The Economics of Obesity-Related Mortality Among High Income Countries

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Contributed paper prepared for presentation at the International Association of Agricultural Economists Conference, Gold Coast, Australia, August 12-18, 2006

April 25, 2006

 Yanni Chen provided valuable research assistance. Funding was received from a USDA-ERS Cooperative Research Agreement.

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I. Introduction

The high and rapidly rising adult obesity rates in the United States (U.S.), United Kingdom (U.K.), Australia and New Zealand are associated with major health risks, including cardiovascular disease, diabetes, stroke, and some forms of cancer; large health care costs; and premature deaths annually (OECD 2004). For example, the death rate from diabetes mellitus has been rising in the U.S. and across developed countries. In contrast, the death rates from circulator diseases have a strong negative trend, but rising obesity rates almost certainly have slowed this trend (OECD 2005).

Rising obesity rates have not been stopped by past policies, e.g., food and exercise guidelines, education programs or weight loss programs, and in developed countries, at least one-half of the cost of obesity is borne by society rather than the individual (US DHHS and USDA; Finkelstein et al. 2003; US DHHS). Although obesity rates in Japan, Norway, Switzerland, France, Austria, Sweden, Netherlands, Denmark, Finland, Spain, Italy, Portugal, Ireland and Canada are much lower than for the U.S., U.K., Australia and New Zealand, and rising less rapidly, the upward trend in obesity rates is ubiquitous in high income countries (OECD 2004). Loureiro and Nayga (2005) present an empirical examination of obesity rates in 10 OECD countries over the 1990s and conclude that rising calorie intake and usage of cars for transportation are major contributing factors.

Rising obesity rates are the result of a long-term human energy imbalance--larger energy intake than energy expended on work and basal metabolism. Over the past 30 years, developed countries have undergone major changes that contribute to a human energy imbalance. First, rapid improvement in the technology of household production and smaller family sizes have reduced the amount of work to be done in household production (Huffman 2006). Second, new market goods,

including baked and processed foods, sweetened drinks, and sweet and salty snacks, are substituted for home produced goods (Cutler et al. 2003; Kuchler et al. 2005). Availability of unhealthy foods has increased, and much leisure has become sedentary, e.g., TV viewing, web surfing and computer games. Third, the relative price of food but especially of unhealthful food has fallen and consumption has increased (Huffman and Evenson 2005; Huffman 2006). Fourth, mechanization and automation of market work and the shift of workers from agriculture and manufacturing to service industries has reduced the energy requirement of work (Lakdawalla and Philipson 2002). Finally, an improvement in transportation has reduced energy intensity of commuting especially in the U.S., Canada, Australia, and New Zealand. Hence, adults and children in developed countries are increasingly affected by over-nutrition associated with an unhealthy lifestyle.

For developed countries, obesity data are generally available only since about 1990 and then infrequently (OECD 2005). This severely limits long-term analysis. Obesity, however, translates with a lag into higher mortality rates for certain causes, e.g., diabetes mellitus and diseases of the circulatory system, and mortality. Data on obesity-related mortality for the past 30 years are reported in *OECD Health Data 2005*. The *objective* of this paper is to establish the econometric underpinning of an aggregate household health production function for obesity-related mortality[1] and an aggregate household health supply function using data for18 high income countries over the past three decades. Our fitted aggregate household health production function shows that mortality is related significantly with a lag to diet, socialized medicine, and trend-dominated factors of medical knowledge and technology. We also present first estimate of an aggregate household supply function for obesity-related mortality in high income countries and show that low food prices increase obesity related mortality.

[1] From this point forward, we will refer to obesity-related mortality (rates) when we say mortality (rates).

