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Wallace Huffman, Sonya K. Huffman, Abebayehu Tegene, Kyrre Rickertsen

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# The Economics of Obesity-Related Mortality Among High Income Countries

**Wallace E. Huffman**
C.F. Curtiss Distinguished Professor of Agriculture and Professor of Economics
Iowa State University
Ames, IA 50011
515-294-6359
whuffman@iastate.edu

**Sonya Huffman**
Adjunct Assistant Professor of Economics
Iowa State University
Ames, IA 50011

**Abebayehu Tegene**
Branch Chief, Food Economics Division
Economic Research Service, USDA
Washington, DC 20036-5831

**Kyrre Rickertsen**
Professor of Economics
Norwegian University of Life Sciences
Aas, Norway

**Abstract**: Health production and supply functions based on models for productive households are established. Data for 18 high income countries over 1971-2001 are used in the empirical analysis. In the health production function, mortality from cardiovascular diseases and diabetes is positively related to inputs of calories and sweeteners but not to input of fat or to national health care. In the health supply function, a high real price of food, real wage rate and non-labor income, a modest level of socialized medicine, and a low labor force participation rate decrease mortality. A cheap food policy erodes gains from reduced smoking and better treatments for high cholesterol levels and hypertension that have occurred over the last three decades.

**Key Words**: health, household production, food prices, obesity, mortality, high income countries
J**EL codes**: D13, I12, I18, Q18

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## I. Introduction

Adult obesity rates are rising in all high income countries, and they are both high and rising in the United States (US), United Kingdom (UK), Australia and New Zealand. Although obesity rates in other high income industrialized countries are much lower than in the US, UK, Australia and New Zealand, and rising less rapidly, the upward trend in obesity rates is ubiquitous in these countries (OECD 2004).[1]

Obesity is associated with elevated health risks, including cardiovascular diseases, diabetes, and some forms of cancer; higher health care costs due to chronic illnesses; and premature deaths (US DHHS 2001; Finkelstein et al. 2003; OECD 2004; AIHW 2004). Over the past four decades, reduced smoking (OECD 2005a, pp. 80-81) and better treatment of high cholesterol levels and hypertension have contributed to reduced human mortality from cardiovascular diseases in high income countries (OECD 2005a; Cutler 2001). However, cardiovascular diseases remain the leading cause of death in all high income industrialized countries except for France and Japan, where cancer is the leading cause of mortality (OECD 2005a, pp. 22-23). Increasing rates of obesity and diabetes mellitus are major risk factors for cardiovascular diseases (AIHW 2004). There is, however, a time lag between the onset of obesity and related health problems (OECD 2005a, p. 15; AIHW 2004) and, hence, as adults and children in developed countries become obese, future risks of chronic health problems will rise dramatically.

Human energy imbalance is due to a complex set of factors. First, the availability of unhealthy foods has increased both at home and away from home—vending machines and fast food stores are ubiquitous. New market goods, including baked and processed foods, caloric-sweetened

[1] Mendez and Popkin (2004) emphasize that with as poorer countries develop more their populations become susceptible to obesity, too. Hence, obesity is not only a problem in rich countries.

drinks, and sweet and salty snacks, are substituted for home-produced goods (Cutler et al. 2003; Kuchler et al. 2005). Second, mechanization and automation of marketplace work and the shift of workers from agriculture and manufacturing to service industries have reduced the energy requirement of labor market work (Lakdawalla and Philipson 2002; Mendez and Popkin 2004). Third, improvements in transportation have reduced the energy intensity of commuting, especially in the U.S., Canada, Australia, and New Zealand. Fourth, rapid improvements in home production technology and smaller family sizes have reduced the amount of work to be done in household production (Huffman 2006). Fifth, in high income countries leisure time has become passive or sedentary, e.g., TV viewing, web surfing and playing computer games (Juster and Stafford 1991, pp. 477; US Department of Labor 2006). One summary measure of these cumulative changes is that the price of food at home, relative to a comprehensive market basket of inputs into household production, has fallen by 1 percent per year over the past five decades (Huffman 2006; Huffman and Evenson 2006).[2] Also, the prices of caloric-sweetened drinks and high fat fast foods have fallen relative to the price of fresh fruits and vegetables (USDA 2005).

Data on obesity, e.g., the body mass index or BMI, on a national random sample of individuals with economic data over time do not exist, and national aggregate data on obesity rates exist only since about 1990, and then sporadically (OECD 2005a; Louriero and Nayga 2005). The new OECD health report provides national aggregate data for high income industrialized countries on mortality due to cardiovascular and other diseases over the past 30 years (OECD 2005a). Because obesity is a major risk factor for cardiovascular diseases and diabetes that tend to be progressive, the risks due to these diseases increase. Hence, a three decade analysis of aggregate health in high income countries, as reflected in mortality statistics, is new, interesting, and possible.

[2] The real price of food consumed away from home has been fairly stable because of slow technical change in this sector combined with increasing real wage rates.

Gerdtham and Jonsson (2000, p. 19) emphasize that prior empirical studies of national aggregate data on health outcomes and expenditures are noteworthy for having a weak theoretical basis. A recent paper by Loureiro and Nayga (2005) examines obesity rates in ten OECD countries over the 1990s and concludes that rising calories consumed and increased use of cars for transportation are major contributing factors. Their paper has the same shortcomings referenced by Gerdtham and Jonsson. The objectives of our paper are to formulate models of household health production and supply, to formulate an econometric specification of these models that is appropriate for aggregate data, and to prepare a panel of refined data for high income countries to which the models will be fitted and evaluated. We move beyond earlier studies by using our theoretical model to limit the focus to a set of most relevant variables. Also, we use our theoretical model and selective prior empirical results to form expected signs of regression coefficients in our econometric models. The available data are aggregate annual data for 18 high income industrialized countries over 1971-2001, and econometric models fitted to these data provide strong empirical support for the aggregate household health production and supply functions. In the final section of the paper, we present a summary of key results and develop some policy implications.

## II. Conceptual Model

The framework underpinning the empirical analysis of human health builds upon productive household models of health, as developed by Grossman (2000) and Rosenzweig and Schultz (1982), and of agricultural household models, as developed by Strauss (1986) and Huffman (1991). The household has a utility function

$$U = U(H, X, C, L; Z_1) , \tag{1}$$

where utility is derived from the current health status of household members ($H$), food (and drink) consumed ($X$), purchased consumption goods other than food ($C$), and leisure time of it adults ($L$).[3] In addition, a household's utility is determined by a vector of fixed observables, e.g., education and age of the adults in the household, local climate/weather and human congestion denoted by a vector $Z_1$.

The household's production of health uses multiple input technology:

$$H = H(X, I, L; Z_2, \mu), \tag{2}$$

where $H_X$ , $H_L <, =, > 0$, $H_I > 0$ and $I$ is a vector of purchased health inputs or health care, e.g., medical services and drugs.[4] In equation (2), additional input of healthful foods—lean meat and fish, fresh fruits and vegetables, whole grain breads—has a positive marginal product in the health production function. However, additional amounts of foods that are high in added sugar or caloric-sweeteners and salt, and high in fat, can be expected to have a positive marginal product at low levels of consumption but a negative marginal product at high levels of consumption. Likewise, if marginal input of leisure time is of a physically active nature, the marginal product of leisure is positive. Alternatively, if marginal leisure time is passive or sedentary—TV and video viewing, playing video games, web surfing—the marginal product of leisure may be negative. Hence, leisure time can have a positive or negative marginal product in the health production function.

Fixed factors in the health production function are denoted by $Z_2$, which represents observable attributes, including age and education of adults in the household, society's stock of medical and dietary knowledge and technologies, society's organization of the health care industry and public health practices, levels of air and water quality, and human congestion. The health

[3] See Huffman and Orazem (2005) for a three-period model of household behavior where the household produces human capital or health in the early periods and only consumes in the later period(s). However, this model is not well suited to aggregate data.

