resampling methods to derive variance estimates. Third, we include a measure of fruit and vegetable consumption in our regression. Fourth, we also use a different measure of tobacco consumption that yields different inferences compared to those of Frech and Miller. Finally, and most important, we find that failure to adjust for the effect of a country’s age distribution may create an omitted-variable bias in the coefficients for other determinants of life expectancy.4 This bias emphasizes the importance of age distribution variability in macroeconomic and public economic studies that incorporate aggregate life expectancy or mortality as a determinant of economic behavior.

We discuss these methodological issues and their effects on our results in the remainder of the paper. Accordingly, the next section of this paper discusses our methodology. In a following section, we discuss the results, with an emphasis of the effects of lifestyle factors and pharmaceutical consumption on life expectancy. In particular, we calculate and discuss the changes in lifestyle and pharmaceutical expenditures necessary to promote an additional year of life in different age–gender strata. Couched in these terms, our results suggest policy tactics for improving societal longevity and behavioral strategies for extending individual life expectancy. We close with a discussion of the robustness of our results, a brief discussion of some of the limitations of our work, and our conclusions.

2. Methods

Sample

Data are taken from the OECD Health Data 2000 database, which contains aggregate data on the health care systems of 29 of the 30 OECD countries. The database includes over 1200 indicators spanning the period 1960 to 1999, with official data up to 1998 and selected estimates for 1999. In

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3 We have described only the methodological differences between Peltzman (1987) and Miller and Frech (2000). The Babazono and Hillman (1994) study was “seriously flawed,” according to Miller and Frech (2000), so we will mention only that the study found pharmaceutical expenditures to be an insignificant determinant of infant mortality in a sample of OECD countries. The other studies mentioned in the paragraph (Lichtenberg 1996, 1998) used disease as the unit if observation and are not directly comparable.

4 Frech and Miller (1999, p. 54) refer to this as the “endogeneity of spending” effect, which is related to what has been called the “Sisyphus effect” (see Zweifel and Ferrari 1992). That is, if both pharmaceutical spending and life expectancy are functions of age distribution, then omitting the age distribution from a regression of life expectancy on pharmaceutical expenditures causes an omitted-variable bias. They argue that their regressions do not suffer from this issue.
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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, nonmedical determinants of health (alcohol and tobacco consumption), and economic references (GDP and monetary conversion rates). Variable definitions and descriptive statistics are presented in Table 1, and a complete explanation of the data is contained in the Data Appendix.

Since we are using data on OECD countries, inferences drawn from this study are valid only for more developed countries. We recognize the importance of ascertaining the determinants of population life expectancy in developing countries. However, it has been shown in many studies that public health services, such as clean water supply and sanitation services, provide the biggest benefits to societal health in these countries. According to Miller and Frech (2000, p. 34), these “services are a matter of civil engineering rather than healthcare.” Since our study focuses on the health care determinants of life expectancy, we select a sample containing developed countries only. In addition, data regarding drug consumption in developing nations are limited, precluding a detailed analysis of the effect of drug consumption in these countries.

We hypothesize that health care and lifestyle factors will have cumulative effects on life expectancy. That is, the consumption of factors over time by an individual will have either positive or negative effects on that individual’s longevity. While it is conceivable that the consumption of certain factors (e.g., alcohol, tobacco) by a mother would influence the life expectancy of her offspring, this represents a different model from the one we are interested in estimating. Thus, we choose 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 choose to lag the explanatory variables by roughly 15 years. The literature suggests that a lag of 20 years or more would be appropriate for alcohol and tobacco consumption (Corrao et al. 1993; Savolainen et al. 1993; Wise 1997; Khuder 2001). However, there is little empirical evidence regarding the appropriate lag length for indicators of health care consumption. Missing data preclude 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 include only one lag per variable.

It is widely held that studies of the productivity of health care may suffer from endogeneity bias. The allocation of medical resources to health should promote increased life expectancy, but as longevity increases, so do outlays on medical care. A relatively old population is likely to consume more health care than a relatively young population because of a greater prevalence of health problems. This quandary has been called the Sisyphus syndrome by Zweifel and Ferrari (1992) and others. In our model, the Sisyphus syndrome is not an issue of reverse causality as much as an omitted-variable problem. Life expectancy in a given year cannot cause the consumption of medical care in a preceding year. However, both life expectancy and health care consumption may be influenced by the age structure of a population measured concurrently or in previous years. To account for this effect and to ensure the consistency of our regression estimates, we include in our model the percentage of the population 65 years of age or older in 1985 (the same year in which pharmaceutical and other health care consumption are measured).