II. Conceptual Household Model

The framework underpinning the empirical analysis of human health builds upon productive household models of health by Grossman (2000) and Rosenzweig and Schultz (1982). The household has a utility function

$$U = U(H, X, C, L; Z_1) \quad (1)$$

where H denotes current health status of household members, X denotes food (and drink) consumed, C denotes purchased goods other than health inputs or food, and L denotes leisure. Health inputs will increase utility through increased health status as described by equation (2) below. Utility generating social interactions frequently are associated with eating--meals, parties, coffee breaks. These activities might be associated with leisure time activities. In addition, utility is determined by a vector Z_1 of fixed observables, e.g., education, age, local climate/weather and congestion.

The household has a health production function

$$H = h(X, I, L; Z_2, \mu) \quad (2)$$

where I denotes a vector of purchased health inputs or medical care, e.g., medical services and drugs. Food X also enters the human health production function and the human time component of health production is leisure time L.[2] Impacts of these inputs on health may not occur immediately. We expect each input (X, I, L) to have positive marginal products over some range of use. Increased food consumption at low levels can be expected to increase health status, but over a high range of consumption, increased food consumption leads to obesity and poor health. At low levels of consumption, leisure time activities increase health status, but over some high range, increased leisure time leads to sedentary activities which contribute to obesity and poor health. Z_2 is observable and includes education, age, stock of medical and dietary knowledge and technologies,

[2] As a modeling strategy, we keep the number of different types of human time small, but sedentary leisure can contribute to poor health outcomes.

organization of the health care industry, and public health practices, air and water quality, and human congestion. The health production function also includes μ which is unobservable and might relate to genetic pre-disposition for good health and other things affecting health production.

The household allocates a fixed time endowment (T) to leisure and market work (t_w)

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

In addition, the household's cash income constraint is

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

where W is the wage rate per unit 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 goods *(C)*.

Let us confine our analysis to an interior solution of the household model and substitute equations (2) into (1) and (3) into (4). The household chooses *X, I, L,* and *C* (and *t*) 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 λ is the LaGrange multiplier representing the marginal utility of household full-income $\left(F = WT + V\right)$. The first-order conditions for an optimum are

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

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

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

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

$$WT + V = P_X X + P_I I + P_C C + WL \tag{10}$$

where $U_H = \partial U / \partial H, U_C = \partial U / \partial C, h_X = \partial h / \partial X, h_I = \partial h / \partial I, and\ h_L = \partial h / \partial L.$ Food input *(X)* affects utility indirectly through health production, providing energy, protein, vitamins and

mineral, and directly. With over-nutrition, total calories and sugar intake may have a negative marginal product on health even at an optimum. Purchased health inputs *(I)* are assumed to have no direct impact on utility. The purchased household good *(C)* is a pure consumption good. At an optimum, the household exhausts full-income.

At an interior solution, equations (6)–(10) yield household demand functions for *X, I, L* and *C*:

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

Hence the demand for "inputs" into health production depends on market prices of the purchased inputs (P_X, P_I, P_C), the wage *(W)*, nonlabor income (*V*), fixed factors *(Z_1, Z_2)*, and unobserved heath efficiency factor (μ).

Given demand functions for *X**, *I** and *L** from equation (11), substitute these equations into the health production function, equation (2), to obtain the household's health supply function:

$$H^* = S_H(P_X, P_I, P_C, W, V, Z_1, Z_2, \mu).^3 \tag{12}$$

The supply of health is determined by the price of food (*X*), of medical care (*I*), and of purchased consumption goods (*C*), the wage rate (*W*), nonlabor income (*V*), fixed factors (Z_1, Z_2), and unobserved health efficiency factor(μ).