[4] Because of the aggregate nature of data to which the models will be fitted, the critical distinguishing uses for human time are work for pay and other time or leisure. Thus, our leisure variable includes time for recreational exercise, household and yard work and purer forms of leisure or pleasureful time (Juster and Stafford 1991, p. 477).

production function also includes $\mu$ , which is unobservable and represents other factors that affect the translation of inputs into health output, including genetic pre-disposition of adults for obesity and obesity-related diseases (Reed et al. 1997).

The household is assumed to allocate the fixed time endowment per period ($T$) of its adult members between hours of work for pay ($t_w$) and a second residual category, denoted as leisure hours $L$:

$$T = t_w + L. \tag{3}$$

In addition, the household's cash income constraint is

$$Wt_w + V = P_X X + P_I I + P_C C\,, \tag{4}$$

where $W$ is the wage rate per unit of time, $V$ is a household's nonlabor income, and $P_X, P_I$ and $P_C$ denote the price in the market for food ($X$), purchased health inputs ($I$), and other purchased consumption goods ($C$).

Let us confine the analysis to an interior solution of choices for the household, and then substitute equations (2) into (1) and (3) into (4). The household chooses $X, I, L,$ and $C$ by maximizing

$$\phi = U[H(X, I, L; Z_2, \mu), X, C, L; Z_1] + \lambda[WT + V - P_X X - P_I I - P_C C - WL]\,, \tag{5}$$

where $\lambda$ is the Lagrange multiplier representing the marginal utility of household full-income ($WT$ + V). The first-order conditions for an optimum are

$$U_H H_X + U_X = \lambda P_X \tag{6}$$

$$U_H H_I = \lambda P_I \tag{7}$$

$$U_H H_L + U_L = \lambda W \tag{8}$$

$$U_C = \lambda P_C \tag{9}$$

$$WT + V - P_X X - P_I I - P_C C - WL = 0\,, \quad \text{where} \tag{10}$$

$U_H = \partial U / \partial H$, $U_X = \partial U / \partial X$, $U_L = \partial U / \partial L$, $U_C = \partial U / \partial C$, $H_X = \partial H / \partial X$, $H_I = \partial H / \partial I$, and $H_L = \partial H / \partial L$.

Food input ($X$) affects utility directly and indirectly, and with over-nutrition, marginal food consumption may have a negative marginal product (effect) on health, even at an optimum. Purchased health inputs ($I$) are assumed to have no direct impact on utility. At an optimum, the household exhausts full-income (Becker 1965).

At an interior solution, jointly solving equations (6)–(10) yields implicit household demand functions for $X$, $I$, $L$ and $C$, denoted as follows:

$$\Omega = D_\Omega(P_X, P_I, P_C, W, V, Z_1, Z_2, \mu), \quad \Omega = X^*, I^*, L^*, C^* \tag{11}$$

Hence, the household's demand for inputs into health production depends on market prices of food, purchased health inputs and other purchased consumption goods $(P_X, P_I, P_C)$, the wage rate $(W)$ or opportunity cost of time, nonlabor income ($V$), fixed factors $(Z_1, Z_2)$, and other factors affecting health production $(\mu)$.[5] After substituting the demand functions for $X^*$, $I^*$ and $L^*$ from equation (11) into the health production function (2), we obtain the household's supply function for health:

$$H^* = S_H(P_X, P_I, P_C, W, V, Z_1, Z_2, \mu).^{6} \tag{12}$$

Since the household is supplying health to itself, one might call this function a demand function.

**III. Data, Econometric Models, and Other Issues**

Given the available data, the econometric specifications of the household health production and health supply functions are presented and discussed, and alternative estimation procedures are evaluated.

[5] $T$, the time endowment, is assumed to be a constant across households, and it is absorbed into the intercept of equation (11).

[6] This is analogous to the derivation of the supply function for farm output in an agricultural household model where household members do not work off-farm for a wage (see, for example, Huffman 1991, p. 96-97).

**The Data**

The key data set for this study is the international aggregate data contained in OECD (2005a), which reports annual data on age-adjusted death rates by cause, food consumption (total calories, total fat, sugar, and fruits and vegetables), expenditures on health (public and private), and share of the population in the labor force. Aggregate data on the consumer price index for all items, for food and for all items less food, and for compensation per employee are available from OECD (1993-2002). The data for the real gross domestic product (GDP in $USPPP) per adult equivalent are available from the *Penn World Tables* of Heston et al. (2002), and aggregate data on educational attainment for individuals who are 25 years of age and older are available in Barro and Lee. With these sources, a panel of refined data on health related variables for 18 high income countries over 1971-2001 is constructed.[7] The empirical measure of health that we choose is the age-adjusted death rate of the population due to cardiovascular diseases and diabetes mellitus per 100,000 people (OECD 2005a). These are diseases that have a major dietary component. In the remainder of this paper, the death rate due to cardiovascular diseases plus diabetes mellitus is denoted as "mortality."[8]

Table 1 provides the empirical definitions and summary statistics of the variables in our econometric models. Figure 1 plots mortality, our dependent variable ($H$), and shows that mortality differs by about 60 percent across countries, and that it has been declining exponentially by roughly 2.6 percent per year. One can easily visualize that after accounting for trend, there remains to be explained considerable variation within and across countries over time.

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[7] The following high income countries are included in our sample: Australia (AUS), Austria (AUT), Canada (CAN), Denmark (DNK), Finland (FIN), France (FRA), Ireland (IRL), Italy (ITA) Japan (JPN), the Netherlands (NLD), New Zealand (NZL), Norway (NOR), Portugal (PRT), Spain (ESP), Sweden (SWE), Switzerland (CHE), United Kingdom (GBR) and the United States (US). Germany and Greece were excluded because of major data problems.

[8] Mortality rates are estimated based on the crude number of deaths due to selected causes as provided in the World Health Organization Mortality Database (OECD 2005a, p. 24; Mathers et al. 2005). Mortality rates have been age-standardized to the 1980 OECD population structure to remove variations arising from differences in age structures across counties and over time within each country.

**The Econometric Models**

We complete the specification of the health production and supply functions. First, we hypothesize the following aggregate Cobb-Douglas type aggregate econometric household health production function, or technical relationship:

$$\ln(H_{it}) = \beta_1 + \beta_2 \ln(Calories_{it}) + \beta_3 \ln(Sugar_{it}) + \beta_4 \ln(Fat_{it}) + \beta_5 \ln(Fru\&Veg_{it}) + \beta_6 \ln(Health\_care_{it}) + \beta_7 \ln(LFPR_{it}) + \beta_8 Ed_{it} + \beta_9 Sm2_{it} + \beta_{10} Sm3_{it} + \beta_{11} Trend + \in_{it1}, \quad (13)$$

where the *i* subscript refers to a particular country, *t* subscript refers to a particular year, and *H* is mortality. *Calories* are the average daily input of total energy derived, from data on the amount of food available for human consumption and on nutrients availability. *Sugar* is the input of caloric sweeteners—refined sugar, corn sweeteners, honey, and edible sweet syrups—in kilograms per person per year. *Fat* is the input of fat in meat, fish and dairy products, and in salad and cooking oils, shortening, lard, edible tallow, and margarines in grams per person per day. *Fru&Veg* is the input of fresh and processed fruits and vegetables and nuts in kilograms per person per year. *Heath_care* is public and private health care input, measured as per capita real expenditures. *LFPR* is the labor force participation rate of the population 14 years of age and older, which is a proxy for adult leisure time. A higher labor force participation rate is an indicator suggesting less adult leisure time. *Ed* is the average number of years of schooling completed by individuals 25 years and older. *Sm*2 and *Sm*3 are dummy variables denoting countries that have a medium and high levels of socialized medicine, respectively, based on the public share of total health care expenditures.[9] $\beta_1$ - $\beta_{11}$ are parameters of the aggregate health production function to be estimated.