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5 These are the same data used by Miller and Frech (2000), except our data are more current.
6 While it was not formally tested, it could be argued that the set of factors affecting life expectancy at birth are different for those affecting life expectancy in adulthood. Excluding life expectancy at birth from this study may have been the determining factor in allowing us to pool data over the adult ages 40, 60, and 65 years.
Table 1. Variable Definitions and Descriptive Statistics<sup>a</sup>

<table>
<thead>
<tr>
<th>Variable</th>
<th>Definition</th>
<th>Mean</th>
<th>Standard Deviation</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Continuous variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LE97&lt;sub&gt;40M&lt;/sub&gt;</td>
<td>Years of life expectancy for males at age 40, 1997</td>
<td>36.55</td>
<td>1.00</td>
<td>34.70</td>
<td>38.10</td>
</tr>
<tr>
<td>LE97&lt;sub&gt;60M&lt;/sub&gt;</td>
<td>Years of life expectancy for males at age 60, 1997</td>
<td>19.17</td>
<td>0.82</td>
<td>17.40</td>
<td>20.10</td>
</tr>
<tr>
<td>LE97&lt;sub&gt;65M&lt;/sub&gt;</td>
<td>Years of life expectancy for males at age 65, 1997</td>
<td>15.46</td>
<td>0.76</td>
<td>13.70</td>
<td>16.30</td>
</tr>
<tr>
<td>LE97&lt;sub&gt;40F&lt;/sub&gt;</td>
<td>Years of life expectancy for females at age 40, 1997</td>
<td>41.71</td>
<td>1.11</td>
<td>39.50</td>
<td>43.50</td>
</tr>
<tr>
<td>LE97&lt;sub&gt;60F&lt;/sub&gt;</td>
<td>Years of life expectancy for females at age 60, 1997</td>
<td>23.38</td>
<td>0.98</td>
<td>21.50</td>
<td>25.20</td>
</tr>
<tr>
<td>LE97&lt;sub&gt;65F&lt;/sub&gt;</td>
<td>Years of life expectancy for females at age 65, 1997</td>
<td>19.19</td>
<td>0.91</td>
<td>17.40</td>
<td>20.80</td>
</tr>
<tr>
<td>GDP85</td>
<td>Gross domestic product per capita, 1985 U.S. dollars</td>
<td>11,719.11</td>
<td>2,751.13</td>
<td>6,105.00</td>
<td>16,976.00</td>
</tr>
<tr>
<td>PHARM85</td>
<td>Pharmaceutical expenditures per capita, 1985 U.S. dollars</td>
<td>171.26</td>
<td>72.20</td>
<td>73.21</td>
<td>400.34</td>
</tr>
<tr>
<td>HEALTH85</td>
<td>Health expenditures (not including pharmaceuticals) per capita, 1985 U.S. dollars</td>
<td>1,077.71</td>
<td>461.27</td>
<td>260.48</td>
<td>1,938.25</td>
</tr>
<tr>
<td>AGEDIST85</td>
<td>Percentage of population 65 years of age and older, 1985</td>
<td>13.02</td>
<td>2.08</td>
<td>10.10</td>
<td>17.19</td>
</tr>
<tr>
<td>SMOKE80</td>
<td>Grams of tobacco consumed annually per capita by persons age 15 or older, 1980</td>
<td>2,727.33</td>
<td>530.18</td>
<td>1,492.00</td>
<td>3,588.00</td>
</tr>
<tr>
<td>ALCOHOL80</td>
<td>Liters of ethyl alcohol consumed annually per capita by persons age 15 or older, 1980</td>
<td>11.99</td>
<td>3.82</td>
<td>5.30</td>
<td>20.60</td>
</tr>
<tr>
<td>BUTTER80</td>
<td>Kilograms of butter consumed annually per capita, 1980</td>
<td>6.01</td>
<td>4.05</td>
<td>0.50</td>
<td>13.90</td>
</tr>
<tr>
<td>VEG80</td>
<td>Kilograms of fruits and vegetables consumed annually per capita, 1980</td>
<td>187.01</td>
<td>66.20</td>
<td>70.90</td>
<td>362.20</td>
</tr>
<tr>
<td><strong>Discrete variables</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MALE</td>
<td>Dummy variable taking on value of 1 if dependent variable was life expectancy for males and 0 otherwise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AGE60</td>
<td>Dummy variable taking on value of 1 if dependent variable was life expectancy at age 60 and 0 otherwise</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>AGE65</td>
<td>Dummy variable taking on value of 1 if dependent variable was life expectancy at age 65 and 0 otherwise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPAIN</td>
<td>Dummy variable taking on value of 1 if country was Spain and 0 otherwise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> 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.
**Model Specification**

We use a log-linear functional form in modeling the data. There are several reasons for this. First, it allows us to interpret our parameter estimates as elasticities. Second, it allows 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 are centered to yield a more plausible interpretation of the marginal effects. A dummy variable for Spain is added to the model to control for the imputation of missing tobacco consumption data for this country.

