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DETERMINANTS OF HEALTH OUTCOMES IN INDUSTRIALISED COUNTRIES: A POOLED, CROSS-COUNTRY, TIME-SERIES ANALYSIS

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INTRODUCTION

Rising health expenditure has been a major preoccupation of the health policy debate during the 1990s in most industrialised countries. A substantial literature has been devoted to explaining cross-country variations in health inputs, particularly medical expenditures. However, the main issues concerning health care inputs have been less frequently examined from a perspective of global effectiveness in terms of health outcomes.

Recently there has been a renewed interest in achieving a better appraisal of health outcomes at the aggregate level. The development of relevant and homogeneous measures of the health status of populations is recognised as an important first step in order to assess and compare country performances and to establish targets for health policy. However, if these targets are to be achieved, it is also necessary to distinguish the factors behind the country differences in health performance. This, in turn, requires a multidisciplinary approach, which seeks to disentangle the relative impact of medical, social, economic and institutional factors on health outcomes.

Actual knowledge on the determinants of health outcomes of populations, at the level of countries, suffers from the partiality of models and frameworks used in empirical investigation. While there has been some interest in developing theoretical models of health determinants over the past 15 years (see, for example, Evans and Stoddart, 1990; Hertzman, 1990; Feinstein, 1993), the empirical studies at an aggregate level are rare because of the difficulty in obtaining data on a comparable basis across countries or regions. Moreover, amongst the few aggregate studies that have been carried out, it is not uncommon to find contradictory results, depending on the model and the indicators used, concerning the impact on health of different factors, such as health expenditure or income.

The analysis in this paper seeks to build upon these previous studies by incorporating a wider set of medical and non-medical factors. Health outcomes are compared using a measure of premature mortality instead of more conventional indicators such as standardised mortality rates. In order to quantify the relative impact of each factor (namely, life style, physical and socio-economic environment) on health, a production function approach is adopted, using country-level data for OECD countries over the period 1970-1992. The results of the estimated model are then used to explore some of the factors behind international differences in health performance.

SPECIFICATION OF A HEALTH PRODUCTION FUNCTION: THEORETICAL FRAMEWORK

This section develops a basic health production function in order to investigate factors that determine the health status of a population. In the widest sense, a health production function describes the relationship between combinations of medical and non-medical inputs and the resulting output (Smith, 1993). The production process depends, in part, on the health-care system and its resource input but also on the non-medical, social, economic and physical conditions. Following this reasoning, general form of a health production function can be specified as:

$$H = f(M, E)$$

where H is a measure of the health status of the population, M an indicator of medical resources, and E is a vector of non-medical social, economic and life-style indicators. For simplicity, this paper refers to all non-medical determinants as environmental factors.

Measuring health status

The definition and measure of health outcomes is the first problem that must be confronted in order to estimate a health production function. At present, there is no comprehensive health index available, which captures various aspects of health status (such as the quality and length of life) at the macro-level and on a comparable basis across countries. Most empirical studies rely on mortality rates as a substitute partial indicator because they are objectively measured, relatively precise and readily available.[1]

The use of mortality rates as a global measure of a population's health status is, however, subject to some important limitations. Especially in the industrialised countries, mortality rates are heavily influenced by the relatively high number of deaths at older ages and are not very sensitive to the relatively few deaths occurring among the young. Furthermore, it does not seem easy to fix a political objective based on mortality rates. It is not possible to delay death indefinitely, so a "zero mortality rate" is not a realistic option. It is not easy either to decide how much more reduction is possible from the levels of mortality actually observed.

Consideration of *premature mortality* rates instead of conventional death rates allows one to distinguish deaths, which could be potentially avoided and can give some new insights for developing priorities for policy and assessing efficiency across different health systems. Different indicators can be used to measure premature mortality. In the equations estimated below, the health status of each country's population is measured by standardised, gender-specific Potential Years of Life Lost (PYLL).

PYLL is preferred as a summary measure of premature mortality since it treats the life year saved – rather than life – as the unit of output.[2] In effect, in the calculation of PYLL deaths are weighted according to their prematurity preceding an age limit – 70 in this study. With this age limit, the death of an infant (70 life-years lost) will be given fourteen times the weight given to the death of a person aged 65 (5 years lost). Conventional mortality rates, on the other hand, implicitly give the same weight to all the deaths irrespective of age. Usually, for cross-country comparisons, the number of PYLL is expressed as rate for 100 000 population.[3]

The choice of age limit in the calculations of PYLL implies an element of judgement and may affect the results.[4] For example, some authors have concentrated on the ages between 15 to 65 years old. Others have proposed to include the population from one year old (1 to 65) arguing that an infant is "replaced" rapidly in the society (for example, Romeder et Mc Whinnie, 1988; Rodriguez et Motta, 1989; Lery et Vallin, 1975). This study adopts the age limits 0 to 70. The population under one year old has been included since it is important to evaluate the capacity of industrialised countries to prevent deaths occurring at early ages. The upper limit is fixed at 70. The limit of 65, used in most studies, seems rather low today given that life expectancy is much longer. On the other hand, using a very high age limit, for example 100 years as the biological limit to human life, as proposed by Murray (1988), is not easy to justify since it becomes more and more difficult to consider a death as "preventable" at very advanced ages. Certain causes of death, which are avoidable for younger age groups, may appear as a "natural" end of life for others.

Impact of the medical system

A *priori*, one would expect a positive relation between health care resources and health status if increasing resources implies an improvement in the level and/or quality of health services supplied to the population. It is also likely that there may be diminishing returns to scale above some level of expenditure.

The empirical evidence that emerges from the studies that have been carried out so far in this area is rather weak and conflicting. Most studies based on aggregate data for industrialised countries at a single point in time have not found any significant relation between the total level of resources devoted to health care and health outcomes measured by death rates, infant mortality or life expectancy.[5] However, this type of cross-country analysis is subject to some severe limitations: it is not easy to establish the robustness of the estimations given the small number of observations and the results are highly sensitive to the choice of indicators for health outcomes and resources. Using pooled cross-country time-series data, a small negative relationship between health expenditure and mortality rates is found in a study by Hitiris and Posnet (1992). But their study controls for few factors other than health expenditure and uses crude mortality rates to measure health outcomes. Based on a richer model in terms of explanatory variables but with fewer countries, Grubaugh and Santerre (1994) also find that there is a positive impact on health – as measured by infant mortality rates – of certain health inputs, such as the number of doctors and hospital beds.