[9] Countries are grouped by total public and private health care expenditures that are in the public sector (OECD 2005a). The US has the smallest share and is the reference country or *Sm*1. The countries in the highly socialized medicine group *Sm*3 are Denmark, Finland, Japan, Norway, Sweden, and the United Kingdom. The other 13 high income countries are in *S*m2. These variables could also characterize how a society feels about the provision of public services to its citizens. Also, the older countries tend to have a different community and transportation design than the newer

Including *Trend* accomplishes three things. First, it de-trends the dependent variable; second, it de-trends all of the regressors; and third, it controls for other trend-dominated factors that might be correlated with ln(*H*) or the included regressors but that are otherwise excluded from the econometric model (Wooldridge 2002, p. 350-351). These left-out trend-dominated factors might include declining smoking rates, a growing stock of dietary, health, and medical information, and increasing availability of drugs and medical technologies.[10] Of course *Trend* may capture other factors, but including *Trend* will greatly clean up the interpretation of the estimated regression coefficients on the included variables in equation (13) [and equation (14)] below.

The random disturbance term $\varepsilon_{it1}$ represents the impact of idiosyncratic factors on the production of *H* or mortality.[11] With an intercept being included in equation (13) and our panel including almost all high income industrialized countries, it is reasonable to assume that $\varepsilon_{it1}$ has a zero mean. Unmeasured factors associated with mortality might be a source of correlation over time. Also, changes in these unmeasured factors and the general availability of medical care might cause the variance of the random disturbance for each country to be different over time. Because knowledge about human diet, medical treatments, and medical technology is an international public good that diffuses across boundaries of high income countries, it is a likely source of contemporaneously correlated of random disturbances across countries. Hence, $\varepsilon_{it1}$ is most likely autocorrelated over time and heteroskedastic, and contemporaneously correlated across countries.

In the health production function, the hypothesis is that marginal input of calories in high income countries increases the risk of obesity and obesity-related diseases and, eventually,

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countries, i.e., the Australia, Canada, New Zealand, and the US, and this may contribute to less obesity in the older countries (Frank et al. 2004).

[10] Recall that our empirical mortality variable, *H,* is already adjusted for the age composition of the population.

[11] Given that mortality has causes other than cardiovascular diseases and diabetes mellitus, measurement error exists in the dependent variable. This type of measurement error does not generally affect the size of the estimated regression coefficients, but it does reduce the $R^2$ for the fitted equation (Greene 2003, p. 84-85).

mortality due to these factors ($\beta_2 > 0$). Likewise, an increase of caloric-sweetener input is expected to increase mortality ($\beta_3 > 0$). For example, Ludwig (2002) has shown that high sugar and carbohydrate intake causes an abrupt and large fluctuation in the blood glycemic load, and that this produces physiological stress on the body and tends not to satisfy hunger. The impact of an increase in fat input on health is uncertain ($\beta_4 < = > 0$), because fat intake produces a modest impact on the blood glycemic load and also tends to satisfy hunger (Ludwig 2002). Hence, calories obtained from sugar and other caloric-sweeteners may be more harmful to human health over the long-term than those from fat. Fruits and vegetables contain vitamins, micronutrients and fiber, and generally have a low density of calories. Such intake also reduces the capacity of the stomach to hold other more energy-dense foods. We expect that an increase in the input of fruits and vegetables improves health and lower mortality ($\beta_5 < 0$).

We expect a marginal increase in health care input to reduce mortality ($\beta_6 < 0$). If leisure time at the margin is of a physically active nature, additional leisure will also reduce mortality. In contrast, if leisure time at the margin is sedentary, additional leisure will increase mortality. Hence, we have imprecise expectations about $\beta_7$ ($\beta_7 < = > 0$). We expect an increase in adult education to increase the efficiency of translating variable inputs in the health production function, or to reduce mortality ($\beta_8 < 0$). See Grossman (2000) and Huffman and Orazem (2005) for a summary of the productive impacts of education in households and small firms.

The organization of the health care system affects the equality of distribution of the health care input and access to medical services and treatments. The US health care system relies heavily upon private funding until individuals qualify for Medicare, a federally funded health insurance system for individuals who are aged 65 and older.[12] Most other countries have the public sector

[12] Medicare also covers some disabled individuals less than 65 years of age and people with end-stage kidney disease.

involved to a greater extent in health care—financing it through taxes or compulsory social insurance contributions. Countries that have socialized medicine promote equitable access to available medical services. Therefore, we expect $\beta_9$ and $\beta_{10} > 0$.

Next, we complete the specification of the household health supply function (12) by hypothesizing the following aggregate econometric behavioral relationship:

$$\ln(H_{it}) = \delta_1 + \delta_2 \ln(P_{Xit}) + \delta_3 \ln(P_{Cit}) + \delta_4 \ln(W_{it}) + \delta_5 \ln(V_{it}) + \delta_6 \ln(LFPR_{it}) + \delta_7 Ed_{it} + \delta_8 Sm2_{it} + \delta_9 Sm3_{it} + \delta_{10} Trend + \varepsilon_{it2}, \quad (14)$$

where $P_{X\,it}$ is the real price of food in country *i* and year *t*, $P_C$ is the real price of consumer goods less food, *W* is the real wage rate (a proxy) for the cost of leisure time, *V* is real GDP per adult equivalent (a proxy for nonlabor income given that the wage rate, *W*, is held constant). *H*, *LFPR, Ed, Sm*2*, Sm*3*,* and *Trend* are defined above. The random disturbance term $\varepsilon_{it2}$, just as for $\varepsilon_{it1}$, is assumed to have a zero mean, to be autocorrelated over time, and to be heteroskedastic and contemporaneously correlated across countries. $\delta_1$ - $\delta_{10}$ are parameters of the aggregate health supply function to be estimated.

In the OECD (2005a) and other available data, it is impossible to obtain a separate price index for medical services, $P_I$. Thus, no such variable is included. But the price of purchased health inputs is included in the price index for the price of non-food items, and in that form, it is included in the econometric analysis. This aggregation of effects does complicate the interpretation of the regression coefficient for $\ln(P_C)$.[13] Furthermore, the variables *Sm*2 and *Sm*3 can be viewed as proxy variables for individual and family incentives to engage in healthy lifestyles and for the availability of health care.

[13] The price of smoking materials/tobacco is also included in $P_C$, and in that sense, it is incorporated into the econometric model. In the OECD data, there is no way of extracting the price of smoking materials from the other components of this price index, but the inclusion of *Trend* in the regressions will control for any trend-dominated remaining effects of smoking.

The real price of food can affect the household supply function for health through its impact on the households' demand for food (and drink), purchased health care, and leisure. For high income countries, much empirical evidence exists that the demand for food (and drink) is price responsive; for some recent examples see Carpentier and Guyomard (2001), Dhar et al. (2003), and Huffman (2006). For example, Huffman (2006) uses US annual aggregate data over 1948-1996 for nine major household input groups, including own-housework and leisure, and obtains a compensated own-price elasticity of demand for food-at-home of -0.55. His estimates also show that food-at-home and leisure time are substitutes.[14] We do not have quantitative evidence of the impact of the price of food on the demand for purchased health inputs in high income countries, but we know that, on average, goods are substitutes. If the impact of a change in the real price of food on the supply of health is dominated by its own-price effect and, at the margin, added food represents over-nutrition, then a rise in the real price of food is expected to reduce food intake, the likelihood of obesity, and mortality ($\delta_2 < 0$). Increasing the price of non-food items could either increase or decrease the demand for food, purchased health inputs, and leisure. Hence, $P_C$ could have a positive, negative or zero impact on mortality ($\delta_3 < = > 0$).