Initially, ordinary least squares (OLS) is used to estimate separate models for the six strata \( j = 1, \ldots, 6 \), defined by age and gender. The age–gender strata are ages 40, 60, and 65 for both males and females. Country \( i = 1, \ldots, 19 \) is then the unit of observation in the following life expectancy regression:

\[
\ln \text{LE97}_ij = \beta_{0j} + \beta_{1j} \ln \text{GDP85}_i + \beta_{2j} \ln \text{PHARM85}_i + \beta_{3j} \ln \text{HEALTH85}_i \\
+ \beta_{4j} \ln \text{AGEDIST85}_i + \beta_{5j} \ln \text{ALCOHOL80}_i + \beta_{6j} \ln \text{SMOKE80}_i \\
+ \beta_{7j} \ln \text{BUTTER80}_i + \beta_{8j} \ln \text{VEG80}_i + \beta_{9j} \text{SPAIN}_i + e_{ij},
\]

Equation 1

All continuous variables are measured in logarithms; variable definitions are given in Table 1. Tests on the OLS residuals indicate that the life expectancy data for the six age–gender strata can be pooled into a single regression. (See the Technical Appendix for test details.) This is in contrast to the approach adopted by Miller and Frech (2000), who estimated a separate regression for each age stratum but pooled data across genders.

Based on the preceding results, we pool life expectancy data for the six age–gender strata, adding dummy variables for age and gender to the model to control for differences in the intercept term.\(^7\) We use residual maximum likelihood to estimate a mixed model treating country as a random effect.\(^8\) Given our small sample size and concerns regarding possible heteroscedasticity, inferences are based on jackknife estimates of the standard errors (MacKinnon and White 1985). However, for comparative purposes, empirical standard errors are also calculated (but not presented) based on the sandwich estimator (Huber 1967; White 1980). An important empirical finding is that the jackknife standard errors are always greater than or equal to the empirical standard errors, implying that the statistical significance of estimates in previous studies may have been overstated.

Equation 2 is 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 \( e_{ij} \) are independently identically distributed errors (at the level of age–gender stratum within country) from a \( N(0, \sigma_e^2) \) distribution and are independent of the \( u_i \).

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\(^7\) For the pooled regression, we used a sequential modeling procedure to determine which interactions between the continuous covariates and indicator variables for age–gender strata should be included in the final model.

\(^8\) Residual maximum likelihood produces unbiased estimates of the conditional variance components by correcting the usual maximum likelihood estimator for the degrees-of-freedom loss associated with estimating the conditional mean. The usual maximum likelihood variance estimates are biased in small samples. See Patterson and Thompson (1971) for the theory and Brown and Prescott (1999) for applications to mixed models.
\[
\ln LE97_{ij} = \beta_0 + \beta_1 \ln GDP85_i + \beta_2 \ln PHARM85_i + \beta_3 \ln HEALTH85_i + \beta_4 \ln AGEDIST85_i
+ \beta_5 \ln ALCOHOL80_i + \beta_6 \ln SMOKE80_{ii} + \beta_7 \ln BUTTER80_i + \beta_8 \ln VEG80_i
+ \beta_9 \ln SPAIN_i + \beta_{10} \ln MALE_{ij} + \beta_{11} \ln AGE60_{ij} + \beta_{12} \ln AGE65_{ij} + \beta_{13} \ln MALE_{ij} \times \ln AGE60_{ij}
+ \beta_{14} \ln MALE_{ij} \times \ln AGE65_{ij} + \beta_{15} \ln AGE60_{ij} \times \ln GDP85_i + \beta_{16} \ln AGE65_{ij} \times \ln GDP85_i
+ \beta_{17} \ln AGE60_{ij} \times \ln PHARM85_i + \beta_{18} \ln AGE65_{ij} \times \ln PHARM85_i
+ \beta_{19} \ln SMOKE80_i + \beta_{20} \ln AGE65_{ij} \times \ln SMOKE80_i
+ \beta_{21} \ln MALE_{ij} \times \ln ALCOHOL80_i + \beta_{22} \ln MALE_{ij} \times \ln VEG80_i
+ \beta_{23} \ln AGE60_{ij} \times \ln VEG80_i + \beta_{24} \ln AGE65_{ij} \times \ln VEG80_i + u_i + \epsilon_{ij}. \tag{2}
\]

A number of tests are performed to assess the model’s goodness of fit. Also, sensitivity analyses are performed for the lag structure and monetary conversion rates that are used. See the Technical Appendix for details. All statistical analyses are performed using SAS Release 8.02 (SAS Institute, Inc., Cary, North Carolina) and Stata/SE 8.0 (Stata Corporation, College Station, Texas).

3. Results and Discussion

Table 2 presents our results for the estimation of Equation 2. To highlight the importance of a country’s age distribution in estimation of the production function, we present two regressions: one including AGEDIST85 and one excluding it. To the right of the coefficient estimates are the corresponding jackknife standard errors, which we find to be more conservative than the empirical robust standard errors (clustered or unclustered). First, we find that the age distribution of a population in 1985 is a significant determinant of life expectancy with an elasticity of −0.073. That is, if the percentage of the population over 65 in an average OECD country were doubled, average life expectancy for the population of males and females in the three age strata would decline 7.3%. If the percentage of the population over 65 increased 1%, average life expectancy would decline approximately 54 days.9

The only independent variable that is appreciably affected by the inclusion or exclusion of the age distribution variable is pharmaceutical expenditures in 1985 (PHARM85). When AGEDIST85 is excluded, its magnitude is 0.009 and is insignificant. When AGEDIST85 is included, the estimate increases to 0.027 and is significant at the 10% level based on conservative jackknife standard errors. This means that when pharmaceutical expenditures are doubled, the life expectancy at 40 years increases 2.7% (or 411 days for females, 360 days for males).10 It also means that the conditional correlation between pharmaceutical expenditures and the age distribution in 1985 may be such that excluding the age distribution variable biases the pharmaceutical coefficient downward. Older populations use more drugs, and this must be taken into account. Again, we do not consider this a simultaneity issue because life expectancy in 1997 cannot cause drug consumption in 1985. It is an omitted-variable problem that should be recognized in subsequent health care research. As such, all discussions that follow are for the model including the age distribution measure.

