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Abstract

Health economists have studied the determinants of the expected value of health status as a function of medical and nonmedical inputs, often finding small marginal effects of the former. This paper argues that both types of input have an additional benefit, viz. a reduced variability of health status. Using OECD health data for 24 countries between 1960 and 2004, medical and nonmedical inputs are found to reduce the variability of life expectancy. While the evidence supports the "flat-of-the-curve medicine" hypothesis with respect to the expected value of life expectancy and its variability, healthcare expenditure is comparatively effective in reducing variability.

JEL classification: I10, I12, J10

Keywords: production of health, control over health status, Gini coefficient

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1 Introduction and motivation

Industrial countries have been spending a rising share of their economic resources on healthcare. From 1960 to 2004 healthcare expenditure (HCE) of OECD countries increased from 3.8 percent of GDP on average to 8.9 percent. Over the same period, health has improved, with average life expectancy at birth increasing from 68.4 to 78.5 years. However, this increase has slowed recently. In the United States, it has been 0.19 percent per annum between 1980 and 2004, down from 0.3 between 1960 and 1980. Since HCE continued to grow at a rate of 7.7 percent between 1980 and 2004, this was often interpreted as evidence of decreasing marginal returns ("flat-of-the-curve medicine"), raising the question of why citizens and governments failed to reallocate resources away from medicine.

However, this conclusion may be premature on at least two acounts. The first derives from the "production of health" concept (Grossman [1972]), which emphasizes nonmedical inputs, notably individuals' own efforts at maintaining health. Specifically, Zweifel et al. [2009, ch. 4] argue that possibly individuals reduced health-enhancing efforts or used more unhealthy consumption goods, thus counterbalancing the positive effect of medical care on life expectancy. Second, the implicit assumption that individuals only value changes in the expected value of health status is open to criticism. If people are risk-averse with regard to their health, they are made better off by a reduction in the variance of health status (Lichtenberg [1998]). Following up on this second aspect, one is led to ask a few additional questions. How has the variability of health status developed over time? Can this development be related to inputs to the production of health? And if so, what is the relative effectiveness of medical vs. nonmedical inputs? This contribution seeks to provide answers to these questions.

The remainder of this paper is structured as follows. After a literature review in Section 2, the Gini coefficient is introduced as an indicator of uncertainty with regard to the length of life

in Section 3. Section 4 is devoted to the econometric specification, the description of the data, and variable definitions. Estimation results are presented and discussed in Section 5. Section 6 concludes with a summary of key findings and suggestions for future work.

2 Survey of the literature

This survey is in two parts. First, research relating the expected value of health status to medical and nonmedical inputs is discussed, checking whether the choice of output indicator matters and which model specification is most appropriate for the model to be estimated in Section 3. Second, the survey reports on work focusing on the variability of health status and its determinants. Since this investigation is limited to OECD country data, the review cites only studies based on aggregate observations.

2.1 Determinants of health status at the aggregate level

At the aggregate level, the choice of output variable in a production function of health is constrained by data availability. Traditionally, mortality rates and life expectancies have served as proxies of health status.

In their seminal contribution, Auster et al. [1969] relate age- and sex-adjusted mortality rates of U.S. states of 1960 to medical inputs (viz. number of physicians, pharmaceutical outlay, capital stock of hospitals, and medical auxiliary staff), economic factors (income, years of schooling, and degree of urbanization), factors related to lifestyle (alcohol consumption, smoking), and organizational factors (share of group practices and medical schools). Schooling and income tend to reduce mortality rates, but both effects are not significantly different from zero. Only medical auxiliary staff is found to reduce mortality rates, while physicians seem to increase it. However, this might be due to reverse causality to the extent that physicians

work in areas where there is demand for their services, indicated by a high risk of death. In a two-stage least squares estimation, all medical inputs have the expected sign but are not significant, suggesting that they are not effective at the margin.

The follow-up study by Thornton [2002] provides more recent evidence for the United States. It modifies the approach by Auster et al. [1969] in two major ways. First, additional determinants are included (share of married couples and crime rate). Second, in addition to medical inputs, income is treated as endogenous as well. Using U.S. data for 1990, Thornton [2002] finds only higher education and married couples to have a significantly negative effect on mortality. Significantly positive effects emanate from cigarette and alcohol consumption, insignificantly positive ones, from the crime rate and percent of population employed in manufacturing. With HCE insignificant, the study confirms the "flat-of-the-curve medicine" hypothesis.

However, studies based on OECD data tend to contradict this hypothesis. Zweifel and Ferrari [1992] introduce two changes to the health production function. First, they take remaining life expectancy at ages 40 and 65 as their dependent variable, arguing that it is especially longevity in retirement that creates problems for the financing of health care. Second, they account for lagged HCE per capita (with a lag of 10 years due to data availability) on the grounds that health status is not so much influenced by current but past medical interventions. Since health is likely to be exposed to similar unobserved shocks across OECD countries, they run a seemingly unrelated regression (SURE) on 1980 data. They find a significant elasticity of remaining life expectancy w.r.t. lagged HCE of 0.11.

Miller and Frech [2000] relate life expectancy at birth, at age 40, and age 60 to pharmaceutical and non-pharmaceutical HCE, cigarette and alcohol consumption, animal fat consumption, and the share of women in the population (all variables in logarithms), using 1996 OECD data. They find that a one percent increase of pharmaceutical expenditure results in a 0.02 percent increase of remaining life expectancy at the age of 40 and even a 0.04 percent increase at the age

of 60. Furthermore, the marginal effect of pharmaceutical consumption is greater for females than for males. By way of contrast, nonpharmaceutical HCE does not seem to have a significant impact on remaining life expectancy.

Shaw et al. [2005] estimate a health production function using OECD data for the year 2000. In view of small sample size, they use residual maximum likelihood and estimate a random effects rather than a fixed effects model (with country dummies).[1] An important extension to previous studies is the inclusion of a country's age distribution in an attempt to avoid the problem of reverse causality, with an older population consuming more HCE. When the age distribution is entered, a one percent increase in pharmaceutical expenditures is estimated to increase life expectancy at age 40 by 0.03 percent (0 percent otherwise).

In their re-estimation of Zweifel and Ferrari [1992], Zweifel et al. [2005] use an OECD panel data set. Taking into account that HCE figures may be driven up by mortality (caused by high HCE of individuals in their last year of life) and testing for the appropriate lag for HCE (which turned out to be 10 years again), they estimate an elasticity of remaining life expectancy at age 65 w.r.t. lagged HCE of 0.06 for females and 0.07 for males. However, these values are dominated by GDP (an indicator of nonmedical inputs), its elasticity being 0.12 and 0.08, respectively.