III. Data and the Econometric Model

In this study, the household health production and health supply functions are of primary interest. The key data are *OECD Health Data 2005*, which contains annual data on age-adjusted death rates by cause, food consumption (per capita total calories, fat intake, sugar intake, fruit and vegetable intake), expenditures on health (public and private), and share of the population in the labor force. Data on the consumer price indices 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

[3] Since the household is supplying health to itself, nothing is lost by considering equation (12) to be the household's health demand function.

gross domestic product (GDP in $USPPP) per equivalent adult are available from the *Penn World Table* of Heston et al (2002), and education attainment data for individuals who are 25 years of age and older are available in Barro-Lee Data. With these sources, a panel data set on health related variables for 18 high income countries over 1971-2001 is constructed.[4]

First, we establish an econometric relationship between mortality and diet. Based on available data, the aggregate household health production function is:

$$\ln(H) = \beta_1 + \beta_2 \ln(Calories) + \beta_3 \ln(Sugar) + \beta_4 \ln(Fat) + \beta_5 \ln(Fru \,\&\, Veg) + \beta_6 \ln(Health\_\exp) + \beta_7 Sm2 + \beta_8 Sm3 + \beta_9 Trend + \varepsilon_1 \quad (13)$$

where H is the age-adjusted mortality due to diseases of the circulator system and diabetes mellitus per 100,000 people. *Sugar, Fat,* and *Fru&Veg* are average daily intakes of sugar, fat, and fruits and vegetables, respectively. *Heath_exp* is real($USPPP) per capita public, and private health expenditures, *Sm2* and *Sm3* are dummy variables denoting countries that have a medium and high amount of socialized medicine, respectively, based on the public share of health expenditures.[5] The labor force participation rate, a proxy for leisure time, was never significant in any of our fitted models and is excluded from eq. (13). *Trend* represents effects of a growing stock of medical and dietary information worldwide and available medical technologies.

Although it is plausible that health inputs impact mortality, the impact is not immediate. However, the long-term effects of higher daily intake of calories, other things equal, are expected to increase obesity, obesity-related diseases and eventually death. Likewise, an increase of sugar intake is expected to increase obesity-related mortality. Ludwig (2002) has shown that high sugar and carbohydrate intake causes sharp and large fluctuation in the blood glycemic load, and this produces stress on the body and tends not to satisfy hunger. The long-run effect of an increase in

[4] The following countries are in our sample: Australia (AUS), Austria (AUT), Canada (CAN), Denmark (DNK), Finland (FIN), France (FRA), Ireland (IRL), Italy (ITA), Japan (JPN), Netherlands (NLD), New Zealand (NZL), Norway (NOR), Portugal (PRT), Spain (ESP), Sweden (SWE), Switzerland (CHE), United Kingdom (GRB) and the United States (USA).

[5] The U.S. is the reference group.

fat intake (but reducing carbohydrates to keep calories constant), holding other things equal, is uncertain because fat intake produces a modest impact on the blood glycemic load and also tends to satisfy hunger (Ludwig 2002). We expect that with a lag an increase in total health expenditures will reduce mortality.[6] Countries that have socialized medicine are expected to have more equal distribution of health care and high quality of public health measures which we expect to reduce mortality.

Second, we establish an aggregate household supply function for mortality:

$$\ln(H) = \delta_1 + \delta_2 \ln(P_X) + \delta_3 \ln(P_C) + \delta_4 \ln(W) + \delta_5 \ln(V) + \delta_6 Ed + \delta_7 LFPR + \delta_8 Sm2 + \delta_9 Sm3 + \delta_{10} Trend + \varepsilon_2 \quad (14)$$

where P_X is the real price of food, P_C is the real price of consumer goods less food, W is the real wage which is a proxy for the cost of leisure time, V is real GDP per equivalent adult ($USPPP), *Ed* is the average number of years of formal schooling completed per person 25 years of age and older, *LFPR* is the labor force participation rate of the population, and *Sm2, Sm3, Trend* are as defined above.

Much empirical evidence exists that the demand for food (and drink) in high income countries is price responsive. If the real price of food is reduced over an extended period, food and calorie intake will increase. If the amount of work to be done is unchanged, larger calorie intake over time will lead to weight gain and eventually to obesity. If obesity persists, the likelihood of acquiring diabetes and/or circulatory-related diseases increases, and if these diseases persist, the likelihood of death increases (δ_2<0). Increasing the price of non-food items (which includes the price of health inputs) could have a positive or negative impact on mortality. In the available data it is impossible to obtain a price index for medical services. However, *Sm2* and *Sm3* can be viewed as proxy variables; in countries with a high amount of socialized medicine, the price of (at least a part of) the health input is low.