9 Frech and Miller (1999) found a similar measure of the population age distribution to be insignificant. We suspect that the difference in significance may be attributed to the decreased sampling variability associated with the larger sample size afforded from pooling the data across age–gender strata.

10 For example, the estimated number of days of life expectancy gained for females at age 40 was 0.027 × 365 × 41.71, where 41.71 was the average female life expectancy at that age. Similar calculations were performed for other age categories and for males.
### Table 2. Regression Parameter Estimates: Life Expectancy Regression

<table>
<thead>
<tr>
<th>Variable</th>
<th>Including Age Distribution</th>
<th>Excluding Age Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>Standard Error</td>
</tr>
<tr>
<td>CONSTANT</td>
<td>3.726a</td>
<td>(0.004)</td>
</tr>
<tr>
<td>MALE</td>
<td>-0.132bc</td>
<td>(0.004)</td>
</tr>
<tr>
<td>AGE60</td>
<td>-0.579bc</td>
<td>(0.003)</td>
</tr>
<tr>
<td>AGE65</td>
<td>-0.777bc</td>
<td>(0.003)</td>
</tr>
<tr>
<td>MALE × AGE60</td>
<td>-0.067bc</td>
<td>(0.003)</td>
</tr>
<tr>
<td>MALE × AGE65</td>
<td>-0.085bc</td>
<td>(0.005)</td>
</tr>
<tr>
<td>ln GDP85</td>
<td>-0.033</td>
<td>(0.058)</td>
</tr>
<tr>
<td>AGE60 × ln GDP85</td>
<td>0.031a</td>
<td>(0.009)</td>
</tr>
<tr>
<td>AGE65 × ln GDP85</td>
<td>0.056a</td>
<td>(0.013)</td>
</tr>
<tr>
<td>ln PHARM85</td>
<td>0.022b</td>
<td>(0.014)</td>
</tr>
<tr>
<td>AGE60 × ln PHARM85</td>
<td>0.019b</td>
<td>(0.009)</td>
</tr>
<tr>
<td>AGE65 × ln PHARM85</td>
<td>0.021b</td>
<td>(0.011)</td>
</tr>
<tr>
<td>ln HEALTH85</td>
<td>0.036</td>
<td>(0.030)</td>
</tr>
<tr>
<td>ln AGEDIST85</td>
<td>-0.073a</td>
<td>(0.032)</td>
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<tr>
<td>ln SMOKE80</td>
<td>-0.067a</td>
<td>(0.026)</td>
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<tr>
<td>AGE60 × ln SMOKE80</td>
<td>-0.036a</td>
<td>(0.015)</td>
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<td>AGE65 × ln SMOKE80</td>
<td>-0.045a</td>
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<td>ln ALCOHOL80</td>
<td>-0.019</td>
<td>(0.019)</td>
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<tr>
<td>MALE × ln ALCOHOL80</td>
<td>-0.034b</td>
<td>(0.018)</td>
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<td>ln BUTTER80</td>
<td>0.022a</td>
<td>(0.010)</td>
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<tr>
<td>ln VEG80</td>
<td>0.081a</td>
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<td>MALE × ln VEG80</td>
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<td>AGE60 × ln VEG80</td>
<td>0.028a</td>
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<tr>
<td>AGE65 × ln VEG80</td>
<td>0.041a</td>
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</tbody>
</table>

*Significantly different from 0, p < 0.05, two tailed.
*Significantly different from 0, p < 0.10, two tailed.
*To be interpreted as an elasticity, this must be converted using the formula: E = e^β - 1 (Kennedy 1998).
*Significance tests were performed using jackknife standard errors, reported in parentheses.

Because we pool the data and include age interactions in our model, the effect of pharmaceuticals on life expectancy at ages 60 and 65 cannot be directly inferred from Table 2. Using the estimated "AGE60 × PHARM85" and "AGE65 × PHARM85" interactions of 0.019 and 0.021, respectively, our regression yields an elasticity of 0.046 for the effect for pharmaceuticals on life expectancy at age 60 and an elasticity of 0.048 for the effect for pharmaceuticals on life expectancy at age 65. With standard errors of 0.016, both elasticities are significant at the 95% level. While the elasticity of pharmaceutical consumption increases with age (e.g., 0.027 at age 40 and 0.046 at age 60), the actual predicted effect (in terms of life expectancy gained per unit increase in pharmaceutical consumption) is decreasing in age.11