Regional studies carried out within different countries do not lead to a consensus either. In several studies examining cross-regional differences of mortality and morbidity in the United States, the impact of health care is usually shown to be very small or even negative (see, for example, Auster *et al.*, 1969; Silver, 1972; Benham and Benham, 1975; Diehr *et al.*, 1979; Newhouse and Friedlander, 1980; Ruhm, 1996). Hadley (1982), however, shows a positive relationship between health expenditure and health using county-level mortality data in the United States. In Europe, there is also some evidence pointing to a positive relationship between health care input and health outcomes (Collins and Klein, 1980; Forbes and McGregor, 1984; Elola *et al.*, 1995). Furthermore, in order to assess the impact of health care on mortality, a number of studies have examined variation in mortality from conditions amenable to medical intervention, either over time or between geographical regions in Europe. Using a list of causes behind "unnecessary untimely mortality" developed by Rutstein *et al.* (1976, 1980), these studies analyse mortality trends and variations for a selection of conditions in which death can be avoided by adequate preventive or therapeutic intervention. Most studies, in which socio-demographic variables are used to control for the influence of external factors, have shown that medical intervention has had a substantial effect on the decline in mortality, especially over the past 30 years (Poikolainen and Eskola, 1986; Charlton *et al.*, 1983; Jougla *et al.*, 1987; Mackenbach *et al.*, 1988).

One reason for these conflicting results might be the difficulty in measuring health care inputs and the partial nature of many of the available indicators of health resources. A potentially relevant indicator of the aggregate volume of resources devoted to health care in each country is total real expenditure on health. Nevertheless, for the purposes of international comparisons, these expenditures still need to be converted into a common currency. In this study, Purchasing Power Parities (PPPs) for medical consumption are used to obtain a comparable measure of the *volume of health resources*. The measurement of health PPPs is, however, subject to some important limitations. OECD (1994) indicates two major problems. First, the number of products included in the medical consumption basket is relatively limited and therefore does not fully reflect the number and complexity of health services provided in each country. The second problem concerns the weighting method. Since, in many countries, deflators are not available for public and private sectors separately, private-sector weights are often used in calculating PPPs for health care expenditure. In countries like Norway, Sweden and the United Kingdom, where the share of private health consumption is low, the private sector weights might not be very appropriate. Some of these potential measurement errors can be explicitly allowed for by using a fixed-effects model.[6]

Most studies ignore the distribution of health care in a country, a factor that might be as important for health outcomes as the overall level of expenditure on, or consumption of, health services. The availability and accessibility of health services for all is not only a political commitment for most OECD countries but might be a crucial condition to assure efficiency in terms of health outcomes. In terms of international comparisons, it is not easy to measure the distribution of health care within each country. Therefore, it is necessary to adopt an indirect approach to the issue. One such possibility is to focus on the way health care is financed. There is some evidence of a positive impact of public financing of medical care on overall mortality and morbidity rates (Anderson, 1975; Leu, 1986; Babazano and Hillman, 1994). For example, Leu (1986) suggests that public intervention can influence the amount and the quality of services provided as well as the relative prices of inputs into the health-care system, and so may have a direct and indirect impact on accessibility. Therefore, in order to represent cross-country differences in "equality of access", the share of public financing in health expenditure is used as a proxy indicator.[7] Given the redistributive influence of public intervention, a positive correlation between public financing and health outcomes is expected.

Impact of environmental factors

A large number of environmental factors have been suggested as possible determinants of health by different epidemiological, demographic and economic studies. They can be classified into three major categories to simplify the discussion: physical environment, life styles and socio-economic factors. Although the associations between particular risk factors and health have been established by different studies, there is not much evidence on the relative importance of these factors in a global framework.

The impact on health of factors relating to the *physical environment* such as water and soil quality, as well as noise and air pollution, is difficult to integrate into health production analyses mainly because of the lack of data. In this paper, the representation of the physical environment will be limited to ambient air quality. The impact of air pollution on health is a growing concern in most industrialised countries (see for example, Derrienic *et al.*, 1989; Sunyer *et al.*, 1991; Dockery and Pope, 1994; ORS, 1994). For lack of better indicators, NO_x emissions per capita will be used as an approximation of the air pollution level in different countries.[8]

The results of various epidemiological studies have led to a growing awareness about the strong relationship between health and *life styles*. In the most general sense, "life style" refers to all the factors over which individuals have some control, such as alcohol and tobacco consumption, physical exercise, personal hygiene, etc. The equations estimated below take into consideration the three most well recognised risk factors: cigarette smoking, alcohol use, and dietary patterns. Per capita alcohol and tobacco consumption are introduced separately as major risk factors on health. Tobacco has been shown to be the intervening factor for about 6 per cent of the deaths from cardio-vascular disease, 25 per cent of deaths from heart attacks and 50 per cent of deaths from chronic respiratory diseases (Hirsh, 1988). Similarly, in terms of mortality and morbidity, alcohol is recognised as an important risk factor for most chronic diseases (diseases of the digestive system, cancer, cirrhosis, etc.) as well as for accidents and violent deaths (see for example, Chick *et al.*, 1986; Guignon, 1990; Choquet and Ledoux, 1989). In order to assess the relationship between dietary patterns and health, two indicators are used: per capita consumption of sugar and butter. Over-consumption of nutrients such as fat and sugar is argued to have a direct negative impact on health; it has been identified as a risk factor for most diseases in which diet makes an important contribution (Manson *et al.*, 1995; O'Connor, 1992). Given that the problems of nutrition in industrialised countries are no longer ones of scarcity, it is expected that there will be a negative impact of sugar and butter consumption on health outcomes.

In order to capture the impact of *socio-economic differences* across industrialised countries, three factors are considered which determine the socio-economic environment both for individuals and for society: income, education and work.

A *priori*, it would appear reasonable to assume that there is a positive relation between income level and health. Higher income results in higher consumption of goods that have a direct impact on the quality of life such as food, housing, schooling, etc. However, empirical studies over the past twenty years have given contradictory results on this relationship (see, for example, Auster *et al.*, 1969; Rodgers, 1979; Wilkinson, 1992; Christiansen, 1994). On a macro-economic level, it is suggested that the relation between income and mortality is not linear and may be characterised by diminishing returns to scale in rich countries (Wilkinson, 1992; Heerink, 1994). Consumption of alcohol and cigarettes, pollution and stress may be associated with economic development and could potentially explain what appears to be a diminishing impact of income on health. Thus, it is important to measure the effect of income on health while controlling for these confounding factors. Real Gross Domestic Product (GDP) per capita, corrected by Purchasing Power Parities, is used to control for income variation across countries.