Conceptually, the wage effect on the demand for leisure cannot be signed. However, Huffman (2006) provides evidence for the aggregate US that the demand for household work and leisure responds negatively to the wage or opportunity cost, and hence, the impact on leisure is negative. If, at the margin, added leisure is allocated to healthy lifestyles, then reduced leisure will decrease the supply of good health and increase the supply of mortality. In contrast, if marginal leisure is allocated to unhealthy lifestyles, then reduced leisure will increase the household supply of good health and decrease mortality. These impacts may be moderated by food-at-home and

[14] Food-away-from-home is included with purchased service of commercial laundry and dry cleaning and domestic services, which as a group has an own price elasticity of demand of -0.89. This input category and women's and men's housework are substitutes. The pure income elasticity of demand for food-at-home is 0.79 and for the service category including food-away-from-home is not significantly different from zero.

leisure being substitutes (Huffman 2006), so that when the wage increases, it increases the demand for food-at-home and away-from-home. We do not know the impact of the wage on the demand for purchased health inputs, and following the logic of our above discussion, we suggest that they are most likely substitutes. However, if added leisure goes into an unhealthy lifestyle and the own-price effect of leisure is the dominating factor of a wage change, then an increase in the wage decreases leisure and mortality ($\delta_4 < 0$).

Although food, purchased health input, and leisure can conceptually be normal or inferior goods, Huffman (2006) shows that food at home and leisure (and housework) are normal goods for US households. If marginal food consumption in high income countries is concentrated in unhealthful foods and drinks, such as sweetened drinks, salty snacks, and fast food, as it seems to be, then added food contributes to poorer health and raising the risk of obesity. We believe that non-labor income impacts the demand for purchased health input positively (Grossman 2000). Hence, we expect an increase in household non-labor income to increase the demand for all three of the inputs in the health production function. However, the impact on the supply of health or mortality is uncertain ($\delta_5 < = > 0$).

We expect that individuals who are working in the labor market burn more energy in calories per day than those who are not in the labor force. The labor force participation rate of women has been rising over the study period, except for Japan, and women remain largely responsible for planning and preparing meals eaten at home. As women work in labor market with higher frequency, this tends to increase the demand for eating out and for pre-packaged, processed, and take-out foods that tend to be detrimental to good health over the long run. Hence, a higher labor force participation rate results in opposing forces on the likelihood of obesity and mortality ($\delta_6 < = > 0$).

Adult education could have a positive or negative effect on the supply function of mortality.[15] We expect an individual's education to improve the quality of decisions on food, purchased health care, and leisure. For example, Grossman (2000) summarizes existing empirical evidence and suggests that a strongly positively association exists between own education and the supply of good health; applying this logic, we would expect a negative effect on obesity-related mortality. In the US, individuals who have more education tend to work more and have less time for leisure, but in other high income countries, those with more education work less (Blundell and MaCurdy 1999, p. 1574-1582). Also, individuals who have lower levels of education generally work in jobs requiring greater physical activity and, as their education increases, they switch to white collar or sedentary jobs. Hence, the effect of education on leisure, other things equal, is uncertain. Thus, the net effect of own education on households' supply of health or mortality is uncertain ($\delta_7 < = > 0$).

Socialized health care, as opposed to private health care, can be expected to impact not only the demand for purchased health inputs (Gerdtham and Jonsson 2000), but also the demand for food and leisure. When there is a high level of socialized medicine, individuals and their families face weaker incentives for healthy lifestyles than at modest or low levels of socialized medicine, and this increases the demand for health care. Society, however, bears the burden of paying for this health care. Socialized medicine at a moderate level is a blend of public and private health care. It provides stronger incentives for healthy lifestyle choices by individuals and families and some added options through private treatment. However, countries differ greatly in how decisions to adopt and pay for new health-related technologies are made, and this affects diffusion and use. With highly socialized

[15] From individual-level data, there is a large amount of empirical evidence that an individual's years of schooling increase his or her wage (Card 1999). Our data are aggregate, however, and they refer to the average years of schooling completed by all individuals who are 25 years and older, irrespective of whether they are in the labor force. Hence, the relationship is in aggregate data is likely to be much weaker. In our preferred specification of the econometric health supply equation, we control for the impact of *Ed* on earnings or compensation for labor market work.

medicine, single providers may limit consumers' choices of health care and treatments, and shortages may occur in the available supply of medical care, leading to queuing and rationing of the available supply and, perhaps, inequities in access to medical care (Marmot 1999; Gerdtham and Jonsson 2000; OECD 2004, p. 14-15). Hence, at an intermediate level, socialized medicine is likely to reduce mortality ($\delta_8 < 0$), but at a high level the outcome is uncertain ($\delta_9 < = > 0$).

**Other Issues**

Timing weights and estimators are now chosen.

*Timing weights*

The regressors in equations (13) and (14) do not have immediate impacts on mortality, e.g., we do not expect a single short episode of excessive food and drink consumption and sedentary lifestyle to have any impact on long-term health status or mortality. However, if this behavior persists over a substantial period of time, for example, a decade, we expect it not only to affect an individual's weight or risk of obesity, but also to elevate risks for cardiovascular diseases and diabetes mellitus, and eventually for related mortality. Hence, significant lags occur from unhealthy behavior to mortality, but with aggregate data we are constrained in how we pursue this dimension of the econometric modeling.

For continuous variables, even with national aggregate data we are quite limited on data going back in time. It is very difficult to push health-related data series back to 1960, and going further back in time is impossible for a large set of countries. Given available aggregate data, what type of timing weights or lag pattern should we use? Using a free-form lag pattern greatly increases the number of parameters to be estimated, which asks a lot of the data. It also leads to unsatisfactory results, in that regressors become highly correlated, creating a high degree of multicollinarity, the estimated coefficients of successive lagged values of a variable oscillate in sign, and only a few

estimated coefficients are significantly different from zero. The switching of signs is difficult to rationalize conceptually, and we need to dramatically reduce the multicollinearity problem.

Hence, let's consider two more-structured sets of timing weights that impose smoothness.[16] First, we consider exponentially declining timing weights. This set of weights puts most of the weight on early experience or behavior and the impact rapidly fades away, as shown in figure 2. Second, we consider weights of zero in $t$ and $t$+1, i.e., no long-term impact on health status due to short-term (un)healthy behavior, and then the impact is represented by trapezoidal shaped timing weights. For example, let the weights be positive and rise for two years, then reach a peak impact which is maintained for the next four years. Finally, the timing weights decline to zero over the next two years for a total lag pattern of 10 years, as shown in figure 2.[17] The latter set of timing weights captures the belief that the impact of the variables in the health production and supply functions occurs with a significant lag, but also that significant impacts then occur over several years before fading out (AIHW 2004, p. 2). This makes the second set of structured timing weights preferable to the first set.[18] Furthermore, we proceed under the hypothesis that it is reasonable to impose the same set of timing weights on all of the continuous regressors.

*Estimation*

We have suggested above that disturbances in equations (13) and (14) are most likely to be autocorrelated over time, and contemporaneously correlated and heteroskedastic across countries. Several strategies exist for fitting panel data models of this type. First, one could apply the feasible-

[16] Although we use deterministic priors for smoothing the timing weight pattern, our approach has similarities to the smoothness priors applied by Bayesians on lag patterns of variables (Kitagawa and Gersch 1996; Geweke and Kean 2005).

[17] We are limited in the length of lag that we can consider, due to data unavailability. This is the main reason for the 10- year limit.