A reasonable policy question is, What amount of pharmaceutical expenditure is required to increase average life expectancy by one year in each of the age–gender strata? Table 3 provides some

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11 At age 40, the remaining number of years of expected life is large. Therefore, a small increase in the elasticity of pharmaceutical consumption would be expected to yield a large increase in the predicted gain in life expectancy. However, at more advanced ages the remaining life expectancy is sufficiently small such that even a large increase in the elasticity would yield a small increase in the predicted gain. This result applies only to the sample-wide estimated parameters. The result ignores differences across countries in the percentage of drugs administered to each age-group and their relative effectiveness in each age-group of increasing life expectancy. This source of variability is unavailable in the OECD data and could not be incorporated into the analysis.
Table 3. Additional Activity Required to Increase the Life Expectancy by One Year

<table>
<thead>
<tr>
<th>Activity (units)</th>
<th>Males Age 40</th>
<th>Males Age 60</th>
<th>Males Age 65</th>
<th>Females Age 40</th>
<th>Females Age 60</th>
<th>Females Age 65</th>
</tr>
</thead>
<tbody>
<tr>
<td>1985 pharmaceutical expenditures (U.S. $/capita)</td>
<td>101.33%</td>
<td>113.40%</td>
<td>134.76%</td>
<td>88.80%</td>
<td>92.98%</td>
<td>108.56%</td>
</tr>
<tr>
<td></td>
<td>(173.54)</td>
<td>(194.21)</td>
<td>(230.79)</td>
<td>(152.08)</td>
<td>(159.24)</td>
<td>(185.92)</td>
</tr>
<tr>
<td>1980 tobacco consumption (g/capita)</td>
<td>-40.84%</td>
<td>-50.65%</td>
<td>-57.75%</td>
<td>-35.78%</td>
<td>-41.53%</td>
<td>-46.53%</td>
</tr>
<tr>
<td></td>
<td>(1113.84)</td>
<td>(1381.39)</td>
<td>(1575.03)</td>
<td>(975.84)</td>
<td>(1132.66)</td>
<td>(1269.03)</td>
</tr>
<tr>
<td>1980 fruit/vegetable consumption (kg/capita)</td>
<td>24.65%</td>
<td>37.53%</td>
<td>42.55%</td>
<td>29.60%</td>
<td>39.24%</td>
<td>42.71%</td>
</tr>
<tr>
<td></td>
<td>(46.10)</td>
<td>(70.18)</td>
<td>(79.57)</td>
<td>(55.36)</td>
<td>(73.38)</td>
<td>(79.87)</td>
</tr>
<tr>
<td>1980 butter consumption (kg/capita)</td>
<td>124.36%</td>
<td>237.11%</td>
<td>294.01%</td>
<td>108.98%</td>
<td>194.42%</td>
<td>236.87%</td>
</tr>
<tr>
<td>1980 alcohol consumption (liters/capita)</td>
<td>-51.62%</td>
<td>-98.42%</td>
<td>-122.04%</td>
<td>-126.18%</td>
<td>-225.11%</td>
<td>-274.27%</td>
</tr>
<tr>
<td></td>
<td>(-6.19)</td>
<td>(-11.80)</td>
<td>(-14.63)</td>
<td>(-15.13)</td>
<td>(-26.99)</td>
<td>(-32.88)</td>
</tr>
</tbody>
</table>

insights. The left (right) half of the table depicts the results for males (females); the columns represent the age strata (40, 60, and 65 years). For example, the table indicates that for a 60-year-old male, an average increase in pharmaceutical expenditures of about $194 per capita (a 113% increase over the sample average of $171) would increase average life expectancy by one year. The corresponding number for 60-year-old females is somewhat less, that is, about $159 (93%) per capita. Doubling annual pharmaceutical expenditures from the sample average of $171 per capita adds about one year of life expectancy for males at age 40 and a little less than one year of life expectancy for females at age 65. It is also clear from Table 3 that the marginal benefit of pharmaceutical spending is decreasing in age. For example, for males age 40, 60, and 65 years to gain an additional year of life expectancy requires pharmaceutical spending increases of 101.33%, 113.40%, and 134.76%, respectively. These results are averages across all OECD countries in the sample; however, we could use the percentages in Table 3 to impute country-specific results. For example, in the United States annual per capita spending on pharmaceuticals was $155 in 1985; therefore, to add an additional year of life expectancy for 60-year-old males would only require an increase of about $155 \times 1.134 = $176 per capita per annum.\footnote{This calculation ignores three things. First, there are slight differences in life expectancies across counties that should technically be taken into account. Second (and more important), the average sample-wide parameter estimates do not accurately capture differences in drug product mix within a country. Since different drugs produce different longevity effects, the calculation will underestimate life expectancy gains in a country that uses a higher (than average) percentage of drugs that enhance life expectancy. It will similarly overestimate in a country that uses a higher percentage of drugs that do not enhance life expectancy. For example, antidepressants probably do not enhance life expectancy directly, so a country that consumes large relative quantities of antidepressants will tend to have overestimated life expectancy gains from increasing drug consumption. Finally, the calculation ignores price differentials across countries for individual drugs (although this is partially mitigated by indexing aggregate drug expenditures to the U.S. dollar). See, for example, Danzón and Furukawa (2003) and Danzón and Ketcham (2003).}