Summing up, the "flat-of-the-curve" hypothesis cannot be maintained in its strict sense, stating that additional medical inputs have no discernible effect on health status in the aggregate. Still, the available evidence suggests that nonmedical inputs may be more effective than medical ones in improving health of the population at large, calling for a reallocation of resources e.g. in favor of education to the detriment of health. However, an increasing share of GDP devoted to the healthcare sector might be justified if individuals not only value higher life

[1] Residual maximum likelihood produces unbiased estimates of the conditional variance components by correcting the usual maximum likelihood estimator for the degrees-of-freedom loss associated with estimating the conditional mean (Patterson and Thompson [1971]).

expectancy but also a reduced uncertainty of premature death, thanks to HCE. The existing evidence regarding improved control over one's health status is summarized in the following section.

2.2 Evidence on increased control over health status

The ideal of western lifestyle presumably is to live in perfect health, followed by sudden death. To the extent that individuals are successful in pursuing this ideal, premature death is avoided, resulting in the well-known rectangularization of the survival curve. The conventional wisdom is predicated on a biological limit to life. However, this age limit may well move over time. This calls for a measure of concentration that is invariant to the length of life. The Gini coefficient satisfies this requirement (see Section 3 below). Yet studies focusing on the variability of (or conversely, control over) health status have used indicators failing this requirement, except for the study by Shkolnikov et al. [2003].

Heligman and Pollard [1980] analyze Australia's age-specific mortality and its development over time. They distinguish infant mortality, excess mortality among young adults, and "pure" age-related mortality. They identify a variance parameter in the infant mortality component and trace the development of this parameter for both genders from 1946 to 1972, concluding that it decreased more markedly than general age-related mortality. The same method is applied by the Swiss Federal Statistical Office [1996] to Swiss data. Between 1876 and 1973, the variance parameter in the infant mortality component decreased sharply for both genders, followed by a slight increase for females between 1973 and 1993. On the whole the study confirms the result of Heligman and Pollard [1980].

Wilmoth and Horiuchi [1999] use the interquartile range [2] of age at death as an indicator of variability in length of life. Applying this measure to Sweden, Japan, and the United States they find a marked decrease. Between 1901 and 1995, the interquartile range fell from 46.4 to 15.5 years in Sweden and from 46.9 to 19.1 years in the United States. In Japan it decreased from 23 to 15.2 years between 1951 and 1995. Whereas the authors attribute the reduction of variability until the 1950s to lower infant mortality rates, they claim decreased mortality rates at older ages to be crucial since.

Shkolnikov et al. [2003] use life table information from several industrial countries to estimate differences in longevity. Emphasizing the analogy between the distribution of life years and income, they favor the Gini coefficient as a concentration measure. The lower the Gini coefficient, the more equal a distribution; in the present context, this means that x percent of life years are enjoyed by approximately x percent of the population. Conversely, this implies that death is heavily concentrated among the aged within a given population. The authors find marked differences in Gini values between countries, suggesting different degrees of uncertainty with regard to survival and hence health status.

The present study builds on the work of Shkolnikov et al. [2003] by using the Gini coefficient as an indicator of health status uncertainty. It follows Heligman and Pollard [1980] by tracing the development of their indicators for both genders over time. However, it goes beyond these contributions by asking the question whether this development is related more to medical or nonmedical inputs to the production of health, thus generalizing the approach adopted by the literature cited in section 2.1.

[2] This is the difference between the ages where the survival curve crosses the third and the first quartile of the age distribution.

3 Measuring uncertainty with regard to length of life

The Gini coefficient is traditionally used for the analysis of inequality in the income and wealth distribution (Atkinson [1970]). It is defined as the area between the diagonal and the Lorenz curve, divided by the whole area below the diagonal. The Lorenz curve in turn represents the cumulative income share as a function of the cumulative population share (Lorenz [1970]). Since the Gini coefficient is mean-independent, it is an ideal indicator for measuring inequality (or variability, respectively) when the quantity of interest changes over time (Sen [1973], ch. 2). It is therefore suited to measure variability in the length of life. Following Hanada [1983], the Gini coefficient can be applied to the length of life as follows. Let x be years lived rather than income. In order to measure the number of years lived, the person's death must be observed. Therefore the density function of x is redefined as

$$f_{xi} = \frac{d_{xi}}{l_0}, \tag{1}$$

with d_{xi} denoting the number of deaths at age x of cohort i and l_0 the number of survivors at year 0 (the size of the cohort). To simplify the discussion, the following analysis is limited to one cohort. Thus, the cumulative distribution function can be written as

$$F_x = \sum_{x=0}^{n-1} f_x. \tag{2}$$

It defines the horizontal axis of Figure 1, with n denoting the oldest age in the life table. The share of the total amount of years lived by the share F_x of the population is

$$\Phi_x = \sum_{x=0}^{n-1} \left(\frac{d_x x}{\sum_{x=0}^{n-1} d_x x}\right), \tag{3}$$

representing the vertical axis of Figure 1. The Lorenz curve is defined over $[0, 1]$, the range of F_x. In a situation of perfect equality, the share of the population F_x coincides with its share in the total of life years lived, Φ_x. Therefore, the Lorenz curve runs diagonal in this case, from points $(0, 0)$ to $(1, 1)$. The higher the variability in years lived across a population, the greater the divergence between the diagonal and the Lorenz curve.

Figure 1 below displays Lorenz curves for Portugal for 1960, 1980, and 2004 based on the Human Mortality Database [2008]. They approach the diagonal, indicating that more individuals die around the same age. However, the Lorenz curve can also be interpreted as an indicator of uncertainty (Davidson [2008]).[3] A Portuguese born in 1960 would have faced a situation of great uncertainty, because about 20 percent of that cohort already died by the age of 52, or conversely only 80 percent could count on living at least 52 years. By 1980, the distribution of life years had approached a situation where most people died at the same age (around age 78). Now, 80 percent of the cohort could count on living at least 62 years, indicating less uncertainty. And by 2004, 80 percent of the cohort is predicted to live at least 69 years - a prospect that corresponds to an almost perfect rectangularization of the survival curve.

Recall that the Gini coefficient is defined as the area between the diagonal and the Lorenz curve divided by the area under the diagonal. Noting that the total area below the diagonal of Figure 1 is 0.5 and integrating the areas stepwise[4], one obtains for the Gini coefficient for the distribution of length of life, using eqs. (2) and (3),

$$G = \frac{\frac{1}{2}\sum_{x=0}^{n-1}(F_x - F_{x+1})(F_x - \Phi_x + F_{x+1} - \Phi x + 1)}{\frac{1}{2}}, \tag{4}$$

[3] Saying that the Lorenz curve of the distribution $F(\cdot)$ Lorenz dominates the Lorenz curve of the distribution $G(\cdot)$ is equivalent to saying that $F(\cdot)$ is the less risky distribution.