[18] Furthermore, a variable created using trapezoidal weights can be viewed as a proxy variable for the "true" variable (Greene 2003, p. 86-90). Any variable constructed using similarly shaped timing weights over a 10 year period will be highly correlated and yield similar regression results. Also, see Huffman and Evenson (2006) for the use of trapezoidal weights in another application of lagged impacts of variables.

generalized least-squares (FGLS) estimator where first-round OLS residuals are used to estimate values of ρ, which is the first-order autoregressive coefficient of an (AR(1)) stochastic process, a variance for each country, and the contemporaneous correlation of disturbances across pairs of countries. This is a procedure developed by Parks (1967). However, Parks' estimator has good statistical properties only if the number of time periods (*T*) is much larger than the number of cross-sectional observations (*N*). Also, Beck and Katz (1995) have shown that the full FGLS variance-covariance estimates are typically unacceptably optimistic when used in panels of modest size and length. Second, the Prais-Winsten estimator (Greene 2003 p. 325-326) can be applied to estimate the regression coefficients in equation (13 and 14) and an autocorrelation coefficient (AR(1)).[19] One can then adjust the standard errors for contemporaneous correlation and heteroscedasticity across countries, i.e., panel corrected standard errors (PCSE). This is an alternative to FGLS. Third, White (1980) and MacKinnon and White (1985) suggest another strategy where regression parameters are estimated by OLS and standard errors are corrected for a general, rather than a specific, form of heteroscedasticity. This latter methodology was extended by Newey and West (1987) to a general form of standard error correction for autocorrelation or combined general heteroscedasticity and autocorrelation.[20]

The Newey-West standard errors ignore available information that permits a major simplification of the variance-covariance matrix of the disturbances. After weighing alternative strategies, we choose to use the Prais-Winsten estimator of regression coefficients with one AR(1) coefficient and then to correct the standard errors and *z*-values for heteroscedasticity and

[19] The program permits both one autocorrelation coefficient per country and one across all countries. Beck and Katz (1995, pp. 121) make a case against estimating panel-specific AR(1) parameters and for a single AR(1) parameter across all countries.

[20] Our empirical measure of ln(*H*) is de-trended and naturally bounded. Hence, it cannot explode to plus or minus infinity over time. Inputs are on a per person basis, and prices and income are in real terms and all are de-trended, so they are most likely stationary. These considerations moderate concerns about unit roots or non-stationarity of the series. Moreover, the unit root test developed for panel data by Im et al. (2003) has low power for our panel size of 18 and our time period of 30 years.

contemporaneous correlation across countries.[21] The estimator for the regression coefficients is consistent and the estimate of the variance-covariance matrix of the parameters is asymptotically efficient under the assumed covariance structure of the disturbances.

**IV. Empirical Results**

Estimates of the household health production function (13) fitted to refined data for 18 countries over 1971-2001 are reported in table 2 and estimates of the household health supply function (14) are reported in table 3. In table 2, model (1) is fitted by OLS to provide a benchmark for comparison with model (2), which is estimated using the Prais-Winsten estimator.[22] In both estimation methods, we report *z*-values corrected for heteroscedasticity and contemporaneous correlation of disturbances across countries, i.e., panel corrected standard errors.

In table 2, strong econometric evidence exists of an aggregate household health production function for mortality. The signs of the estimated coefficients of all of the regressors are the same in the OLS and Prais-Winsten estimates, but we prefer the latter estimates because they control for autocorrelation. An increase in input of *Calories* and *Sugar* increases mortality, and the effects are significantly positive at the 1 percent level. A 10 percent increase in *Calories* increases mortality by 7 percent and a 10 percent increase in *Sugar*, other things equal, increases mortality by 1.7 percent. However, a higher input of *Fat*, other things equal (including total calories), does not significantly affect the production of health as reflected in mortality. Hence, *Fat*, in contrast to *Sugar*, is not closely linked to mortality. On the other hand, higher *Fru&Veg* decreases mortality, and the effect is statistically significant at the 1 percent level. A 10 percent increase in the *Fru&Veg* reduces mortality by 1.4 percent.

[21] This estimation is implemented in STATA 8.2 using the Prais-Winsten estimator with subroutines ar1 and xtpcse (STATA 2005, pp. 226-235). Hence, the xtpcse subroutine constructs the PCSEs.

[22] We considered two models with country random and fixed effects, but rejected both of them for the following reasons. Random country effects cannot be justified because of their almost certain correlation with the regressors. However, in highly aggregated data over time, the use of country fixed-effects leads to over-fitting, and country dummies frequently account for too much. See Wooldridge (2002, pp. 247-279) for a discussion of these issues.

An increase in *Health_care* does not significantly reduce mortality, holding constant total calories and with the composition of the diet fixed. This is contrary to medical profession beliefs about the contribution of health care to mortality. The estimated coefficient of ln(*LFPR*) is negative, suggesting that added leisure reduces mortality. This result supports the hypothesis that marginal leisure time is sedentary and worse for the production of good health than work in the labor market. The coefficient, however, is not significantly different from zero at the 5 percent level. An increase in adult education tends to improve human health as reflected in a reduction in mortality. Although the direction of the impact is as expected, the effect is not significantly negative at the 5 percent level.

Contrary to the health care input, the organization of the health care system does affect the production of mortality. Countries that have a moderate level of socialized medicine have a 16 percent lower mortality than the US, and those with highly socialized medicine a 15 percent lower mortality. The negative regression coefficient for *Trend* is significantly different from zero and controls for a linear trend in the dependent variable, the regressors, and for effects of trend-dominated excluded factors that affect mortality.

In summary, the results from fitting (13) are consistent with expectations, except for the health care input. We can also perform a hypothesis test that all of the regression coefficients in the health production function are jointly equal to zero, except for the coefficient of *Trend* and the intercept/constant term. The sample value of the Wald chi-squared statistic from this test is 34.6 in model (2), table 2, and the tabled value of the chi-squared statistic for 9 degrees of freedom at the 1 percent significance level is 21.7. Hence, we reject the null hypothesis of no explanatory power of our health production function at the 1 percent significance level. In fitting the health production function, we have shown that an empirical relationship exists between mortality due to cardiovascular diseases and diabetes mellitus and diet—total calories and composition of the diet—

after controlling for a modest set of other factors. Since excess calories consumed on a long-term basis are a major risk factor for obesity and obesity is one of the major risk factors for cardiovascular diseases and diabetes mellitus, one might conclude that we have successfully linked food consumption to obesity-related mortality.

Next, we examine the estimates of the aggregate household health supply function (14), which is the key behavioral relationship. In table 3, we report three sets of estimates; an OLS benchmark and two Prais-Winsten estimates. The three reported models in table 3 have associated with them *z*-values that are constructed from panel-corrected standard errors due to heteroscedasticity and contemporaneous correlation across countries.

In model (2), the estimated coefficients for $\ln(P_X)$ and $\ln(W)$ seem small and the associated *z*-values are very small.[23] This could be an indication of measurement error. Any measurement error in a regressor is serious if it is correlated with the disturbance term $\varepsilon_2$ because it causes the estimated regression coefficients to be statistically inconsistent and attenuated toward zero (Greene 2003, p. 74-86). One treatment for this problem is to instrument $\ln(P_X)$ and $\ln(W)$. If there is no attenuation, the use of the instrumental variable estimator (IV) will not significantly change the size of the estimated regression coefficients and the significance levels (Hausman 1978). However, if the estimated coefficients under the IV estimation deviate significantly from those without the instrument, we accept the hypothesis of significant measurement error.