The distribution of income in a country has also been suggested as an important factor determining health status (Preston, 1975; Wilkinson, 1992; Winegarden, 1978, 1984; Saunders, 1996; Kawachi and Kennedy, 1997). Regional studies in several OECD countries have indicated a direct relationship between income inequality and mortality, even after controlling for major risk factors such as alcohol and tobacco consumption (Marmot *et al.*, 1984; Helmert and Shea, 1994; Kennedy *et al.*, 1996; Kaplan *et al.*, 1996). Smith (1998) has argued that being at the bottom end of an unequal social ranking order raises levels of psycho-social stress which negatively affects health, more than material deprivation. Since there are very few data concerning income inequalities both across countries and over time, its relative impact on health cannot be directly measured.

Education appears to be an important determinant of health in most empirical studies, irrespective of the output measure used, and even when differences in income are controlled for (Auster *et al.*, 1969; Silver, 1972; Grossman, 1972; Valkonen, 1988). Several explanations have been given for the way education influences health. In summary, education seems to determine many of the decisions, which affect the quality of life: choice of job, ability to select a healthy diet and avoid unhealthy habits, efficient use of medical care, etc. Occupation is also suggested as an important intervening variable in this relationship (Leigh, 1983; Kemna, 1987).

The use of a direct measure of educational attainment based on years of schooling or level of education completed is precluded in this study by the absence of time-series data on a comparable cross-country basis. The share of white-collar workers in the total work force is therefore used instead as a proxy measure of social and educational status. The shift of the workforce out of blue-collar jobs into white-collar occupations has gone hand-in-hand with a rising level of educational attainment in OECD countries. Moreover, the nature of one's work has a direct impact on health: fatality rates tend to be much higher in blue-collar occupations than in white-collar ones (Marmot and McDowall, 1986). Recent evidence in different countries show that not only there is a large gap in illness and death rates between different social classes but this has not diminished over time (Feinstein, 1993; Helmert and Shea, 1994; Benzeval *et al.*, 1995). Therefore, it is assumed that the shift from blue-collar to white-collar jobs represents an improvement of working conditions as well as in the average level of education of the work force, and thus should have a positive impact on health outcomes.

DATA AND METHODS

The data set consists of a pooled sample of 21 OECD countries covering the period 1970-1992 (*i.e.* a total of 483 observations).[9] The major data source is the OECD Health Data files. Other data on socio-economic variables – GDP, share of white-collar workers in total employment – are taken from OECD N*ational* A*ccounts* and national surveys. The PYLLs are calculated from unpublished mortality statistics provided by WHO and cover deaths from all causes except suicides.

The following simple fixed-effects model is used to estimate the determinants of health outcomes across countries and over time:[10]

$$H_{it} = \alpha_i + M_{it}\beta + E_{it}\gamma + \varepsilon_{it}, \qquad (1)$$

where H is the measure of health outcome (PYLL), M is a vector of medical variables, E is a vector of non-medical factors, and the subscripts *i* and *t* refer to country

Table 1. **Variable definitions**

H	Potential years of life lost (per 100 000 persons, aged from 0 to 69 years) – all causes except suicides
Texp	Total health expenditure per capita, US$ at 1990 price levels and PPPs for medical consumption
Pubexp	Share of public expenditure in total health expenditure
GDP	Gross Domestic Product per capita, US$ at 1990 price levels and PPPs for GDP
Status	Share of white-collar workers in total work force
Polut	NO_x emissions per capita, kg
Alcohol	Consumption of alcoholic beverages, litres per head of population aged 15 and over
Tobacco	Consumption expenditure on tobacco per head of population aged 15 and over, US$ at 1990 price levels and PPPs for tobacco consumption
Fat	Butter consumption per head, kg
Sugar	Sugar consumption per head, kg

and time, respectively. β and γ are vectors of the coefficients on M and E, respectively, which are assumed to be constant across countries and over time. The constant terms, α, control for country characteristics which are presumed to be stable over the period studied. The list of medical and non-medical factors included as explanatory variables in the estimated equations is given in Table 1.

Given the level of aggregation and problems concerning data quality across countries, it is preferable to employ a simple estimation technique for the regression analysis. Therefore, the regression disturbance, ε, is assumed to be independent and identically distributed across countries and Ordinary Least Squares (OLS) is used to estimate the equations presented below. The robustness of the results is verified by using a Generalised Least Squares (GLS) procedure to take into account cross-sectional heteroscedasticity and time-wise autocorrelation in the residuals.[11] However, since these results do not substantially differ from the OLS estimates in terms of either the relative importance of each variable or the significance of each of the coefficients, only the OLS results are presented.

RESULTS

This section presents the estimated parameters of the fixed-effects model as specified in Equation 1. Regressions were carried out separately for men and

Table 2. **Fixed-effect estimates of the determinants of premature mortality in 21 OECD countries, 1970-1992**

Variable	Women		Men	
	Coefficient	t-statistic	Coefficient	t-statistic
Texp	–0.1771	–4.5	–0.0375	–1.1
Pubexp	–0.1663	–2.6	–0.1774	–3.2
GDP	–0.3499	–5.3	–0.4395	–7.7
Status	–0.8098	–10.2	–0.7441	–10.7
Polut	0.0496	2.0	0.0949	4.4
Alcohol	0.2049	6.4	0.1621	5.8
Tobacco	0.0916	3.2	0.1790	7.1
Sugar	0.1220	3.5	0.1096	3.6
Fat	0.0148	0.9	0.0445	3.1
Australia	0.0181	0.3	–0.0223	–0.4
Austria	0.0319	0.5	0.1067	1.9
Belgium	0.1521	2.3	0.1531	2.6
Canada	0.1921	2.8	0.1221	2.0
Denmark	–0.0070	–0.1	–0.0821	–1.2
Finland	–0.1504	–2.0	0.0971	1.5
France	–0.0071	–0.1	0.1725	2.4
Germany	0.0936	1.3	0.0909	1.4
Greece	–0.3481	–7.1	–0.2772	–6.4
Ireland	–0.1840	–2.8	–0.2633	–4.6
Italy	–0.0570	–1.1	0.0447	1.0
Netherlands	0.0018	0.0	–0.0453	–0.9
New Zealand	0.1579	2.1	0.0688	1.1
Norway	0.0917	1.3	0.1581	2.6
Portugal	–0.3326	–7.0	–0.0511	–1.2
Spain	–0.5083	–11.0	–0.2255	–5.6
Sweden	0.0938	1.4	0.0207	0.4
Switzerland	–0.0379	–0.5	0.0839	1.2
United Kingdom	0.2066	3.3	0.0815	1.5
United States	0.4151	5.5	0.3591	5.4
Intercept	8.1596	15.8	7.8469	17.3
R^2	0.94		0.95	
F	252		292	
DW	2.08		2.20	