[4] The area between the diagonal and the Lorenz curve can be divided into trapezes.

or

$$G = \sum_{x=0}^{n-1} (F_x - F_{x+1})(F_x - \Phi_x + F_{x+1} - \Phi x + 1). \tag{5}$$

[Figure 1 about here]

The Gini coefficient varies between 0 (perfect equality and hence minimum uncertainty) and 1 (perfect inequality and hence maximum uncertainty). It is equal to 0 if all individuals of a cohort die at the same age (live to the same age, respectively) and equal to 1 if everyone dies at age 0 while one individual dies at the maximum age. Using eq. (5), Gini coefficients are calculated for 24 countries between 1960 and 2004. Gini coefficients of all countries decrease over time indicating that variability of age at death declined (see Tables 4 to 6 in the Appendix but also Figure 2). The maximum drop, from 0.21 in 1960 to 0.10 in 2004, is found for Portugal. The top and bottom five countries are listed in Figures 4 and 5 (see Appendix) for the years 1960 and 2003. The countries with least variability were all Scandinavian (plus the Netherlands) in 1960, while Portugal had maximum variability. By 2003, three of the five low-variability countries were still Scandinavian, while Hungary had taken the place of Portugal, followed by the United States.

Three more findings are worth mentioning. First, Figure 2 below shows that in 1960, the Italians, the Portuguese, and the Japanese faced a higher longevity risk than U.S. citizens. However, this ranking has changed since. By 2004 Americans faced a considerably higher risk with regard to length of life than the citizens of these countries. Second, the fall of the Gini coefficients tends to slow down, most visibly in the 1980s (Figure 2 is fairly typical of Tables 4 to 6). Third, Gini coefficients exhibit a similar pattern of decrease for females and males (see Tables 5 and 6), with a systematic difference in favor of females in all countries sampled, however.

[Figure 2 about here]

In all, there is clear evidence suggesting that individuals in industrial countries have been exposed to less uncertainty regarding their longevity (and presumably health status) since 1960. This observation naturally gives rise to the question of what may have contributed to better control over health status. Figure 3 suggests that factors influencing the expected value of health status (proxied by life expectancy at birth) may also influence its variability (proxied by the Gini coefficient) since countries with higher life expectancy correspond to countries with lower Gini coefficients.

[Figure 3 about here]

4 Econometric specification, data, and variable definitions

For the econometric specification it is important to note that observations are available on the same country, resulting in a panel data set. This calls for an estimating equation of the following (linear) form,

$$y_{it} = \beta x_{it} + c_i + u_{it}. \tag{6}$$

In the present context y_{it} denotes the Gini coefficient of country i in year t, β a vector of coefficients to be estimated, x_{it} a set explanatory variables, , c_i a country-specific effect (specified in more detail below), and u_{it} a stochastic error term. Estimating eq. (6) with pooled ordinary least squares (OLS) entails two problems. First, the c_i component of the error term may be correlated with elements of x_{it}, resulting in biased estimates of β. Second, neglecting the two sources of stochastic risk causes OLS to attribute too little of total variance in y_{it} to the error term. Therefore, eq. (6) is estimated using the fixed effects (FE) or the random effects (RE) specification. The first consists in making the c_i an element of the x_{it} vector by inserting a

set of country-specific dummies. Alternatively, the c_i can be netted out by measuring all variables as differences from the country-specific means. The second approach assumes the c_i to be stochastic, which means they must be uncorrelated with the x_{it} for unbiased estimation of β. Moreover, the c_i and u_{it} components of the error term are assumed to be uncorrelated as well. RE is more parsimonious and hence more efficient than FE estimation. A generally accepted way of choosing between FE and RE is running a Hausman [1978] test. The Hausman test checks a more efficient model against a less efficient but consistent model to assure that the more efficient model also gives consistent results (Verbeek [2004], ch. 10).

Four further issues need to be clarified. First, variability of health status may feed back to HCE, one of the x variables. Countries where individuals face higher uncertainty with regard to longevity may spend more on health than countries where individuals face less uncertainty. Second, such a feedback would likely occur through the political process, in analogy to the feedback relationship found by Zweifel et al. [2005]. But then, the debate revolves around the health share in the GDP *(HCE/GDP)* rather than HCE itself. This calls for entering *(HCE/GDP)* as a regressor. However, the Durbin-Wu-Hausman test (Durbin [1954], Wu [1973], Hausman [1978]) for endogeneity does not reject the null hypothesis of exogeneity of $(HCE/GDP)_{-5}$ as well as of HCE_{-5} at the one percent level. Third, immediate effects of nonmedical inputs on the dependent variable are unlikely. Alcohol consumption, for instance, does not reduce time to death immediately but rather over the course of years. Thus, lifestyle variables are lagged 10 years. Medical inputs are lagged 5 years on the grounds that technological change in medicine occurs at such a rapid pace that interventions farther back in one's lifetime are not relevant for the variability of health status anymore. Fourth, squared variables were included to permit variable elasticities. They proved nonsignificant, however. The choice of variables is based on the empirical findings of the literature review in Section 2. Due to data availability, only the

following are included in the model that will be estimated using both RE and FE (predicted partial effects in parentheses),

$$GINI_{it} = \underset{(-)}{\beta_1 HCE_{it}} + \underset{(-)}{\beta_2 HOSPBED_{it}} + \underset{(-)}{\beta_3 GDP_{it}} + \underset{(+/-)}{\beta_4 POP65_{it}} + \underset{(+)}{\beta_5 ALC_{it}} + c_i + u_{it} \quad (7)$$

All variables are in logarithms, permitting coefficients to be interpretable as elasticities. In this way the coefficients can be easily interpreted as elasticities.

- *GINI:* Gini coefficient of the distribution of length of life, calculated according to eq. (5).
- HCE_{-5}: HCE per capita in 1,000 USD, measured at purchasing power parity. Devoting more resources to health-care is expected to enhance control over health status, reflected in a lowered value of *GINI*.
- $HOSPBED_{-5}$: Number of hospital beds per 1,000 inhabitants. With hospital stays usually triggered by severe health problems that might jeopardize survival, better access to hospital beds is predicted to enhance control and hence lower *GINI*.
- GDP_{-10}: GDP per capita in 1,000 USD, measured at purchasing power parity. This variable reflects two things. First, control over health status is quite likely a normal good, the demand for which increases with average income, ceteris paribus. Second, average income is importantly determined by labor productivity. To the extent that non-market productivity develops in a similar way, a higher per-capita GDP reflects a population that is better able to control their health status, resulting in a lower *GINI* value.
- $POP65_{-10}$: Percent of population over 65. Individuals past 65, being in retirement, may at first have more time available to invest in stabilizing their health status. This advantage probably is balanced after a few years by a decreasing effectiveness of their efforts, resulting in an increasing variability of health status and hence *GINI*. On the

other hand, a high share of individuals reaching age 65 or more ten years previously may indicate a population "purged" of individuals unable to avoid large negative shocks to their health status, thus composed of survivors who successfully control their health status. Therefore, the sign of *POP65* is ambiguous.