Lifestyle factors, such as the consumption of alcohol, tobacco, butter, and fruits and vegetables, also have important effects on life expectancy after controlling for the effects of weight and health care consumption. Contrary to the finding of Miller and Frech (2000), tobacco consumption (SMOKE80) has a statistically significant negative effect on life expectancy. Results in Table 2 indicate that
doubling tobacco consumption per capita is associated with an approximate 6.7% reduction in population life expectancy at age 40 (1020 days for females, 894 days for males). Using the estimated “AGE60 \times SMOK80” and “AGE65 \times SMOK80” interactions of –0.036 and –0.045, respectively, the regression yields an elasticity of –0.103 for the effect for tobacco on life expectancy at age 60 and an elasticity of –0.112 for the effect for tobacco on life expectancy at age 65. With standard errors of 0.033 and 0.035, respectively, both elasticities are significant at the 5% level. Table 3 couches our tobacco results in terms of the reduction needed to increase life expectancy by one year. For example, average females at age 40 years would add an additional year of life expectancy if they decrease tobacco consumption by about 976 grams per year (a 36% reduction from an OECD average of 2727 grams per capita per year in 1980). If a cigarette contains about 1.5 grams of tobacco, this is equivalent to a per capita decrease of about 651 cigarettes per year, or just under two cigarettes per day for this group. It is not entirely clear from Table 3 whether an increase in pharmaceutical expenditures or a decrease in tobacco consumption would be more effective in improving longevity. The units presented are not comparable, and they do not reflect the true cost of creating and implementing public policy. However, the results provide insight into the relative magnitudes of changes in drug consumption and healthy lifestyle required to promote longevity in developed countries.

Per capita fruit and vegetable consumption (VEG80) has a statistically significant positive effect on life expectancy, with a coefficient (standard error) of 0.081 (0.023) for females at age 40 (Table 2). After taking into consideration interactions with the age and gender variables, the following marginal effects (with standard errors in parentheses) result: 0.110 (0.025) for females at age 60, 0.123 (0.026) for females at age 65, 0.111 (0.029) for males at age 40, 0.140 (0.030) for males at age 60, and 0.153 (0.031) for males at age 65. All the results are significant at the 5% level. Table 3 shows that average females at 40 years would add an additional year of life expectancy by increasing fruit and vegetable consumption by about 55 kilograms per year (a 30% increase) from an OECD average of 187 kilograms per capita per year in 1980. This is equivalent to increasing fruit and vegetable consumption by about one-third pound per day.

Our findings with respect to fruit and vegetable consumption likely reflect differences in intake among groups. In many developed countries, fruit and vegetable consumption appears to increase with increasing age among adults (Krebs-Smith et al. 1995a, b; Dong and Erens 1997; Krebs-Smith et al. 1997). 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 (Krebs-Smith et al. 1995a, b). Since we measure intake using the number of kilograms consumed annually, it is not surprising that the effect of fruit and vegetable consumption on life expectancy is greater for males than females.

In Table 2, the parameter estimate for butter consumption per capita (BUTTER80) is 0.022 and is statistically significant at the 5% level. This implies that doubling butter consumption increases average life expectancy by 2.2% across age and gender strata. Interactions of butter with age and gender variables are insignificant. There are several possible explanations for the apparent effect of butter consumption on life expectancy. First, it is possible that the positive effect is the result of vitamin fortification. In developed countries, where milk products are fortified with vitamins A and D, it is conceivable that butter would have a positive effect on a population’s health. Second, one might hypothesize that the positive effect of butter consumption is 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 be due to omitted-variable bias. Since we explicitly control for the
effect of wealth, it seems unlikely that our measure of fat intake is simply capturing an omitted income effect (i.e., that people in wealthier countries consume fattier diets).

Our findings with respect to butter consumption are consistent with those of Wolfe and Gabay (1987), who studied the relationship between negative changes in lifestyle and health status in a sample of OECD countries. Although they found that 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. Others have reported a nonlinear relationship between fat consumption and measures of population health. Gage and O’Connor (1994) 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. Similarly, Frech and Miller (1999) reported that low levels of animal fat (not butter) consumption had a strong positive effect on life expectancy, while higher levels were associated with reduced life expectancy. We chose not to model a nonlinear association between butter intake and life expectancy since there was little empirical evidence supporting a nonlinear relationship (see the Data Appendix for details).