Sample: 21 countries, 1970-1992, 483 observations. See Table 1 for the definitions of the variables.

women. All the variables are in logarithmic form and, hence, the regression coefficients in Table 2 can be interpreted as constant elasticities. Columns 1 and 2 correspond to the regression results for women and men, respectively.

It is interesting to note that the coefficient on health expenditure is negative and significant for women, while it is not statistically significant for men. A much stronger negative association both for men and women appears to exist between premature mortality rates and income per capita. However, one point to bear in mind is that it is difficult to isolate the true impact of health expenditure on health outcomes because of the strong collinearity between health expenditure and GDP per capita.[12] When GDP per capita is dropped from the regressions, health expenditure becomes highly significant for both sexes, even though the elasticity for men is still lower than for women. Moreover, both variables are jointly significant. The way health expenditure is financed also appears to affect health outcomes; a larger share of public financing of health care is associated with lower rates of premature mortality for both sexes.

Among the environmental factors included in the regressions, the impact of the proxy variable for work conditions and education on premature mortality is shown to be particularly large and significant. For both men and women, a 10 per cent increase in the share of white-collar workers in the workforce implies almost a 7 per cent reduction in premature mortality, holding all other factors constant. The results in Table 2 also confirm that there is a small, positive but significant relation between air pollution and premature mortality in developed countries over the past two decades. As expected, both alcohol and tobacco consumption exhibit a positive association with premature mortality, but it would appear that for the same proportionate increase, the impact of alcohol consumption on health is higher than tobacco consumption. Sugar and butter consumption are also positively related with male mortality although butter consumption seems not to be significant for woman.

DISCUSSION

The results of the regressions presented in Table 2 can be used to assess the relative importance of medical and non-medical factors in contributing to the substantial decline in premature mortality that has occurred in all OECD countries over the period 1970-1992. More specifically, for each explanatory variable *v* in columns 1 and 2 of Table 2, its contribution to the decline in potential life years lost in country *i* can be estimated as follows:

$$C_{i,v} = \alpha_v * (\ln(V_{i,92}) - \ln(V_{i,70}))*100 \qquad (2)$$

where $C_{i,v}$ represents the percentage-point contribution of variable *v* to the log-percentage change in premature mortality between 1970 and 1992 in country *i*; α_v is the corresponding coefficient in Table 2; $V_{i,92}$ and $V_{i,70}$ are the values of variable *v* in 1992 and in 1970, respectively. Of course, these estimates are far from being precise and should only be taken as an indication of the relative importance of each factor.

The results of this calculation are shown for women and men in Table 3. The first column corresponds to the actual decline in PYLL, expressed as a log-percentage change. The estimated contribution of each explanatory variable to this decline

Table 3. **Determinants of the evolution of premature mortality in 21 OECD countries, 1970-1992**

Percentages[1]