- ALC_{-10}: Alcohol consumption in liter per capita. While alcohol consumption is associated with a reduced remaining life expectancy, its effect on its variability is not established. Still, one may argue that it undermines individuals' capability to stabilize health. The predicted effect on *GINI* is therefore positive.

Data for the dependent variable is obtained from the Human Mortality Database [2008] (HMD), and for the regressors from the OECD [2007]. The latter source is known for its problems. One of them is national differences with regard to the delimitation of the healthcare sector, resulting in different baskets of benefits, another, the lack of comparability and precision of healthcare deflators. The first difficulty is avoided by controlling for unobserved country-specific effects, the second, by expressing healthcare expenditure in USD purchasing power parity.

Spanning the years 1960 to 2004 and including 24 OECD countries (Greece, Ireland, Mexico, Poland, South Korea, and Turkey had to be excluded entirely), the data set comprises 1,080 observations on the dependent variable, *GINI*. However, due to missing values in the OECD health data base, the panel is unbalanced.

The descriptive statistics are summarized in Table 1 below. The focus on industrial countries explains the low overall mean of the *GINI* value already in 1960. *HCE* steadily increased over time, reaching a mean of 2,210 USD in the year 2000. Interestingly the number of hospital beds per 1,000 inhabitants declined. This is mainly due to data availability, since those countries with a low number of hospital beds (such as Czech Republic, Portugal, and Spain) reported

their figures only recently. The share of population over 65 confirms the demographic trend of an aging population, and the decrease of alcohol consumption between the period 1980 and 2000 points to a healthier lifestyle in industrial countries.

[Table 1 about here]

5 Estimation results

5.1 Variability of life expectancy

This section is devoted to the econometric estimation of eq. (7). Both the RE and FE estimation were found to suffer from positive serial correlation. One way to deal with serial correlation is to estimate RE and FE with a first-order autoregressive error term [AR(1) process].[5] When reestimating eq. (7) accordingly both for the total population and separately for the two genders, the Hausman test prefers RE over FE throughout at the five percent significance level. Thus only the results of the RE estimations are presented in Table 2 below.

[Table 2 about here]

The results are similar across the three estimations. However, the effect of HCE_{-5} on the Gini coefficient of length of life is estimated to be higher for females than for males, suggesting that medical inputs are more effective in the reduction of health variability in the female population. By way of contrast, it is there that $HOSPBED_{-5}$ fails to attain statistical significance. More surprisingly still, $HOSPBED_{-5}$ is positively related to the Gini coefficient in two out of

[5] The AR(1) process calls for a two step procedure. First, the model is estimated using FE. Second, the autocorrelation coefficient, ρ, is estimated from the residuals through $\widehat{u}_{it} = \rho\widehat{u}_{i,t-1} + \epsilon_{it}$. Finally, the transformed model $y^*_{it} = \beta x^*_{it} + c_i + u_{it}$ can be estimated either with FE or RE, where $y^*_{it} = y_{it} - \rho y_{i,t-1}$ and $x^*_{it} = x_{it} - \rho x_{i,t-1}$ (for further details see Greene [2008], ch. 19). Another way to deal with serial correlation is to estimate the model using OLS but to correct the standard errors. The results are similar to AR(1) estimation and relegated to the Appendix (see Table 7 in the Appendix).

three estimations, contradicting the theoretical expectations stated in Section 4. There are two possible explanations. First, there may be reverse causality, with countries characterized by high variability in length of life investing in hospital beds. However, the Durbin-Wu-Hausman test does not point to endogeneity of $HOSPBED_{-5}$. Second, the variability-reducing effect of $HOSPBED_{-5}$ could already be captured in the variable HCE_{-5}. Hospitals might be assigned the very sick where not much can be done to regain control over health status. This argument is supported by an estimation excluding $HOSPBED_{-5}$ [column (4)]. There, the estimated elasticity of HCE_{-5} is markedly higher in absolute value.

As expected, GDP_{-10} has a reducing impact on the variability of longevity, with a ceteris paribus elasticity of 0.03, which however is lower than the 0.05 of HCE_{-5}. For instance, a 10 percent higher GDP per capita is estimated to lower variability of longevity by 0.3 percent compared to almost 0.5 percent due to *HCE*. Next, the variable $POP65_{-10}$ contributes to a reduction rather than increase of the dependent variable. Apparently the "survival of the fittest" effect exceeds the "loss of control over health" effect. However, the variable is only significant on the 10 percent level in two out of four estimations. Alcohol consumption is significant across all four estimations. A reduction by 10 percent results in a 0.3 percent decrease of the Gini coefficient ten years later. To sum up, both medical and nonmedical inputs reduce the variability of life expectancy. Finally, note that a double-logarithmic formulation necessarily implies decreasing marginal returns if the estimated elasticity is negative[6].

A major drawback of estimations (1) to (3) in Table 2 is the high number of missing values, mainly due to $HOSPBED_{-5}$. Its exclusion from (4) thus serves to check for the importance of missing values. First, the Hausman test again prefers the RE over FE specification. Second, estimated coefficients remain stable (with the exception of $POP65_{-10}$ being no longer signifi-

[6] general terms $\frac{\partial}{\partial X}e(Y,X) = \frac{\partial^2 Y}{\partial X^2}\frac{X}{Y} + \frac{\partial Y}{\partial X}\frac{1}{Y} = 0$ by assumption. Solving for $\frac{\partial^2 Y}{\partial X^2}$ and expanding by $\frac{X}{Y}$, one obtains $\frac{\partial^2 Y}{\partial X^2} = -e(Y,X)\frac{Y}{X^2} > 0$, i.e. decreasing marginal returns in the present context.

cant). Third, HCE per capita continues to have a higher impact on the Gini coefficient than GDP per capita, an indicator of nonmedical inputs.

5.2 Life expectancy itself

Equation (7) was essentially borrowed from Zweifel et al. [2005] (see Section 2.1 above), where remaining life expectancy at age 65 constituted the dependent variable. At the end of Section 3, the tentative hypothesis was stated that the same factors determining the expected value of health also influences its variability, eq. (7) should perform well in explaining life expectancy although the present study uses HMD in addition to OECD data. Still, a few adjustments are necessary to ensure comparability with Zweifel et al. [2005]. First, the variable GDP is treated as reflecting a budget constraint there, which calls for replacing GDP_{-10} by GDP without a lag. Second, $HOSPBED_{-5}$ is excluded[7]. Third, specification tests preferred a quadratic functional form without logarithms.