The instrument for the real price of food (the real wage rate) is a prediction obtained from an OLS regression of $\ln(P_X)$ and $\ln(W)$ on $\ln(P_C)$, $\ln(V)$, $Ed$, $\ln(LFPR)$, a set of 17 country fixed effects, a linear trend and a constant term (see Appendix Table A.1).[24] In model (3), we include the

[23] The quasi-first differencing that occurs in estimating a first-order autoregressive process AR(1) elevates the relative importance of measurement error to systematic variation in regressors (Wooldridge 2002), and this is one explanation for the reduction in the size of estimated coefficients going from model (1) to model (2), table 3.

[24] Predictions from the OLS real price of food and wage equations have a variance that is 29 percent and 188 percent smaller, respectively, than the corresponding actual values. These computations are in ln units.

instrumented rather than the actual value for the real price of food and the real wage. In this model, the estimated coefficient of ln($P_X$) and ln($W$) are of the expected sign, several times larger than in model (2) and significantly negative at the 1 percent level. Because of its superior performance, model (3) is our preferred model. It provides strong econometric evidence of an aggregate household supply function for health as reflected in mortality.

Continuing with model (3), table 3, the estimated coefficient for the price of food implies that a 10 percent increase in the price of food over the long run decreases mortality by 1.1 percent and increases the households' supply of health. The regression coefficient for the real price of nonfood items is also significantly negative, and the impact on mortality, measured as an elasticity, is -1.85. A higher price of leisure, represented by the real wage rate, also significantly reduces mortality at the 1 percent level, and the magnitude of the impact on mortality is an elasticity of -0.04. Summing up, given that other studies have shown that households' demand for food is own-price responsive, an increase in the price of food implies a reduction in the quantity of food demanded. With the other prices (and income) held constant, this implies a decrease in calories consumed but no significant change in the hours of labor market work. Hence, an increase in the price of food reduces tendencies for obesity, and this is consistent with the decline in obesity-related mortality that we observe in our results.

The impact of nonlabor income on the household supply of mortality is negative and statistically significant at the 1 percent level. A 10 percent increase in $V$ causes a 2.4 percent decline in mortality. A higher aggregate labor force participation rate (*LFPR*) significantly increases mortality with an elasticity of 0.27. This result is consistent with the dominant effect of a rising labor force participation rate being the cause of increased consumption of food-away-from home, which is frequently unhealthful food. An increase in *Ed* has a direct positive impact on mortality

that is significantly different from zero.[25] This result contradicts Grossman (2000), but still is plausible, in that the dominant effect may be the increasingly sedentary lifestyle that accompanies rising levels of adult education.

Countries that have a moderate level of socialized medicine (*Sm*2) have an aggregate supply of health as reflected in mortality that is significantly lower (at the 1 percent level) than the US, by 8.9 percent. However, contrary to the results of the household health production function, countries that have highly socialized medical systems (*Sm*3) have a supply of mortality that is not significantly different from zero, which implies that countries that have highly socialized medical care perform no better than the US. This last result supports the belief that too high a level of socialized health care may be less effective for reducing mortality. Moreover, we do not see any contradiction between these results and those obtained for the aggregate health production function. The health production function is a technical relationship between a health outcome and health inputs, but the health supply function better summarizes optimizing behaviors of households, given the economic environment in which they find themselves.

Just as with equation (13), the estimated coefficient of *Trend* is negative and significantly different from zero. Growing urbanization is an additional excluded factor that *Trend* controls for in this equation. We also perform a test of the hypothesis that all of the coefficients in the health supply function are jointly equal to zero, except for the coefficient of *Trend* and the intercept/constant term. The sample value of the Wald chi-squared statistic from this test is 153.6 in model (3), table 3, and the tabled value of the chi-squared statistic for 8 degrees of freedom at the 1 percent significance level is 20. Hence, we reject the null hypothesis of no explanatory power for the household health supply function at the 1 percent significance level, and conclude that our health supply equation has major explanatory power. Moreover, there is no glaring indication in our

[25] Recall that *Ed* is used in instrumenting ln(*W*), but it is not statistically significant.

econometric results that imposing the same weighting pattern in constructing regressors is inappropriate.

## V. Conclusions

Strong econometric evidence is reported for both an aggregate household human health production function and an aggregate household health supply function for high income countries over the past three decades. In both models, the dependent variable is the age-adjusted mortality rate from cardiovascular diseases and diabetes mellitus per 100,000 people, which might be referenced as obesity-related mortality. Using data for 18 high income countries over 1971-2001, we found that decreased input of calories and sugar and increased input of fruits and vegetables reduces mortality due to cardiovascular diseases and diabetes mellitus. Input of fat, holding total calories constant, has no significant effect on this form of mortality. Hence, marginal input of sugar is more harmful to the production of good health than fat. The result is consistent with fat consumption suppressing hunger and resulting in lower physiological stress than high sugar consumption. These results provide strong empirical evidence that input of total calories and the composition of the diet have long-term impacts on mortality.

We found a surprising number of significant results in the aggregate household health supply function. First, countries that have a high real price of food have lower rates of obesity-related mortality. Hence, high income countries that have pursued cheap food policies have increased the likelihood of higher obesity-related mortality rates than other high income countries. Second, countries that have higher real wage rates have lower mortality, which is consistent with increased labor market work of those in the labor force, including commuting, using up more calories than alternative daily activities and this effect dominating the other impacts of the wage on the demand for food and leisure. This increases the supply of good health. Third, an increase in nonlabor income reduces obesity-related mortality. Fourth, higher labor force participation rates,

holding the real wage constant, increase mortality. This result is consistent with rising labor force participation rates in high income countries due to the increased labor market participation of women. Since women are the primary at-home meal preparers, this leads to less time for women's meal preparation and to less healthful diets for themselves and their family members. Fifth, an increase in the schooling of adults directly increases mortality, which is inconsistent with earlier studies, but consistent with our expectations. Adults who have more schooling are more likely to have sedentary lifestyles than individuals at lower education levels.

Countries that have an intermediate level of socialized medicine have lower obesity-related mortality rates than the US, which has the most privately-oriented health care system in our study. In contrast, we find that countries that have high levels of socialized medicine have obesity-related mortality rates that are not significantly different from the US and that are significantly higher than for countries with an intermediate level. An intermediate level of socialized medicine seems to provide a better set of individual incentives for healthy lifestyles, but also to exert a more positive influence on access and distribution of medical care than highly socialized medicine or the US plan. With highly socialized medicine, individual and family incentives for a healthy lifestyle are weaker. Also, medical care is frequently organized around a single provider and, thus, may be slow in adopting new innovations in treatments and may also involve queuing for the available supply of health care. Hence, countries that have a moderate level of socialized medicine, for example, France, the Netherlands, and Canada, may have the best healthcare systems for dealing with cardiovascular diseases and diabetes.

Our regression results were obtained while controlling for a time trend and autocorrelation. The estimated coefficient of trend in our econometric models controls for a number of other factors that are correlated with time, including growing urbanization and declining smoking rates, in the

sample countries. Most important is the fact that inclusion of trend and autocorrelation in our econometric models adds to the confidence that we have in our empirical results.

Some policy implications can be developed from these results. First, the cheap food policies of high income countries are bad for human health, as reflected in mortality due to cardiovascular diseases and diabetes mellitus that have has a major food, and frequently, an obesity component. This could be due to subsidized domestic food prices or subsidized agricultural output indirectly, through input markets or technical change. In particular, a cheap food policy becomes a force reducing the gains from reduced smoking and improved treatments of high cholesterols levels and hypertension. One might ask: "What decline in the real price of food would offset the trend-dominated reductions in obesity-related diseases, as reflected in the 2.8 percent negative trend in the health supply function?" It seems that it would take about a 26 percent compound rate of decline in the real price of food, and this decline is unlikely in most high income countries. A possible exception, however, is Switzerland and perhaps Japan (OECD 2005b), which have very high farm price supports relative to other high income countries, and with which WTO negotiations are underway to reduce the supports. Second, although the medical profession tends to be a strong proponent of a high level of socialized health care, our results suggest that an intermediate level of socialized health care is better for dealing with cardiovascular diseases and diabetes. A high level of socialized health care is no better than the US system. A rationale is that an intermediate level of socialized health care provides a balance of incentives for good individual and family lifestyles, as well as equitable access to health care.