	Women										
	PYLL	*Texp*	*Pubexp*	*GDP*	*Status*	*Polut*	*Alcohol*	*Tobacco*	*Sugar*	*Fat*	*Residual*
oefficient	–	*–0.18*	*–0.17*	*–0.34*	*–0.8*	*0.05*	*0.20*	*0.09*	*0.12*	*0.02*	–
ustralia	–73.9	–10.7	–2.9	–11.7	–19.8	–0.7	–4.3	–6.3	–0.2	–1.7	–15.6
ustria	–71.1	–10.6	–0.8	–19.6	–26.9	–0.1	–2.8	0.7	0.0	–0.2	–10.8
elgium	–59.4	–18.9	–0.4	–17.5	–24.3	–2.0	–0.5	–3.6	0.6	–0.5	7.6
anada	–65.8	–10.0	–0.6	–15.2	–23.1	0.3	0.0	–6.2	–1.2	–1.3	–8.5
enmark	–27.7	–9.4	0.8	–14.2	–30.1	1.8	6.5	–0.5	–2.0	–0.6	19.9
inland	–53.0	–13.6	–1.2	–15.5	–48.0	3.0	8.6	–1.0	–2.6	–1.4	18.8
rance	–57.7	–20.4	0.0	–15.4	–28.7	0.1	–7.5	3.5	–0.1	0.0	10.8
ermany	–67.6	–13.7	–0.5	–16.9	–23.1	–0.2	0.9	–0.1	–0.1	–0.4	–13.6
reece	–70.3	–13.5	–5.9	–17.1	–52.1	5.9	5.0	7.3	5.0	0.2	–5.2
reland	–71.8	–13.5	1.2	–24.9	–26.4	2.8	9.0	–2.6	–4.8	–1.1	–11.5
taly	–86.3	–17.2	2.1	–19.3	–39.6	1.5	–11.1	2.9	–0.5	0.2	–5.3
apan	–86.4	–20.2	–0.3	–25.0	–25.1	–1.8	11.3	–2.9	–4.1	0.8	–19.1
etherlands	–44.4	–5.8	1.6	–12.9	–23.5	0.3	–5.4	–3.0	–2.0	–0.1	6.4
ew Zealand	–51.3	–2.6	0.8	–7.1	–26.4	2.5	–0.2	–4.3	2.3	–1.0	–15.2
orway	–37.1	–16.4	–0.6	–22.9	–31.3	1.1	–0.4	–0.6	–0.7	–0.9	35.6
ortugal	–107.8	–26.2	1.0	–22.2	–63.3	5.9	–7.7	2.7	1.1	1.0	–0.1
pain	–66.9	–22.1	–3.1	–18.5	–37.0	4.3	–4.6	4.9	0.6	0.0	8.6
weden	–45.6	–4.8	0.2	–10.0	–23.0	1.0	–2.7	–1.2	–0.8	–0.7	–3.6
witzerland	–50.9	–9.9	–1.6	–8.3	–14.2	–0.1	–2.9	1.2	–0.8	–0.2	–14.1
nited Kingdom	–54.4	–12.5	0.6	–13.4	–20.2	0.3	5.6	–4.7	–1.6	–1.5	–7.0
nited States	–49.8	–11.4	–2.6	–11.2	–14.0	–0.6	–1.1	–3.5	–4.6	–0.1	–0.7
OECD[2]	–61.9	–13.5	–0.6	–16.1	–29.5	1.2	–0.2	–0.8	–0.8	–0.5	–1.1
	Men										
	PYLL	*Texp*	*Pubexp*	*GDP*	*Status*	*Polut*	*Alcohol*	*Tobacco*	*Sugar*	*Fat*	*Residual*
oefficient		*–0.04*	*–0.17*	*–0.44*	*–0.75*	*0.09*	*0.16*	*0.18*	*0.1*	*0.05*	
ustralia	–73.8	–2.3	–3.1	–14.7	–18.2	–1.3	–3.4	–12.4	–0.2	–5.1	–13.2
ustria	–62.2	–2.2	–0.8	–24.6	–24.7	–0.3	–2.2	1.4	0.0	–0.6	–8.1
elgium	–51.2	–4.0	–0.4	–21.9	–22.3	–3.9	–0.4	–7.0	0.5	–1.5	9.7
anada	–65.2	–2.1	–0.6	–19.1	–21.2	0.6	0.0	–12.1	–1.1	–4.0	–5.5
enmark	–26.3	–2.0	0.8	–17.8	–27.6	3.4	5.1	–1.0	–1.8	–1.7	16.2
inland	–59.7	–2.9	–1.3	–19.5	–44.1	5.7	6.8	–1.9	–2.4	–4.1	3.9
rance	–39.7	–4.3	0.0	–19.4	–26.3	0.2	–5.9	6.8	–0.1	0.0	9.4
ermany	–59.1	–2.9	–0.5	–21.3	–21.2	–0.3	0.7	–0.3	–0.1	–1.1	–12.2
reece	–47.6	–2.9	–6.3	–21.5	–47.9	11.3	4.0	14.3	4.5	0.5	–3.7
reland	–57.5	–2.9	1.3	–31.3	–24.3	5.3	7.1	–5.1	–4.3	–3.3	–0.1
taly	–59.5	–3.6	2.2	–24.3	–36.4	2.9	–8.8	5.6	–0.5	0.7	2.6
apan	–75.3	–4.3	–0.3	–31.4	–23.1	–3.4	8.9	–5.6	–3.7	2.5	–14.9
etherlands	–51.0	–1.2	1.7	–16.3	–21.6	0.6	–4.3	–5.8	–1.8	–0.3	–2.1
ew Zealand	–47.7	–0.5	0.9	–9.0	–24.2	4.8	–0.2	–8.4	2.1	–3.1	–10.0
orway	–46.4	–3.5	–0.6	–28.7	–28.7	2.1	–0.3	–1.2	–0.6	–2.9	18.1
ortugal	–67.1	–5.6	1.1	–27.8	–58.2	11.2	–6.1	5.2	1.0	3.1	8.9
pain	–32.8	–4.7	–3.3	–23.2	–34.0	8.2	–3.7	9.6	0.6	0.0	17.8
weden	–42.0	–1.0	0.2	–12.6	–21.1	2.0	–2.2	–2.3	–0.7	–2.2	–2.1
witzerland	–42.2	–2.1	–1.7	–10.4	–13.1	–0.1	–2.3	2.4	–0.8	–0.5	–13.6
nited Kingdom	–55.6	–2.6	0.7	–16.9	–18.6	0.6	4.4	–9.2	–1.4	–4.5	–8.1
nited States	–43.7	–2.4	–2.8	–14.1	–12.8	–1.1	–0.9	–6.7	–4.1	–0.4	1.7
OECD[2]	–52.7	–2.9	–0.6	–20.3	–27.1	2.3	–0.2	–1.6	–0.7	–1.4	–0.2

. Log difference between 1970 and 1992 multiplied by 100. For each explanatory variable, this ratio is multiplied by the corresponding coefficient.
. Simple average of 21 countries.

in premature mortality is presented in the next nine columns. Country-specific effects are assumed to be constant over time and so have no impact on changes over time. The last column of Table 3 gives the residual difference between the actual reduction in premature mortality and the reduction accounted for by the model. In the United Kingdom, for example, the actual decline in PYLL for women is 7 percentage points more than the decline predicted by the model given the rise in health expenditure, income, alcohol consumption, etc.

In terms of the actual reduction in premature mortality, there is a considerable variation across countries. For women, the greatest reduction occurred in Portugal whereas Japan appears to have been the most successful country in reducing male premature mortality over the period 1970-1992. Denmark experienced the smallest decline for both men and women. In almost all countries, the reduction in premature mortality has been about 5 to 20 percentage points more important for women than men. The Netherlands, Norway and the United Kingdom are the only exceptions where the decline in PYLL for men was slightly more important than for women. The gap between the two sexes in the evolution of premature mortality is particularly large in the Mediterranean countries.

In most countries, the rise in the employment share of white-collar workers plays the greatest role in the reduction of premature mortality between 1970 and 1992. Even in countries such as Portugal and Greece, where economic growth over this period has been more rapid than the OECD average, the improvement in health due to the rise in "work status" is more than double the contribution from the rise in per capita income. While it is well established that there are considerable health inequalities across social-economic classes, the reasons for these differences are less well identified. There are probably several, complementary, explanations, as touched on above. Education is an important component in this relationship. The choices of work, social environment, life style and attitudes towards medical prevention and treatment are all affected by education. Given that the estimated equations already control for certain life-style factors such as alcohol and tobacco consumption, as well as for the level of income and medical consumption, there appear to be other factors linked to occupation and/or education which have a significant impact on health. Clearly, work itself, in terms of its difficulty, risks and other working conditions, is an important determinant of health. The incidence of fatal accidents and mortal diseases linked to work are still much higher for manual workers than for non-manual workers. Moreover, the "work-status" variable may also be acting as a proxy for other life-style factors which have not been explicitly controlled for in the estimations. For example, in the United States, persons with a university education do twice as many sporting activities than those with only a secondary education.[13] Similar behavioural variations are observed in many countries.