[Table 3 about here]

Table 3 exhibits only the RE results since for all three estimations the Hausman test again prefers RE over FE. In col. (1), life expectancy at birth for both genders is the dependent variable, whereas in cols. (2) and (3), it is the gender-specific value at age 65. The effects of GDP and HCE_{-5} are significant in all three specifications. For a comparison with Table 2, elasticities evaluated at the means are provided (see the values in brackets of Table 3; values in italics are copied from Tables 2 and 8). Three things are noteworthy. First, the same factors that were found to decrease (increase) the variability of life expectancy indeed are estimated to increase (decrease) its expected value. Second, whereas GDP is less effective than HCE in reducing the variability of longevity, it is more effective in increasing its expected value (which

[7] The variable also proved nonsignificant when included.

is in accordance with most studies on the production of health). Third, HCE has decreasing marginal returns both as an instrument for controlling variability of health status and enhancing its expected value. However, the marginal effectiveness of nonmedical inputs seems to dominate that of the medical ones in both respects.[8]

6 Conclusion

This study addresses an issue that seems to have been overlooked in health economics with its exclusive focus on the determinants of the expected level of health. However, for risk-averse individuals, variability of health also is important. This raises the question of how variability of health status has developed over time and whether the finding of "flat-of-the-curve medicine" (i.e. low marginal returns to healthcare expenditure) carries over. The Gini coefficient of life expectancy serves as an indicator of uncertainty concerning health status. A value of zero indicates that everyone dies at the same age, i.e. minimum variability. Between 1960 and 2004, it decreased for all 24 OECD countries sampled, pointing to improved control over individuals' health status. Next, the Gini coefficient is related to inputs to the production of health. Taking account of hidden heterogeneity through a random effects specification and for first-order-autocorrelation with the lagged residual, nonmedical inputs are found less important in reducing the variability of health status than medical ones, measured by healthcare expenditure per capita five years before. For comparison with the existing literature, a conventional production function with the level of life expectancy is also estimated. The results replicate both the findings with regard to the variability and (from earlier studies) the expected value of life expectancy suggesting that medical inputs exhibit decreasing marginal returns.

[8] From column (1) of Table 3, one obtains the critical value beyond which e(LE, HCE) decreases: $\frac{\partial LE}{\partial HCE} = 0.747 - 2 \cdot 0.157 HCE = 0$. This yields HCE=2.38 or 2,379 USD respectively. For women, one obtains 1,870 USD, and for men 2,468 USD. These values are in the same range as those in Zweifel et al. [2005].

There are limitations to this study that need to be pointed out. First, the variability of health status is only crudely measured by the Gini coefficient of life expectancy. Data at the individual level such as quality-adjusted life years (QALYs) would be more informative. Second, the macroeconomic approach severely constrains the choice of variables in the econometric model. Additional determinants such as education, innovation in health care, and additional lifestyle variables might not only influence the level of health but also its variability. Finally, to derive possible welfare gains of a reduced variability of health status, the empirical model needs to be related more closely to the theory of the production of health (e.g. to the modified Grossman model by Picone et al. [1998]).

However, the finding that not only expected health status but also its variability can be influenced already has important implications. Reduced uncertainty about age at death likely has been modifying the decisions especially of older individuals concerning savings, consumptions, and the purchase of life and long-term care insurance. Quite generally, it helps risk-averse individuals to optimize lifetime consumption, permitting them to reduce precautionary saving (see Palumbo [1999] but also Levhari and Mirman [1977]). To the extent that health care services serve to improve control over health status, "flat-of-the-curve medicine" need not be wasteful.