Though alcohol consumption (ALCOHOL80) does not have a statistically significant effect on female life expectancy, its effect on the life expectancy of males is both significant and negative. This finding most likely reflects a difference in alcohol intake between males and females and is consistent with the findings of Cochrane, St. Leger, and Moore (1978) and Frech and Miller (2000). 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 (Baum-Baicker 1985) and cardiovascular (Moore and Pearson 1986; Stampfer et al. 1988; Boffetta and Garfinkel 1990; Razay et al. 1992) benefits, it also increases risks for hemorrhagic stroke (Camargo 1989), adverse medication reactions (Shinn and Shrewsbury 1988; Gilman et al. 1990), and certain types of cancer (Willett et al. 1987; Klatsky et al. 1988). 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 because of 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.13

4. Conclusions

In a sample of more developed countries, we find that drug consumption, as measured by per capita pharmaceutical expenditures, has a positive effect on population life expectancy at various ages. The predicted number of days or years of expected life per unit increase in pharmaceutical consumption appears to decline with increasing age. Our research also suggests that the correlation between pharmaceutical consumption and a country’s age distribution creates an omitted-variable bias in the elasticity of pharmaceutical consumption when the age distribution is ignored. This is a classic case of the omitted-variable problem, which fortunately seems to affect only the elasticity of pharmaceutical consumption and not those of the other determinants of life expectancy. The omission

13 We have excluded a discussion of the effects of wealth and nonpharmaceutical health care expenditures. Our estimates for the marginal effect of wealth (GDP85) and nonpharmaceutical health care expenditures (HEALTH85) were insignificant. The effect of wealth was highly positively correlated with and swamped by the effect of butter consumption (BUTTER80). This suggests that richer countries consume more butter—a reasonable result. The insignificant effect of nonpharmaceutical health care expenditures is consistent with the findings of Miller and Frech (2000).
creates a downward bias (at least empirically), suggesting the marginal effect of drug consumption on health will be understated if age distribution is ignored. In this case, the nature of the correlation is clear: an older society consumes more drugs in the short run, and drugs may change the age profile of society in the long run. However, correlations between the age distribution of a country and other macroeconomic or aggregate variables may be more subtle and, hence, more easily overlooked in empirical analyses. Insofar as the age distribution of a country affects voting, politics, and ultimately policy, it is important to acknowledge the potential for correlations between the age of a society and any aggregate measure that may be influenced by policy.

Data Appendix

Data are taken from the OECD Health Data 2000 database. Except where noted, the data are identical to those in Miller and Frech (2000) with the difference that our data are more current (taken from a more recent version of the OECD Health Data database). Because of missing data, we restrict our analysis to 19 of the 30 OECD countries. We exclude Switzerland from our sample because of the limited availability of pharmaceutical and health-specific purchasing power parity (PPP) exchange rates as well as tobacco and alcohol consumption data. We also exclude Turkey from our sample since it is relatively underdeveloped when compared with the other member countries of the OECD. Variable definitions and descriptive statistics are presented in Table 1. All continuous variables are measured in logarithms.

Life Expectancies (LE97)

The dependent variables include life expectancies for males and females at ages 40, 60, and 65. These are measured in number of years of life expectancy for each age–gender stratum in 1997. Life expectancy data are missing for Ireland in 1997, and we substitute 1995 data in each age–gender grouping for this country in our model. We include life expectancy at age 65, while Miller and Frech (2000) did not.

Wealth (GDP85)

We measure wealth or income using per capita GDP in 1985. Following Miller and Frech (2000), it is converted into U.S. dollars by dividing by the appropriate 1985 PPP conversion factor provided in the OECD Health Data database.

Pharmaceutical Consumption (PHARM85)

Pharmaceutical consumption is measured in 1985 per capita expenditures for each country. It is computed as total per capita expenditures on pharmaceuticals and other medical nondurables minus per capita expenditures on medical nondurables (in cases where data for the latter are available). Our measure of pharmaceutical consumption includes expenditures for outpatient prescription and over-the-counter medications as well as pharmacists’ remuneration. In addition to conventional GDP-based conversion factors, the OECD Health Data database includes PPP conversion factors for pharmaceutical expenditures. Following Miller and Frech (2000), we use the 1985 PPP conversion factor for pharmaceutical expenditures to convert expenditures to U.S. dollars. Miller and Frech (2000) argue that pharmaceutical expenditures converted to U.S. dollars using GDP PPP exchange rates underestimate actual pharmaceutical expenditures outside the United States. The drug-specific PPP exchange rates yield results that are consistent with those obtained using more accurate conversion factors developed by Szuba (1986) and others. Unfortunately, the exchange rates are available only for a limited number of years (i.e., 1980, 1985, 1990, 1993, and 1996). Therefore, this influenced the lag we use for pharmaceutical expenditures.

Nonpharmaceutical Health Care Consumption (HEALTH85)

Our measure of health care expenditures in 1985 is computed by subtracting PHARM85 from total per capita expenditures on health care. Total health care expenditures in 1985 are missing for Greece and are estimated by summing total current expenditures on health and total investments in medical facilities. The OECD Health Data database also includes specific PPP conversion factors for health care expenditures. Thus, following Miller and Frech (2000), these exchange rates are used to convert HEALTH85 to U.S. dollars.

Age Distribution (AGEDIST85)

This is measured as the percentage of the population 65 years of age or older in 1985 (the same year in which pharmaceutical and other health care consumption are measured). We also experimented with the percentage of the population 65 years of age or older in 1980, but this had little effect on our results. Indeed, the unconditional correlation between the age
distribution in 1980 and 1985 is 0.94 and is significant at the 5% level. Frech and Miller (1999) did not include this variable in their primary analysis, though they claimed to have evaluated its influence in sensitivity analyses.