The second most important factor behind improvements in health outcomes would appear to be the rise in per capita income which is estimated to have contributed between 10 to 30 percentage points of the decline in PYLL between 1970 and 1992. This result is consistent with the conclusions of most other studies using cross-country data. It is reasonable to think that economic development would improve the quality of global infrastructure such as housing conditions, road quality and public hygiene, which have a direct impact on health.[14] There are also more direct links with health outcomes such as safety measures in public and private transport which are not measured by health expenditures. At the same time the negative impact of air pollution is particularly visible in the countries where there has been accelerated economic growth and rapid industrialisation during the past 20 years, such as Greece, Portugal and Spain.

In most countries the estimated impact of health expenditure in terms of lowering female mortality appears to be quite close to that of GDP. In fact, in the United States as in the United Kingdom, about 12 percentage points of the reduction in female PYLL is explained, in the equations, by the increase in health resources. This contribution goes up to more than 25 percentage points in Portugal and accounts for around 20 percentage points in Japan and France. For men, a rise in health expenditures would appear to play only a very modest role in reducing premature mortality in the industrialised countries. This significant difference between the two sexes with respect to the impact of total health expenditures on health outcomes might partly be explained by contrasting mortality patterns. Considering the major causes of death for both sexes, male mortality rates appear to have been less sensitive to medical interventions. In most OECD countries, around 30 per cent of the premature mortality for men is a result of "external causes" such as violence, accidents, etc. For women, malignant neoplasms are the first cause (between 20 and 30 per cent) of premature mortality, while external causes represent only about 16 per cent. Specific prevention programmes designed for women such as systematic screening for breast and cervical cancer have been generally shown to be particularly effective. It is also reasonable to think that women may have a greater propensity to consume medical services regularly because of biological differences and their primary role in rearing children. Clearly further research using separate data by gender on health care utilisation would be necessary to verify these hypotheses.

Since the public share of total expenditure on health has remained fairly constant over the past two decades, its contribution to the decline in premature mortality has been negligible. Similarly, the small, estimated effects of butter and sugar consumption in Table 3 are explained by relatively little change over time in dietary patterns as well as by the small coefficients for these variables. Substantial variations across countries in the evolution of alcohol and tobacco consumption, on the other hand, give rise to very different results in terms of their impact on changes in health outcomes over time. For example, comparing the United Kingdom with Australia, it appears that there has been a parallel increase in GDP and health expenditure per capita and in the non-manual share of total employment over the past 20 years. However, the reduction in premature mortality for men has been 18 percentage points more in Australia than in the United Kingdom, mainly because of a decline in alcohol consumption in Australia and a rise in the United Kingdom. Similarly, for Canada, a substantial decline in tobacco consumption made an impressive contribution to the reduction in premature mortality, especially for men. On the other hand, for Greece and Italy, the negative impact of increasing tobacco consumption on mortality is also notable.

The regression results can also be used to assess the relative contribution of different factors to the health disparities between countries for a given point in time. Using Japan as the reference country, it is interesting to see how much the level differences across countries, for each variable in the model, would explain the variances in premature mortality in 1992. Japan is chosen as the reference since, by the available aggregate measures of health outcome, it is currently the best performing country in the OECD area. For each explanatory variable *v* in columns 1 and 2 of Table 2, its contribution to the disparity in premature mortality in country *i* with respect to Japan is estimated as follows:

$$D_{i,v} = \alpha_v * (\ln(V_{i,92}) - \ln(V_{Japan,92}))*100 \qquad (3)$$

where $D_{i,v}$ represents the percentage-point contribution of variable *v* to the log-percentage difference in premature mortality between country *i* and Japan in 1992; α_v is the corresponding coefficient in Table 2; $V_{i,92}$ and $V_{Japan,92}$ are the values of variable *v* in 1992 in country *i* and in Japan, respectively.

Table 4 presents the results of this calculation. Again these results should be taken as suggesting orders of magnitude rather than as precise estimates. The first column in Table 4 gives the actual log-percentage difference in PYLL between Japan and the corresponding country. The positive numbers indicate that, in 1992, all countries have higher premature mortality levels than that of Japan. The next nine columns report the estimated contribution of each explanatory variable to this gap in premature mortality. For instance, in Australia premature mortality for women is about 27 log-percentage points higher than in Japan. The low level of health resources in Australia accounts for 9 percentage points of this difference while consumption of sugar and butter explains more than 14 percentage points. On the other hand, the estimates suggest that premature mortality for women would be even higher in Australia than in Japan – by around 12 percentage points – if it were not for the fact that the employment share of white-collar workers is higher in Australia than in Japan.

In Table 4, the impact of life-style variables on determining cross-country differences in premature mortality becomes particularly visible. For instance, the lower level of sugar and butter consumption in Japan accounts for around 12 percentage points of the difference in PYLL compared with Canada, and 11 percentage points compared with Sweden.