[6] By lowering the age adjusted mortality rate, people on average live long. Since everyone eventually dies, some other types of mortality must increase.

Assuming positive aggregate labor supply elasticity for those in the labor force, a rise in the real wage rate over the long run will increase average hours of market work per year. This is expected to increase the rate of energy use for work, and other things equal, will reduce obesity, obesity-related diseases and mortality (δ_4<0). Holding the real wage constant, an increase in real per capital income (*V)* represents an income effect on the supply of health. Higher income is expected to increase the demand for a variety of goods, some of which contribute to good health like medical care and medical and dietary information. However, increases in consumption of other goods, e.g., sweetened drinks, salty snacks, eating away from home, contribute to obesity, obesity-related diseases and mortality.

Higher adult *Ed* has possible opposing effects on mortality. Grossman (2000) summarizes evidence of a strongly positively associated between education and a wide range of good health indicators. A population with more education is expected to be more efficient in producing good health. However, individuals with low levels of education also work in jobs that frequently require physical work, and as an individual's education increases, they generally switch to white collar jobs. These latter jobs require little physical effort and may require long hours. The work also may be physiologically stressful. In the latter case, little time is available for recreational exercise, preparing or eating well planned meals, and hence, highly educated workers may be pushed toward an unhealthy lifestyle.

Over the past 30 years in the U.S. and Japan, the overall labor force participation rate has risen substantially, but in Europe, it has changed very little. We expect that individuals who are working in the market burn more energy in calories per day than those who are not in the labor force. Other things equal, this rising labor force participation rate would reduce obesity and obesity related diseases. However, except for Japan, the labor force participation rate of women has been rising, and women remain largely responsible for the planning and preparing meals eaten at home. If their labor force participation rate rises, this tends to increase the demand for eating out

and for pre-packaged, processed, and take-out foods, which tends to be detrimental to good health over the long run. Hence, a higher labor force participation rate results in opposing forces on obesity and obesity related diseases.

Sm2, which indicates a modest level of socialized medicine, is expected to reduce mortality. For *Sm3*, which indicates a very high degree of socialized medicine, e.g., in the U.K., the National Health Service runs all the hospitals, and in the Scandinavian countries and Switzerland, medical care is largely guaranteed by the government. It is unclear how highly socialized medicine will affect obesity-related death rates because some treatments may be highly rationed. *Trend,* which represents the impact of increasing medical and dietary information, new medical technologies, and reorganization of the health care systems, is to have negative effect on morality (δ_{10}<0).

The *r*egressors in equation (13) and (14) are assumed to have their impacts distributed over time. Except for *Trend*, we are quite limited on data going back in time, e.g., it is difficult to push health-related data series back to 1960. Hence, we have chosen to represent the lag pattern of each variable as having trapezoidal weights, where the weights in t and t+1 are zero, then they rise for two years and remain constant for the next five years before declining to zero over the next two years. This shape of the lag pattern captures the belief that the impact of a change in the price of food and other variables in the supply of health equation is not immediate on obesity and mortality, but it is distributed over a decade. Also, this lag pattern minimizes endogenous regressor problems.

IV. Empirical Results

We fit pooled time series cross sectional models of obesity-related mortality as represented by equation (13) and (14). The models are fitted to data for 18 high income countries over 1971-2001 using ordinary least squares (OLS) and the Prais-Winsten estimator. The latter method takes accounts of a single first-order autocorrelation coefficient across the countries in obtaining

estimates of the regression coefficients. In both estimation methods, the standard errors and *z*-values are corrected for heteroscedasticity of variances and contemporaneous correlation of disturbances across countries.[7]