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Appendix

Figure 1: Lorenz curves for length of life, Portugal

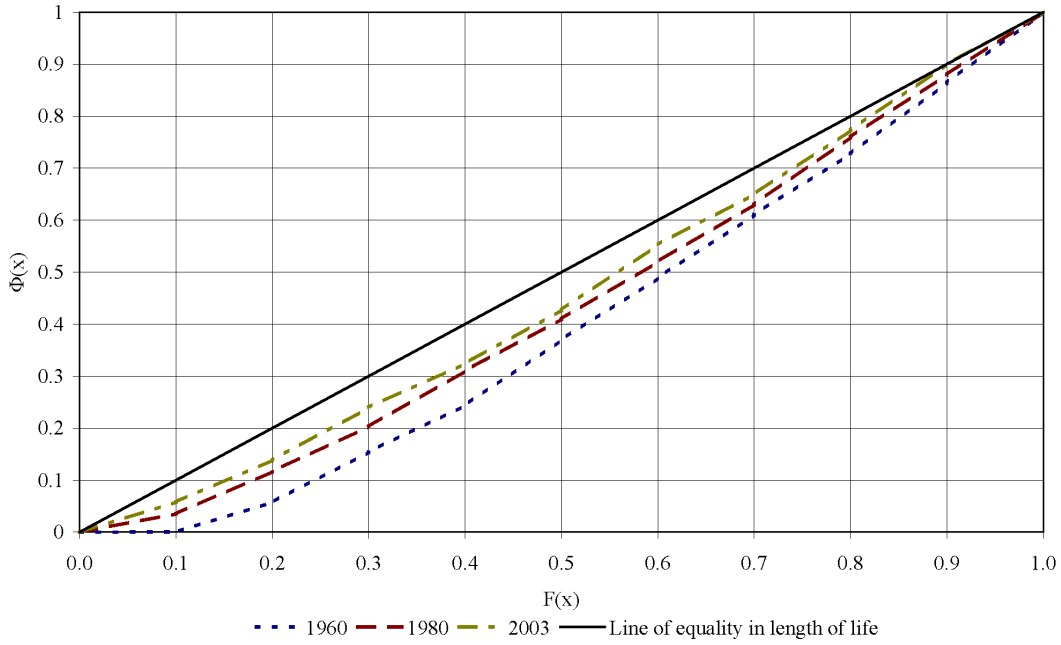


Figure 2: Gini coefficients for the US, Japan, Italy, and Portugal, 1960-2004

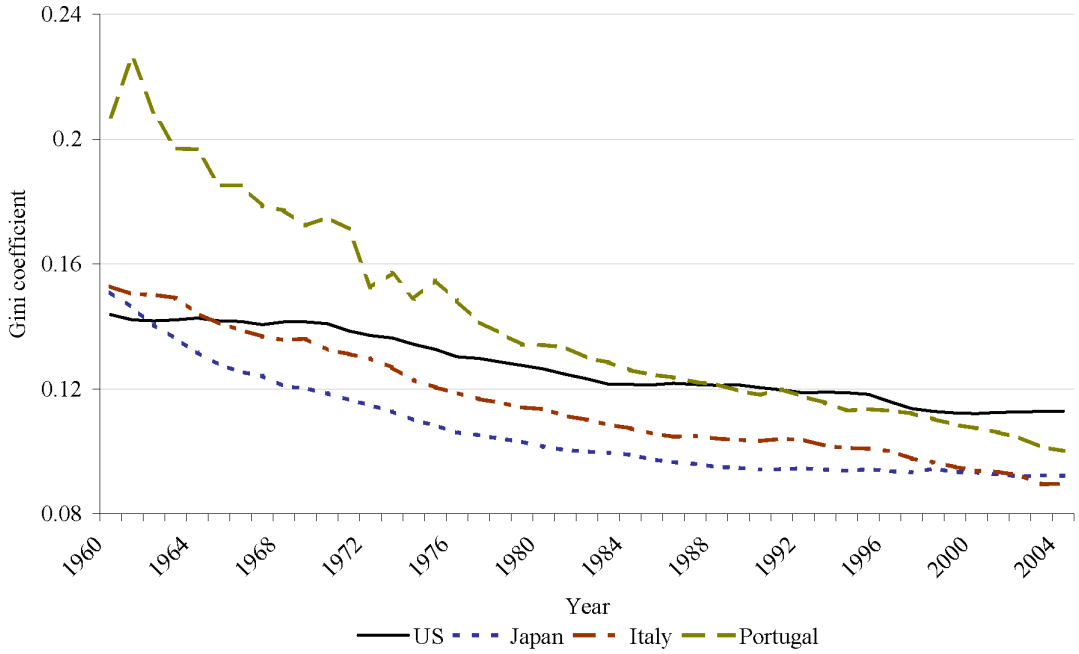
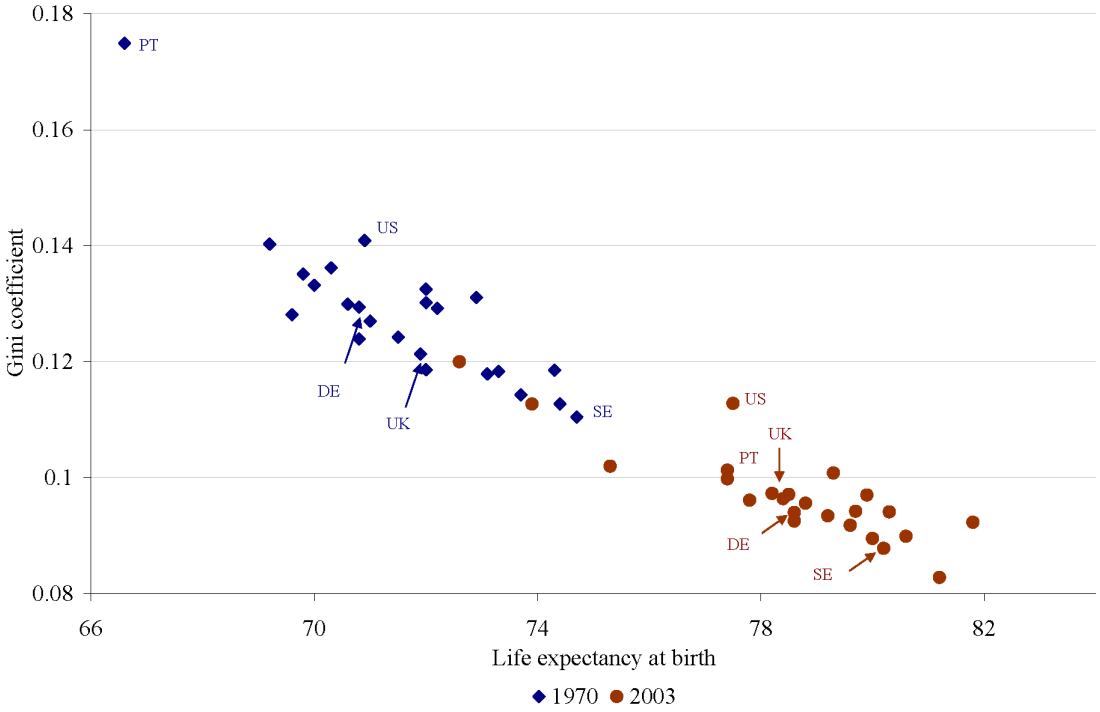


Figure 3: Gini coefficient and life expectancy for 1970 and 2003



Country codes: DE=Germany, PT=Portugal, SE=Sweden, UK=United Kingdom, US=United States

Table 1: Descriptive statistics of variables, selected years

Variable	Mean	1960	1980	2000	s.d.[a]	N
GINI	0.12	0.14	0.12	0.10	0.02	1,080
HCE	1.20	0.01	0.70	2.21	1.02	835
HOSPBED	6.93	9.05	7.48	6.27	2.53	456
GDP	13.74	1.99	10.02	26.11	10.35	999
POP65	12.50	9.44	12.50	14.75	2.71	1,078
ALCOHOL	10.62	7.87	11.92	9.98	3.66	1,003

Note: See text for definitions

[a]s.d.= Standard deviation

Table 2: Determinants of the Gini coefficient for 24 OECD countries, 1960-2004

Explanatory variable	Predicted sign	Total (1)	Females (2)	Males (3)	Total (4)
HCE_{-5}	-	-0.0481***	-0.0518***	-0.0411***	-0.0544***
		(0.0105)	(0.0112)	(0.0119)	(0.0010)
$HOSPBED_{-5}$	-	0.0483***	0.0218	0.0587***	-
		(0.0155)	(0.0161)	(0.0156)	
GDP_{-10}	-	-0.0309**	-0.0293**	-0.0367**	-0.0421***
		(0.0138)	(0.0147)	(0.0167)	(0.0129)
$POP65_{-10}$	+/-	-0.0555*	-0.0642*	-0.0414	-0.0204
		(0.0326)	(0.0341)	(0.0338)	(0.0337)
ALC_{-10}	+	0.0359***	0.0417***	0.0702***	0.0308***
		(0.0126)	(0.0132)	(0.0126)	(0.0106)
Constant		-2.223***	-2.311***	-2.274***	-2.184***
		(0.0967)	(0.1010)	(0.1030)	(0.0815)
ρ		0.902	0.880	0.868	0.855
Observations		297	297	297	607
R^2		0.887	0.882	0.878	0.891

*** p<0.01, ** p<0.05, * p<0.1

Notes: Standard errors are given in parentheses.

All variables are in natural logarithms.