Table 4. **Determinants of cross-country of premature mortality**

Difference[1] between Japan and the other industrialised countries, 1992

	Women											
	PYLL	*Texp*	*Pubexp*	*GDP*	*Status*	*Polut*	*Alcohol*	*Tobacco*	*Sugar*	*Fat*	*Dummy*	*Residual*
oefficient	–	*–0.18*	*–0.17*	*–0.34*	*–0.8*	*0.05*	*0.20*	*0.09*	*0.12*	*0.02*	–	–
ustralia	26.6	9.3	0.9	4.7	–12.2	7.5	3.8	0.5	12.0	2.1	1.8	–3.7
ustria	32.8	4.7	1.3	2.8	3.1	4.1	9.0	0.3	8.1	2.9	3.2	–6.7
elgium	36.6	4.4	–3.7	3.4	–8.2	2.2	8.8	–0.7	9.2	3.4	15.2	2.6
anada	26.4	4.4	–0.4	1.3	–12.9	9.0	2.2	–2.4	9.6	2.1	19.2	–5.7
enmark	46.7	12.8	–2.4	2.9	–8.1	7.6	8.5	0.0	9.4	3.2	–0.7	13.3
inland	22.8	9.8	–1.8	8.8	–9.8	7.8	2.7	–1.7	7.6	3.1	–15.0	11.1
rance	25.3	0.5	–0.8	1.8	–8.2	4.3	14.1	–1.2	7.9	3.8	–0.7	3.9
ermany	31.3	4.8	–0.1	–1.1	–2.9	6.0	12.0	–0.3	7.2	3.4	9.4	–7.1
reece	27.7	26.9	–1.1	31.1	19.2	5.5	–25.0	2.7	5.8	0.5	–34.8	–3.2
reland	35.6	19.3	–1.2	16.7	4.6	5.8	7.5	1.1	7.1	3.2	–18.4	–10.1
taly	21.5	5.8	–1.2	3.7	3.0	5.8	6.3	–1.1	4.1	1.8	–5.7	–0.9
apan	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
etherlands	26.0	6.7	–1.2	4.4	–14.1	6.0	4.9	–1.2	8.8	1.9	0.2	9.6
ew Zealand	49.4	12.5	–1.2	11.0	–2.1	6.6	4.5	–2.1	11.0	3.8	15.8	–10.5
orway	19.6	7.2	–4.8	3.6	–12.9	7.6	–10.8	–3.2	8.2	2.2	9.1	13.4
ortugal	58.9	17.8	4.1	26.7	24.5	4.4	4.0	–2.9	4.9	1.0	–33.3	7.5
pain	27.0	12.7	–1.7	14.8	20.1	5.0	11.1	–1.0	4.7	–0.8	–50.8	13.0
weden	10.0	8.6	–3.0	4.3	–15.4	7.1	–4.4	–1.7	8.5	2.5	9.4	–6.0
witzerland	21.0	4.3	0.2	–4.2	3.0	3.7	9.3	–4.7	10.0	3.3	–3.8	–0.3
nited Kingdom	35.1	9.4	–2.7	6.5	–12.3	6.8	2.5	–0.1	8.9	2.2	20.6	–6.8
nited States	60.2	1.5	8.2	–5.8	–12.7	9.9	2.9	1.2	5.4	1.6	41.5	6.5

	Men											
	PYLL	*Texp*	*Pubexp*	*GDP*	*Status*	*Polut*	*Alcohol*	*Tobacco*	*Sugar*	*Fat*	*Dummy*	*Residual*
oefficient	–	*–0.04*	*–0.17*	*–0.44*	*–0.75*	*0.09*	*0.16*	*0.18*	*0.1*	*0.05*	–	–
ustralia	19.9	2.0	0.9	5.9	–11.2	14.3	3.0	1.0	10.8	6.3	–2.2	–10.9
ustria	38.6	1.0	1.3	3.6	2.8	7.9	7.1	0.6	7.2	8.8	10.7	–12.5
elgium	38.7	0.9	–3.9	4.3	–7.5	4.2	7.0	–1.4	8.2	10.2	15.3	1.4
anada	20.1	0.9	–0.4	1.6	–11.9	17.2	1.8	–4.8	8.7	6.3	12.2	–11.6
enmark	33.0	2.7	–2.6	3.7	–7.5	14.6	6.7	0.1	8.5	9.7	–8.2	5.2
inland	36.1	2.1	–1.9	11.1	–9.0	14.9	2.1	–3.3	6.9	9.4	9.7	–5.8
rance	44.1	0.1	–0.9	2.2	–7.5	8.2	11.1	–2.3	7.1	11.3	17.2	–2.6
ermany	33.0	1.0	–0.1	–1.4	–2.6	11.5	9.5	–0.6	6.5	10.1	9.1	–10.1
reece	31.0	5.7	–1.2	39.1	17.6	10.5	–19.8	5.4	5.3	1.6	–27.7	–5.4
reland	25.8	4.1	–1.2	21.0	4.2	11.0	5.9	2.1	6.4	9.5	–26.3	–10.9
taly	34.3	1.2	–1.2	4.6	2.8	11.1	5.0	–2.2	3.6	5.4	4.5	–0.4
apan	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
etherlands	12.5	1.4	–1.3	5.5	–12.9	11.5	3.9	–2.3	7.9	5.8	–4.5	–2.6
ew Zealand	42.4	2.6	–1.3	13.9	–1.9	12.7	3.5	–4.1	9.9	11.4	6.9	–11.2
orway	17.7	1.5	–5.1	4.6	–11.8	14.5	–8.6	–6.2	7.4	6.5	15.8	–0.8
ortugal	77.1	3.8	4.4	33.5	22.6	8.4	3.2	–5.6	4.4	3.1	–5.1	4.4
pain	48.6	2.7	–1.8	18.6	18.5	9.5	8.8	–2.0	4.2	–2.5	–22.5	15.1
weden	2.0	1.8	–3.2	5.4	–14.1	13.6	–3.5	–3.2	7.6	7.5	2.1	–12.1
witzerland	27.5	0.9	0.3	–5.2	2.8	7.1	7.4	–9.3	9.0	9.8	8.4	–3.7
nited Kingdom	21.3	2.0	–2.9	8.2	–11.3	13.0	2.0	–0.2	8.0	6.7	8.2	–12.4
nited States	62.4	0.3	8.7	–7.3	–11.6	19.0	2.3	2.4	4.9	4.7	35.9	3.3

. Log differences in level between Japan and corresponding country in 1992 multiplied by 100. For each explanatory variable, this ratio is multiplied by the corresponding coefficient.

The coefficient on the dummy variable for each country represents, in principle, the impact on health of unobservable country-specific factors with respect to Japan – the reference country. In Portugal, for example, the level of PYLL (in logs) in 1992 is about 60 per cent higher for women compared with Japan. While this difference is mainly explained by lower levels of per capita GDP, education and health resources, the coefficient of the dummy variable for Portugal is strongly negative. In other words, if all the explanatory variables of the model had exactly the same levels in both countries, premature mortality would be some 30 percentage points lower in Portugal than in Japan. On the other hand, in the United States premature mortality is some 60 per cent higher than in Japan in log terms, despite a higher level of per capita GDP and a higher share of white-collar workers in total employment. This is partly explained by differences between the two countries with respect to the public share of health expenditure, pollution and life-style factors. At the same time, unobserved country-specific factors in the United States contribute around 40 percentage points to the higher rate of premature mortality.

In order to understand the significance of these dummy variables, one needs to consider all the factors which are not explicitly included in the estimated equations and which might vary significantly between countries such as climatic, cultural and genetic factors. For example, a favourable climate may be one reason for the negative coefficients on the country-specific factors (*i.e.* implying, *ceteris paribus*, lower premature mortality than in Japan) for the Mediterranean countries. There are also other medical and environmental factors, such as the quality of medical care, income distribution and water quality which have been shown to have some impact on health outcomes. For example, excess mortality among the black population in the United States appears to be strongly linked to poverty (Geranimus *et al.*, 1996). Overall, the coefficients of the dummy variables provide some important information about distinctive characteristics of countries, even if they cannot be fully explained. They allow for some evaluation to be made of how countries perform with respect to health outcomes after controlling for differences across a range of medical and environmental factors.