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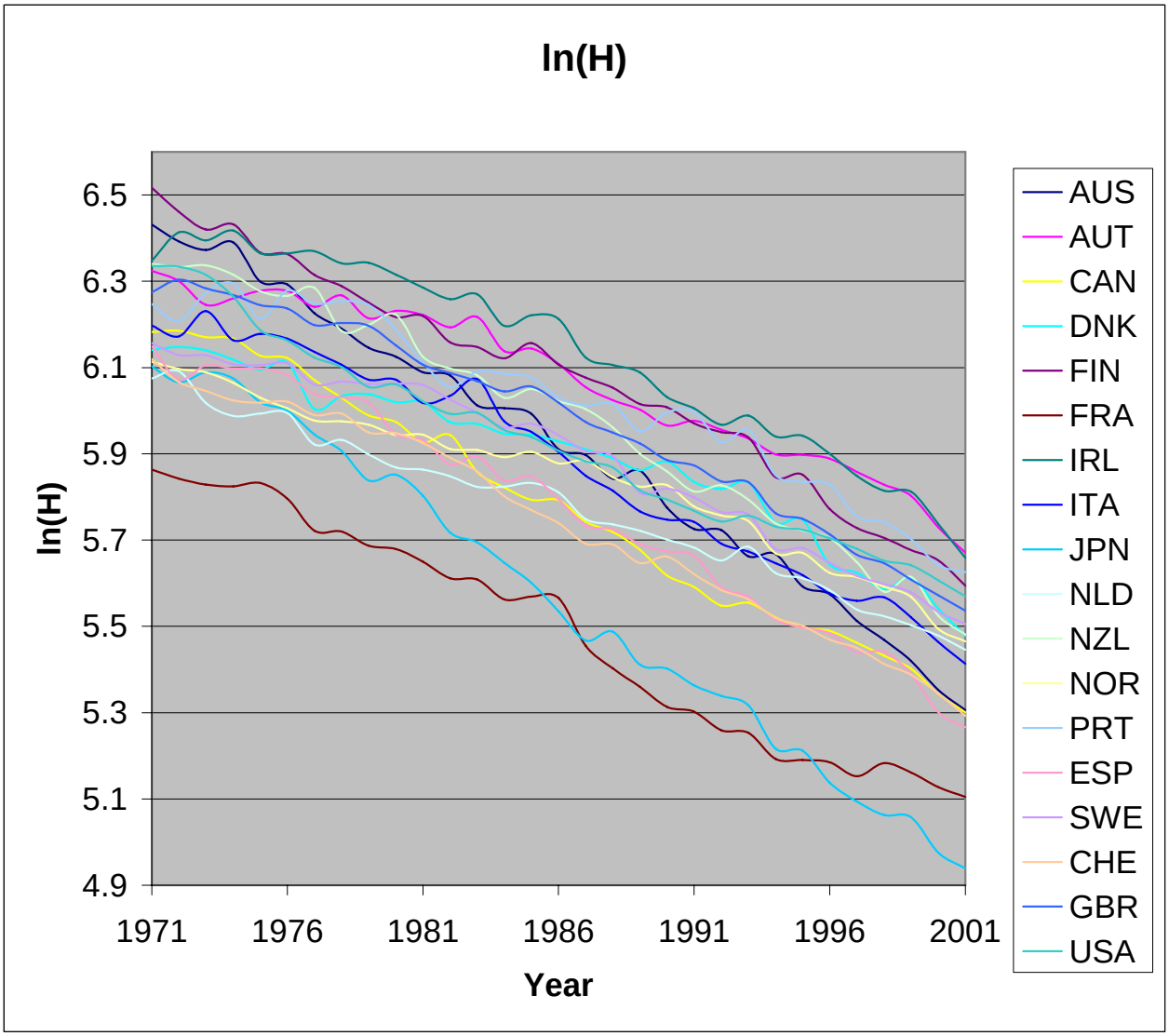
**Figure 1. Mortality Rates Due to Cardiovascular Diseases and Diabetes Mellitus: 18 High Income Countries, 1971-2001**




Source: OECD 2005a.

**Figure 2. Timing Weights for Exponentially Declining and Trapezoidal Distributions**

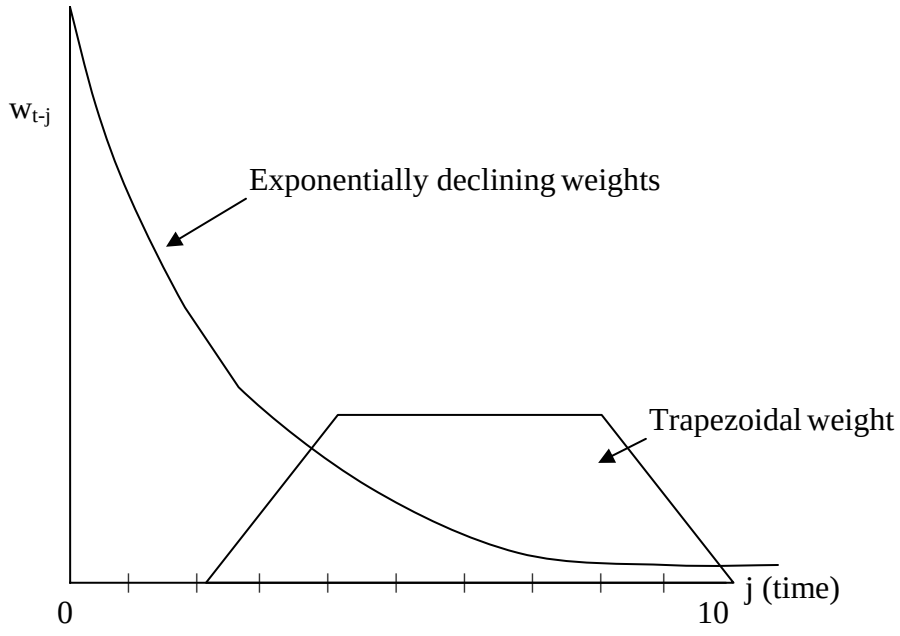


**Table 1. Variable Names, Definitions, Means and Standard Deviations**

| Symbol | Mean (Sd) | Definitions |
|---|---|---|
| ln(*H*) | 5.872 (0.295) | Age-standardized mortality due to cardiovascular diseases and diabetes mellitus per 100,000 people (OECD 2005a) |
| ln(*Calories*) | 8.061 (0.703) | Average daily intake of total calories (OECD 2005a) |
| ln(*Sugar*) | 3.574 (0.227) | Average intake of sugar and caloric-sweeteners, kilograms per capita per year (OECD 2005a) |
| ln(*Fat*) | 4.814 (0.205) | Average daily intake of total fat, grams per person (OECD 2005a) |
| ln(*Fru&Veg*) | 5.165 (0.288) | Average intake of fruits, vegetables and nuts, kilograms per capita per year (OECD 2005a) |
| ln(*Health_care*) | 2.405 (0.530) | Total (public and private) expenditures on health ($USPPP) per person (OECD 2005a) |
| ln($P_X$) | 0.053 (0.230) | The price index for food and drink divided by the consumer price index (OECD 1993-2002) |
| ln($P_C$) | -0.011 (0.500) | The price index for non food and drink divided by the consumer price index (OECD 1993-2002) |
| ln(*W*) | 6.529 (6.308) | Annual compensation per worker divided by the consumer price index (OECD 1993-2002) |
| ln(*V*) | 9.757 (0.296) | Gross domestic product ($USPPP) per equivalent adult (Heston et al. 2002) |
| ln(*LFPR*) | 3.796 (0.124) | The labor force participation rate of the population 14 years and older (OECD 2005a) |
| *Ed* | 8.10 (2.068) | Average years of schooling completed for adults 25 years of age and older (Barro and Lee ) |
| *Sm*2 | 0.661 (0.487) | Dummy variable taking a value of 1 if a country has a modest level of socialized medicine (Australia, Austria, Canada, France, Ireland, Italy, Netherlands, Portugal, Spain, Switzerland and New Zealand) and zero otherwise |
| *Sm*3 | 0.333 (0.472) | Dummy variable taking a value of 1 if a country has a high level of socialized medicine (Denmark, Finland, Japan, Norway, Sweden and United Kingdom) and zero otherwise |
| *Trend* | 1986 | Linear time trend |