First, in table 1, strong econometric evidence exists for an aggregate household production function for obesity-related mortality. Higher intake of calories and sugar increase significantly mortality. In model 2, a 10 percent increase in calories increases mortality with a lag by 7 percent and a 10 percent increase in sugar intake, other things equal, increases mortality by 1.5 percent. However, higher intake of fat, other things equal, does not significantly affect mortality. On the other hand, higher intake of fruits and vegetables decreases significantly mortality; the elasticity is -0.16. Higher real health expenditures reduce mortality in model 1 significantly but not in model 2 that takes autocorrelation into account. Living in a country with moderate or highly socialized medicine also significantly decrease mortality significantly—by 10 to 16 percent, respectively, in model 2. The estimate of the coefficient of *Trend* is -0.025 in model 2. This represents the impact of increasing medical and dietary information, technical change in medical technology, and adjustments in the national health care system, significantly reduces mortality. All of these health input effects are consistent with expectations, and the R-square for models 1 and 2 are quite large.

Second, in Table 2, we report 4 different estimates of the aggregate household health supply function. In models (2) and (4), the estimation of the regression coefficients takes account of a single first-order autocorrelation coefficient, and in models (3) and (4), we instrument the price of food and the wage to alleviate potential problems with measurement errors. The instrument for the price of food (wage) is a prediction obtained from OLS regression of the real price of food (wage) on a set of 17 country fixed effects, a linear trend and a constant term. We

[7] We considered models with country random and fixed effects but rejected both of them. Random country effects cannot be justified because of their almost certain correlation with the regressors. In these highly aggregated data over time, the use of country fixed-effects leads to over fitting and they account for too much. See Wooldridge (2002, p. 247-279) for a discussion of these issues. Our standard errors are adjusted for correlated panels and a single first-order autocorrelation factor. Estimation was in the panel routine of STATA8.2 "xtpcse," ar1 (Beck and Katz).

prefer the results in model 4, which provides strong econometric evidence of an aggregate household supply function for obesity-related mortality. The coefficient on the price of food is negative (-0.15), which implies that a 10 percent decrease in the price of food increases obesity related mortality by 1.5 percent in the long run, other things equal. The coefficient for the price of nonfood items is also significantly negative, and the impact on health measured as elasticity is -0.46. A higher price of leisure, the wage, also significantly reduces obesity related mortality, and the impact on health measured as elasticity is -0.04.

The impact of income on obesity related mortality is negative, but it is not significantly different from zero at the 5 percent level in model 4. Hence, opposing forces associated with rising nonlabor income seem to be approximately off-setting one another. Likewise, education does not have a significant effect on obesity related mortality, which contradicts (Grossman 2000), but still is plausible. However, a higher aggregate labor force participation rate (*LFPR*) reduces mortality (significant at the 10 percent level).

Countries that have a moderate level of socialized medicine (*Sm2*) have obesity-related death rates that are significantly lower than the United States by 13 percent. However, countries that have highly socialized medical systems (*Sm3*) have obesity related mortality rates that are not significantly different from the U.S. This suggests that too much government involvement with the health care system may actually be adverse to good health associated with obesity-related diseases by causing rationing including excessive queuing. The estimated coefficient of *Trend* is -0.026 and significantly different from zero and similar to that in the aggregate health production function (Table 1). The coefficient of *Trend* implies that obesity-related mortality is declining at 2.6 percent per year due to increasing medical and dietary information, new medical technologies, and reorganization of the health care system. The R-squared for this equation is also quite large.

V. Conclusions

We have provided a sound conceptual foundation for an aggregate household health production function and supply function for health. We used a panel of 18 high income countries over 1971-2001 to fit the empirical aggregate production function and supply function of obesity-related mortality. The empirical evidence for these functions is strong and support most of our expectations. Moreover, we find support for cheap food contributing to rising obesity-related mortality in high income countries, for example, a steadily declining real price of food of 17 percent per year would approximately neutralize the negative trend in obesity rates over 1971-2001. This is a high rate of relative price decline compared to past history and seems unlikely to occur. Our results suggest that the price of health care is lowest in countries that have a modest amount of socialized medicine. Hence, one policy implication is that cheap food, especially cheap unhealthful food over the long run, is bad for human health. Another policy implication is that socialized medicine, at least at a modest level, is good for human health, but less than that of the countries with the most socialized medicine in our sample—Scandinavian countries and the U.K.