CONCLUSION

The model presented and estimated in this paper improves upon previous studies at the macro level in terms of including a richer palette of explanatory variables within an estimation strategy that explicitly takes into account unobservable country-specific factors. Nevertheless, every model is inevitably a simplification of reality through which one tries to understand the main features of a system. Hence, it is important to underline certain points that call for prudence when interpreting the results presented above. First, the set of indicators employed in the estimations to represent medical and environmental factors is limited and further refinements to these indicators could be made. For example, it would be interesting to examine separately for men and women variations in health-care consumption, life styles, etc. Second, it is assumed that the medical and environmental factors have only a contemporaneous effect on premature mortality; the estimated equations do not account for dynamic effects. Third, it would be preferable to employ more elaborate indicators of health outcomes, which take quality-of-life aspects into consideration. This point is important to better define the health problems and needs of each country's population. Finally, the reliability of the results in this study is subject to the usual measurement problems, which frequently arise when using aggregate cross-country data. Despite the considerable efforts of the OECD to standardise definitions, the series are not always entirely coherent across countries or within countries over time.

Bearing in mind these limits, the results of this study provide some new evidence on the determinants of premature mortality in OECD countries over the past two decades and raise a number of important issues for policy. Contrary to what has been suggested by some authors, there appears to be a significantly positive relation between health expenditure and health, particularly for women. It is also important to verify the positive effect of public financing on health outcomes given the current policy debate about the appropriate role of the public sector in health-care provision. At the same time, the results strongly suggest that environmental factors – in the wide sense of the term – are more important than medical inputs in explaining variations in premature mortality in industrialised countries. Among these, occupational status appears to play the most important role. This variable reflects quite complex forces. It is not easy to distinguish how much of its effect is linked to changes in actual working conditions and how much reflects rising levels of education. Separating the relative contribution of each by, for example, using an independent indicator for educational attainment, would require harmonised time series across countries which are currently not available.

In terms of future work, several conclusions can be drawn. First, the results of this study suggest that health outcomes across countries can be modelled and useful policy conclusions drawn from this kind of quantitative evaluation, although further work at both the empirical and theoretical level is clearly called for. The rich spectrum of data pooled together in OECD Health Data already provides an important source to investigate cross-country variations in health performances amongst industrialised countries. While this data has been an essential source for investigating the reasons for rising health expenditures, it has, so far, been little exploited for examining the global efficiency of these resources in terms of achieving better health outcomes. Secondly, this analysis clearly demonstrates the need for continuing effort to develop more detailed comparable data on various inputs into the health sector. In order to carry out comparative analysis of health systems, improving the scope of the data and indicators on the social, economic and physical environment as well as on life styles is equally important. Further work is called for to investigate the nature of the relationship between gender-specific, medical-care patterns and the health status of men and women. For example, it would be interesting to examine whether women have a greater propensity to consume preventive care compared with men. Finally, while it is also important to develop more sophisticated and comparable measures of health outcomes, such as quality-adjusted life expectancy, the real challenge will be not only to produce a point estimate for each country but to construct reasonably long time series.

NOTES

1. Although there are still measurement problems in assigning cause of death.
2. See for example, Haenszel (1950), Doughty (1951), Murray (1988)
3. For each country *i* and for each year *t*, the standardised PYLL rate (expressed per 100 000 population) is calculated as follows:

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

where *a* stands for age, *l* is the upper age limit chosen for the measure (in this study, 70), d_a is the number of deaths at age *a*, p_{at} refers to the number of persons aged *a* in country *i* at time *t*, P_a refers to the number of persons aged *a* in the reference population, and P_n refers to the total number of persons aged 0 to *l*-1 in the reference population. In this study, the total OECD population in 1980 is taken as the reference population.

4. The robustness of the estimations has been tested by using age 65 as an upper limit. The estimation results are largely invariant to the choice of the age limit.
5. See for example, Cochrane (1978), Poikolainen and Eskola (1988), Mackenbach (1991), Babazano and Hillman (1994).
6. Indeed, in the estimations with country dummies, the question of choosing the appropriate conversion index becomes irrelevant. The use of country dummies effectively means that the estimates only reflect variations over time *within* countries in both the dependent and independent variables and no account is taken of variation *across* countries in the levels of these variables. Since each country's constant-price series for health expenditure is multiplied by a conversion factor for a single year (1990), the coefficient on health expenditure will be invariant to the choice of conversion index. Switching from GDP PPPs to health-specific PPPs will simply change the coefficients on the country dummy variables.
7. A better approximation might be the level of health insurance coverage of the population in each country. However, differences in the organisation and financing of the various insurance schemes make it difficult to pool time series across countries. We nevertheless verified the robustness of the results for a small group of countries using the percentage of the population covered by health insurance.
8. Ambient air quality is measured by the concentration of air pollutants such as NO_x, in selected cities. Lacking nation-wide observations relative to concentration, total emission estimates of NO_x for each country are used instead as proxies.

9. Of the current 29 OECD members, Luxembourg, Iceland, Turkey, the Czech Republic, Hungary, Poland, Mexico and Korea are not included in the analysis because of insufficiently long time-series data for some variables.

10. This equation does not explicitly allow for dynamic effects, while many of the explanatory variables might be expected to affect outcomes with a sizeable lag. However, the results do not appear to be qualitatively altered by including lags on the explanatory variables (see Or, 1997).

11. A weighted least-squares procedure was also tried where each observation was weighted by the square root of its country's population. The robustness of the OLS estimations was also tested by a number of practical tests such as dropping countries one by one from the sample as well as, separately, groups of countries with a population greater than 100 million or less than 5 million. This procedure was also repeated along the time dimension, first by dropping one year at a time then the years before 1975, after 1987 and between 1978-84 (see Sayrs, 1989). Generally, the estimated coefficients were little affected by these sensitivity tests.

12. The coefficient of correlation between per capita GDP and health expenditure is 0.89 in this panel.

13. US Department of Education, National Centre for Education Statistics, *The Condition of Education*, Washington, 1994.

14. Homelessness and bad quality of housing, for instance, has been found to be one of the most important determinants of health inequality in several OECD countries (see for example, Best 1995).

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