Table 3: Determinants of remaining life expectancy for 24 OECD countries

	At birth		Age 65, Females		Age 65, Males	
	(1)		(2)		(3)	
HCE_{-5}	0.7470*	[0.012]	1.0810***	[0.066]	0.5430**	[0.041]
	(0.3930)	*-0.054*[a]	(0.2610)	*-0.056*[a]	(0.2540)	*-0.063*[a]
HCE^2_{-5}	-0.1570**		-0.2890***		-0.1100**	
	(0.0737)		(0.0479)		(0.0460)	
GDP	0.3780***	[0.089]	0.0600**	[0.059]	0.1560***	[0.190]
	(0.0388)	*-0.042*[a]	(0.0259)	*-0.035*[a]	(0.0251)	*-0.042*[a]
GDP^2	-0.0048***		0.0007*		-0.0011***	
	(0.0006)		(0.0004)		(0.0004)	
$POP65_{-10}$	1.0060***	[0.161]	0.1900**	[0.127]	0.6620***	[0.546]
	(0.1330)	*-0.02*[a]	(0.0836)	*-0.049*[a]	(0.0898)	*-0.01*[a]
$POP65^2_{-10}$	-0.0366***		-0.0023		-0.0248***	
	(0.0053)		(0.0033)		(0.0036)	
ALC_{-10}	-0.3120***	[-0.039]	-0.0326	[-0.017]	-0.2630***	[-0.174]
	(0.0865)	*0.031*[a]	(0.0556)	*0.034*[a]	(0.0564)	*-0.036*[a]
ALC^2_{-10}	0.0031		0.0019		0.0056**	
	(0.0037)		(0.0024)		(0.0024)	
Constant	66.811***		14.393***		9.787***	
	(0.7530)		(0.5120)		(0.5040)	
ρ	0.925		0.924		0.864	
Observations	461		433		462	
R^2	0.915		0.895		0.892	

*** p<0.01, ** p<0.05, * p<0.1

Notes: Standard errors are given in parentheses.

Values in brackets are elasticities evaluated at the means.

[a] Elasticity taken from corresponding col. of Tables 2 and 8.

Table 4: Development of the Gini coefficient over time for 24 countries

Country	1960	1970	1980	1990	2004
Australia	0.1315	0.1294	0.1161	0.1061	0.0932
Austria	0.1478	0.1332	0.1199	0.1068	0.0951
Belgium	0.1368	0.127	0.1160	0.1066	0.0964
Canada	0.1388	0.1311	0.1181	0.1067	0.0963
Czech Republic	0.1264	0.1281	0.1193	0.1166	0.1009
Denmark	0.1211	0.1183	0.1142	0.1110	0.0992
Finland	0.1337	0.1239	0.1120	0.1098	0.0990
France	0.1377	0.1292	0.1196	0.1116	0.1004
Germany	0.1404	0.1299	0.1158	0.1037	0.0930
Hungary	0.1524	0.1403	0.1351	0.1364	0.1198
Iceland	0.1185	0.1185	0.1099	0.1045	0.0875
Italy	0.1528	0.1325	0.1135	0.1034	0.0897
Japan	0.1509	0.1186	0.1015	0.0942	0.0922
Luxembourg	0.1401	0.1362	0.1153	0.1096	0.0957
Netherlands	0.1162	0.1143	0.1063	0.1002	0.0914
New Zealand	0.1243	0.1242	0.1176	0.1125	0.0934
Norway	0.1188	0.1127	0.1060	0.1047	0.0916
Portugal	0.2071	0.1749	0.1340	0.1181	0.1002
Slovakia	0.1356	0.1351	0.1278	0.1255	0.1106
Spain	0.1542	0.1302	0.1099	0.1071	0.0936
Sweden	0.1144	0.1105	0.1048	0.0982	0.0877
Switzerland	0.1257	0.1179	0.1087	0.1032	0.0899
United Kingdom	0.1255	0.1213	0.1127	0.1053	0.0956
United States	0.1438	0.1409	0.1264	0.1204	0.1129

Table 5: Development of the Gini coefficient over time for 24 countries, females

Country	1960	1970	1980	1990	2004
Australia	0.1187	0.1155	0.1012	0.0937	0.0828
Austria	0.1300	0.1154	0.1018	0.0909	0.0814
Belgium	0.1202	0.1119	0.1019	0.0934	0.0838
Canada	0.1234	0.1159	0.1045	0.0947	0.0877
Czech Republic	0.1109	0.1105	0.1020	0.0976	0.0848
Denmark	0.1107	0.1070	0.1038	0.1017	0.0907
Finland	0.1135	0.1018	0.0908	0.0897	0.0819
France	0.1213	0.1109	0.0990	0.0905	0.0837
Germany	0.1254	0.1142	0.1011	0.0904	0.0819
Hungary	0.1393	0.1238	0.1168	0.1143	0.1004
Iceland	0.1045	0.0945	0.0928	0.0930	0.0833
Italy	0.1385	0.1176	0.0982	0.0883	0.0784
Japan	0.1402	0.1066	0.0895	0.0819	0.0794
Luxembourg	0.1196	0.1216	0.1075	0.0977	0.0823
Netherlands	0.1050	0.1011	0.0942	0.0903	0.0848
New Zealand	0.1133	0.1106	0.1083	0.1000	0.0855
Norway	0.1045	0.0957	0.0901	0.0912	0.0825
Portugal	0.1906	0.1571	0.1145	0.0981	0.0825
Slovakia	0.1208	0.1159	0.1077	0.1024	0.0925
Spain	0.1413	0.1162	0.0953	0.0882	0.0772
Sweden	0.1043	0.0987	0.0921	0.0877	0.0800
Switzerland	0.1099	0.1017	0.0933	0.0880	0.0789
United Kingdom	0.1138	0.1102	0.1031	0.0961	0.0872
United States	0.1283	0.1248	0.1118	0.1063	0.1009

Table 6: Development of the Gini coefficient over timefor 24 countries, males

Country	1960	1970	1980	1990	2004
Australia	0.1398	0.1375	0.1238	0.1139	0.0990
Austria	0.1620	0.1462	0.1328	0.1171	0.1040
Belgium	0.1493	0.1373	0.1241	0.1138	0.1028
Canada	0.1507	0.1412	0.1269	0.1132	0.1017
Czech Republic	0.1383	0.1394	0.1304	0.1263	0.1103
Denmark	0.1303	0.1266	0.1193	0.1156	0.1035
Finland	0.1490	0.1389	0.1235	0.1216	0.1098
France	0.1485	0.1404	0.1316	0.1242	0.1091
Germany	0.1529	0.1413	0.1257	0.1117	0.1006
Hungary	0.1641	0.1532	0.1468	0.1484	0.1299
Iceland	0.1310	0.1373	0.1224	0.1124	0.0893
Italy	0.1647	0.1439	0.1237	0.1132	0.0961
Japan	0.1589	0.1272	0.1095	0.1018	0.0981
Luxembourg	0.1572	0.1462	0.1184	0.1154	0.1031
Netherlands	0.1256	0.1236	0.1126	0.1044	0.0939
New Zealand	0.1319	0.1323	0.1220	0.1200	0.0975
Norway	0.1309	0.1252	0.1164	0.1122	0.0964
Portugal	0.2211	0.1896	0.1494	0.1319	0.1115
Slovakia	0.1486	0.1494	0.1424	0.1379	0.1205
Spain	0.1647	0.1417	0.1209	0.1203	0.1043
Sweden	0.1230	0.1191	0.1120	0.1040	0.0914
Switzerland	0.1386	0.1297	0.1185	0.1127	0.0971
United Kingdom	0.1324	0.1261	0.1159	0.1088	0.0999
United States	0.1552	0.1523	0.1370	0.1308	0.1206