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Table 1. Panel Estimates of the Aggregate Household Production Function for Obesity-related Mortality: 18 High Income Countries, 1971-2001 (*z*-values in parentheses, N = 558).

	ln(*H*)	
	OLS with Panel-Corrected Standard Errors	Prais-Winsten Estimator with Panel-Corrected Standard Errors[a/]
	(1)	(2)
ln(*Calories*)	0.795	0.695
	(8.61)	(3.14)
ln(*Fat*)	-0.129	-0.153
	(-2.27)	(-1.34)
ln(*Fru&Veg*)	-0.177	-0.167
	(-6.12)	(-3.01)
ln(*Sugar*)	0.350	0.145
	(8.29)	(1.90)
ln(*Health_exp*)	-0.236	-0.017
	(-9.02)	(-0.31)
Sm2	-0.239	-0.109
	(-9.06)	(-2.04)
Sm3	-0.249	-0.164
	(-12.23)	(-3.23)
Trend	-0.014	-0.025
	(-10.72)	(-8.63)
Constant	29.168	50.329
	(10.64)	(8.60)
R-squared	0.7848	0.9911

a/ In model 2, the value of the first-order autocorrelation coefficient used in this computation was 0.9369 and *z*-values are corrected for heteroscedasticity across countries and contemporaneous correlations across pairs of countries.

Table 2. Panel Estimates of the Aggregate Obesity-related Mortality Supply Function: 18 High Income Countries, 1971-2001 (*z*-values in parentheses, N = 558)

	ln(*H*)			
	OLS with Panel-Corrected Standard Errors	Prais-Winsten Estimator with Panel-Corrected Standard Errors[a/]	IV with Panel-Corrected Standard Errors[b/]	IV Prais-Winsten Estimator with Panel-Corrected Standard Errors[b/]
	(1)	(2)	(3)	(4)
ln(P_X)	-0.074	0.016	-0.189	-0.154
	(-2.31)	(0.32)	(-7.47)	(-2.93)
ln(P_C)	-1.400	-0.416	-1.194	-0.464
	(-8.94)	(-2.07)	(-7.78)	(-2.39)
ln(*Wage*)	-0.005	-0.001	-0.040	-0.041
	(-4.62)	(-2.62)	(-33.90)	(-8.00)
ln(*V*)	-0.466	-0.039	-0.478	-0.100
	(-16.57)	(-0.43)	(-17.52)	(-1.16)
Ed	0.054	0.006	0.048	0.007
	(14.23)	(0.5)	(12.17)	(0.57)
LFPR	-0.002	-0.003	-0.007	-0.005
	(-1.26)	(-0.81)	(-3.73)	(-1.57)
Sm2	-0.087	-0.099	-0.131	-0.128
	(-5.88)	(-1.85)	(-7.64)	(-2.77)
Sm3	-0.050	-0.102	-0.007	-0.050
	(-3.59)	(-2.02)	(-0.61)	(-1.14)
Trend	-0.021	-0.026	-0.021	-0.026
	(-31.32)	(-10.93)	(-26.27)	(-11.61)
Constant	51.793	57.198	51.747	58.233
	(41.76)	(13.43)	(36.04)	(14.44)
R-squared	0.7796	0.9902	0.8379	0.9917

a/ The value of the first-order autocorrelation coefficient used in this computation was 0.9444 in model 2 and 0.9300 in model 4, and *z*-values are corrected for heteroscedasticity across countries and contemporaneous correlations across pairs of countries.

b/ In models 3 and 4 the instrument for the price of food and the wage is obtained from a regression of these prices on 17 country fixed effects, a linear trend, and a constant term.