**Table 2. Panel Estimates of the Aggregate Household Production Function for Health in High Income Countries, 1971-2001**[a/] (absolute *z*-values in parentheses, $N$ = 18x31 or 558).

| | ln(*H*) | |
|---|---|---|
| | OLS with Panel-Corrected Standard Errors | Prais-Winsten Estimator with Panel-Corrected Standard Errors[b/] |
| Regressors | (1) | (2) |
| ln(*Calories*) | 0.804 | 0.699 |
| | (8.05) | (3.06) |
| ln(S*ugar*) | 0.397 | 0.172 |
| | (7.51) | (2.13) |
| ln(*Fat*) | -0.159 | 0.168 |
| | (2.88) | (1.45) |
| ln(*Fru&Veg*) | -0.157 | -0.143 |
| | (4.45) | (2.38) |
| ln(*Health_care*) | -0.222 | -0.005 |
| | (8.22) | (0.08) |
| ln(*LFPR*) | -0.052 | -0.185 |
| | (1.06) | (1.48) |
| *Ed* | -0.006 | -0.010 |
| | (1.25) | (0.89) |
| *Sm*2 | -0.258 | -0.156 |
| | (8.34) | (3.03) |
| *Sm*3 | -0.245 | -0.150 |
| | (8.68) | (2.56) |
| *Trend* | -0.014 | -0.023 |
| | (11.01) | (8.61) |
| *Constant* | 28.357 | 48.643 |
| | (10.43) | (8.66) |
| $R^2$ | 0.785 | 0.991 |

[a/] *H* is the age-standardized death rate due to cardiovascular diseases and diabetes mellitus.
[b/] In model (2), the value of the first-order autocorrelation coefficient used in this computation was 0.9370 with a standard error of 0.106, and *z*-values are corrected for panel heteroscedasticity across countries and contemporaneous correlations across pairs of countries.

**Table 3. Panel Estimates of the Aggregate Household Supply Function for Health in High Income Countries, 1971-2001**[a/] (absolute *z*-values in parentheses, $N = 18x31$ or 558)

| Regressors | OLS with Panel-Corrected Standard Errors (1) | Prais-Winsten Estimator with Panel-Corrected Standard Errors[b/] (2) | IV Prais-Winsten Estimator with Panel-Corrected Standard Errors[c/] (3) |
|---|---|---|---|
| $\ln(P_X)$ | -0.073 | 0.009 | -0.108 |
| | (2.36) | (0.19) | (2.52) |
| $\ln(P_C)$ | -1.423 | -0.426 | -1.846 |
| | (8.67) | (2.12) | (9.04) |
| $\ln(W)$ | -0.005 | -0.001 | -0.039 |
| | (4.67) | (2.64) | (10.71) |
| $\ln(V)$ | -0.447 | -0.047 | -0.248 |
| | (15.68) | (0.52) | (3.24) |
| $\ln(LFPR)$ | -0.070 | -0.156 | 0.273 |
| | (1.04) | (1.17) | (2.34) |
| *Ed* | 0.053 | 0.006 | 0.025 |
| | (13.95) | (0.51) | (2.50) |
| *Sm*2 | -0.080 | -1.112 | -0.089 |
| | (6.18) | (2.33) | (2.75) |
| *Sm*3 | -0.046 | -0.086 | -0.060 |
| | (3.14) | (1.68) | (1.54) |
| *Trend* | -0.021 | -0.025 | -0.028 |
| | (40.97) | (11.44) | (15.20) |
| *Constant* | 52.802 | 57.053 | 62.372 |
| | (57.24) | (14.26) | (18.78) |
| $R^2$ | 0.780 | 0.990 | 0.993 |

[a/] *H* is the age-standardized death rate due to cardiovascular diseases and diabetes mellitus.
[b/] The value of the first-order autocorrelation coefficient used in this computation was 0.942 with a standard error of 0.111 in model 2 and 0.910 with a standard error of 0.104 in model 3, and *z*-values are corrected for panel heteroscedasticity across countries and contemporaneous correlations across pairs of countries.
c/ In models 3 and 4 the instrument for the price of food and the wage is obtained from a regression reported in Appendix Table A.1.

**Appendix Table A.1. OLS Estimates of the Real Food Price and Real Wage Equations for 18 High Income Countries, 1971-2001** (absolute *t*-values in parentheses, $N = 18 \times 31$ or 558).[a/]

| | $\ln(P_X)$ | $\ln(W)$ |
|---|---|---|
| $\ln(P_C)$ | 0.209 | -42.482 |
| | (1.21) | (4.99) |
| $\ln(V)$ | 0.288 | -6.483 |
| | (4.21) | (1.88) |
| *Ed* | -0.045 | -0.250 |
| | (2.64) | (0.29) |
| $\ln(LFPR)$ | 0.145 | 19.352 |
| | (1.22) | (3.23) |
| D(*AUS*) = 1[a/] | -0.035 | -0.935 |
| | (0.96) | (0.50) |
| D(*AUT*) = 1 | -0.801 | -3.360 |
| | (11.70) | (0.97) |
| D(*CAN*) = 1 | 0.014 | 0.783 |
| | (0.34) | (0.42) |
| D(*DNK*) = 1 | -0.030 | -0.655 |
| | (0.71) | (0.31) |
| D(*FIN*) = 1 | 0.139 | -7.369 |
| | (2.12) | (2.24) |
| D(*FRA*) = 1 | -0.076 | 1.972 |
| | (0.95) | (0.49) |
| D(*IRL*) = 1 | 0.130 | -2.178 |
| | (1.48) | (0.49) |
| D(*ITA*) = 1 | -0.024 | -2.320 |
| | (0.23) | (0.45) |
| D(*JPN*) = 1 | -0.059 | 0.265 |
| | (0.98) | (0.09) |
| D(*NLD*) = 1 | 0.136 | -1.955 |
| | (2.00) | (0.57) |
| D(*NZL*) = 1 | 0.113 | 0.713 |
| | (2.86) | (0.36) |
| D(*NOR*) = 1 | -0.079 | -0.998 |
| | (1.28) | (0.32) |
| D(*PRT*) = 1 | -0.021 | -12.075 |
| | (0.14) | (1.65) |
| *D*(*ESP*) = 1 | -0.030 | 1.751 |
| | (0.27) | (0.31) |
| D(*SWE*) = 1 | 0.104 | -1.814 |
| | (2.20) | (0.76) |
| D(*CHE*) = 1 | -0.071 | -3.509 |
| | (1.58) | (1.54) |
| D(*GBR*) = 1 | 0.084 | -4.651 |
| | (1.39) | (1.53) |
| *Trend* | -0.003 | -0.026 |
| | (1.22) | (0.23) |
| Constant | 2.330 | 50.801 |
| $R^2$ | 0.751 | 0.154 |

[a/] Country dummy variables. See footnote 8 for the country definitions. The US is the reference country.