Table 7: Estimation of GINI with panel-corrected standard errors, FE specification

VARIABLES	Coefficient	z	P> z
HCE_{-5}	-0.0349	0.0150	0.0200
$HOSPBED_{-5}$	0.0446	0.0196	0.0230
GDP_{-10}	-0.0430	0.0176	0.0140
$POP65_{-10}$	-0.0372	0.0249	0.1360
ALC_{-10}	0.0716	0.0089	0.000
$Australia_d$	0.0141	0.0195	0.4690
$Belgium_d$	0.0162	0.0053	0.0020
$Canada_d$	0.0441	0.0189	0.0200
$Switzerland_d$	-0.0476	0.0112	0.000
$Czech\ Republic_d$	-0.0435	0.0145	0.0030
$Germany_d$	0.0384	0.0031	0.000
$Denmark_d$	0.0636	0.0122	0.000
$Finland_d$	0.0136	0.0093	0.1440
$France_d$	0.0165	0.0061	0.0070
$Hungary_d$	0.1290	0.0095	0.000
$Italy_d$	-0.0294	0.0086	0.0010
$Japan_d$	-0.0667	0.0144	0.000
$Luxembourg_d$	-0.0117	0.0130	0.3660
$Netherlands_d$	-0.0255	0.0138	0.0650
$Norway_d$	0.0518	0.0138	0.000
$Portugal_d$	0.0536	0.0215	0.0120
$Slovakia_d$	0.0366	0.0130	0.0050
$Spain_d$	-0.0308	0.0213	0.1480
$United\ Kingdom_d$	0.0158	0.0138	0.2520
$United\ States_d$	0.1910	0.0222	0.000
Constant	-2.340	0.121	0.000
Observations	297		
R-squared	0.965		

Notes: Not included are Iceland, New Zealand, and Sweden.

($_d$) denotes country dummies.

Table 8: GINI estimation excluding HOSPBED

	Total	Females	Males
VARIABLES	(1)	(2)	(3)
HCE_{-5}	-0.0544***	-0.0560***	-0.0634***
	(0.0100)	(0.0111)	(0.0121)
GDP_{-10}	-0.0421***	-0.0354**	-0.0422***
	(0.0129)	(0.0142)	(0.0154)
$POP65_{-10}$	-0.0204	-0.0486	-0.0069
	(0.0337)	(0.0359)	(0.0375)
ALC_{-10}	0.0308***	0.0344***	0.0364***
	(0.0106)	(0.0116)	(0.0124)
Constant	-2.184***	-2.276***	-2.159***
	(0.0815)	(0.0867)	(0.0908)
ρ	0.855	0.829	0.811
Observations	607	607	607
R^2	0.892	0.877	0.882

*** p<0.01, ** p<0.05, * p<0.1

Notes: Standard errors are given in parentheses

RE specification. The variables are in natural logarithms.

Figure 4: Top and bottom five Gini coefficients for 1960 (ranks in parentheses)

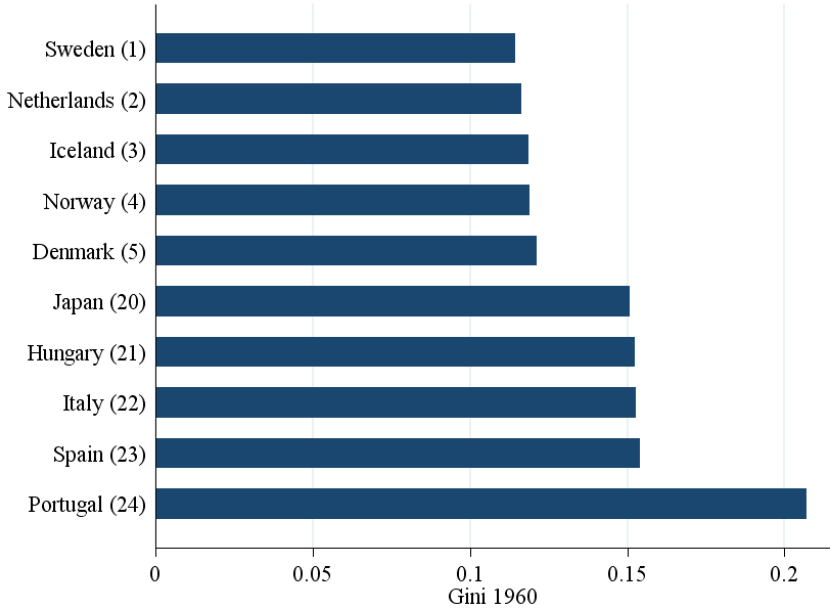
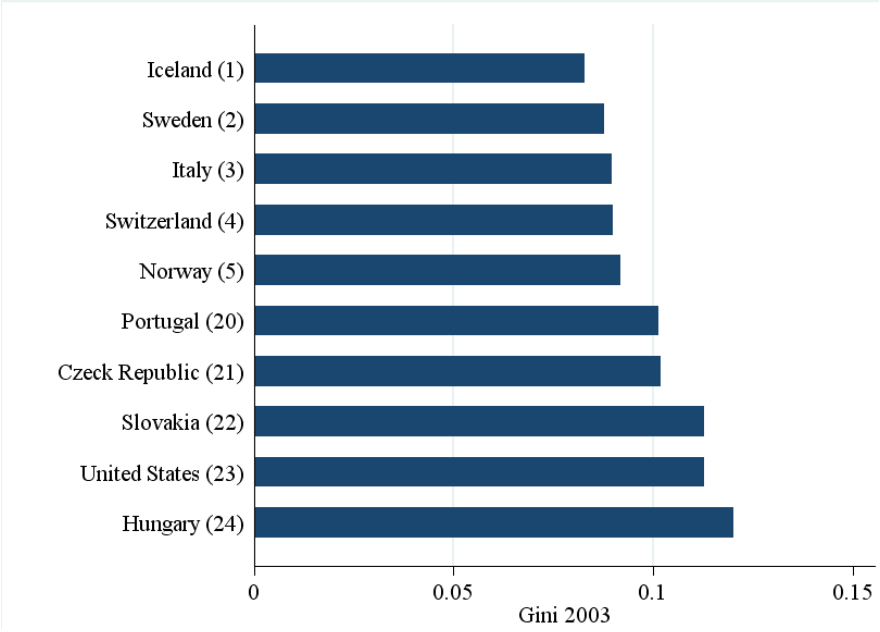


Figure 5: Top and bottom five Gini coefficients for 2003 (ranks in parentheses)



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