Alcohol Consumption (ALCOHOL80)
Alcohol consumption is measured in liters consumed per capita by persons aged 15 or older in 1980. We substitute 1983 data for Greece since data on alcohol consumption in 1980 are missing for this country.

Smoking Behavior (SMOKE80)
Smoking behavior is measured as grams of tobacco consumed per capita by persons aged 15 or older in 1980. Data on tobacco consumption in 1980 are missing for Germany, Ireland, and Italy. For these countries, 1979 data are used instead. Data on tobacco consumption are unavailable for Spain in any year. For this country, we substitute the mean value for tobacco consumption in 1980 for the other countries included in our sample. As noted, a dummy variable is included in the regression analyses to account for this imputation.

Fat Consumption (BUTTER80)
Miller and Frech (2000) 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 is not available in the Health Data 2000 database. Therefore, we use butter consumption in kilograms per capita in 1980 as an alternate measure of animal fat intake. This includes quantities of butter used in food preparations or mixed with other fats to obtain particular types of margarine or cooking fats. Certain studies (Gage and O’Connor 1994; Frech and Miller 1999; Miller and Frech 2000) 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 investigated several methods of accounting for nonlinearity in the association between butter intake and life expectancy (e.g., including quadratic terms, categorization using dummy variables). However, we found no strong evidence supporting a curvilinear relationship.

Fruit and Vegetable Consumption (VEG80)
As a measure of positive dietary intake, we include fruit and vegetable consumption in kilograms per capita in 1980. Miller and Frech (2000) did not include such a measure in their analysis.

Technical Appendix

Poolability Tests
For the regressions specified in Equation 1, we performed a hypothesis test to determine whether the intercept varied among the six age–gender strata followed by a test for homogeneity of regression or parallelism. These tests are often ascribed to Chow (1960); however, they were described earlier in a number of other sources (e.g., Kendall 1948; Kempthorne 1952; Rao 1952). 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–gender strata in the intercept term ($F_{6,99} = 7.711; p < 0.0001$). However, the other parameters did not appear to vary significantly among the strata ($F_{65,54} = 1.29; p < 0.19$).

Conventional tests for poolability assume spherical disturbances (Baltagi 2001). In the presence of nonspherical 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 (2001), conventional tests for poolability should be used only 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 (1957) and Zellner (1962). Using the methods described by Baltagi (2001), 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–gender strata in the intercept term ($F_{65,99} = 2.165; p < 0.0001$), the parameter vectors (excluding the intercept) did not vary significantly among the strata ($F_{65,54} = 1.18; p < 0.28$).

Goodness-of-Fit Tests
Several goodness-of-fit tests were performed for the model specified in Equation 2. The D’Agostino-Pearson test (D’Agostino and Pearson 1973; D’Agostino et al. 1990) was used to confirm the normality of the residuals. The combined
residuals were normally distributed ($\chi^2 = 0.17, p = 0.92$), as were the predicted random effects ($\chi^2 = 3.16, p = 0.21$) and random error component ($\chi^2 = 2.07, p = 0.36$). Multicollinearity was assessed using Belsley’s condition index (Belsley, Kuh, and Welsch 1980). Multicollinearity appeared to be much less of an issue in our model than in some previous research. The condition index for our model was 9.55, which did not exceed the commonly accepted threshold of 20–30 (Belsley, Kuh, and Welsch 1980; Greene 2000). Ramsey’s regression specification error test (Ramsey 1969) was used to test for omitted variables and/or incorrect functional form. The test failed to reject the null hypothesis ($\chi^2 = 1.58, p = 0.66$), suggesting that the model’s functional form was correctly specified. We added second-through fourth-order polynomials of the fitted values to the model and found them to be jointly insignificant. Finally, the Breusch–Pagan Lagrange multiplier test (Breusch and Pagan 1980) and the Hausman test (Hausman 1978) were performed to evaluate the efficiency and consistency, respectively, of the mixed-effects model. The Breusch–Pagan test rejected OLS in favor of a mixed-effects specification ($\chi^2 = 54.47, p < 0.0001$), while the Hausman test failed to reject the null hypothesis that the individual effects were uncorrelated with the other regressors. Thus, the mixed-effects model appeared to be favored over pooled OLS.

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. Excluding Spain from the sample had no appreciable effect on any of our findings. 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. (The primary reason for Switzerland’s exclusion was the lack of OECD PPP exchange rates in 1985.) 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 nondrug health care consumption were somewhat attenuated. However, the significance of the parameter estimates was not greatly changed. 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 United States) 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: (i) 1985 economic/age distribution data (GDP85, PHARM85, HEALTH85, AGEDIST85) and 1980 lifestyle data (ALCOHOL80, SMOKE80, BUTTER80, VEG80) to provide a comparison with our base model; (ii) 1985 economic, age distribution, and lifestyle data; and (iii) 1990 economic, age distribution, 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. The results were generally consistent with those presented in Table 2.

References


