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Sandra Cavaco, Jean-Michel Etienne
and Ali Skalli

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Evidence from European older workforce surveys

Department of Economics

Identifying causal paths between health and socio-economic status: Evidence from European older workforce surveys[1]

Sandra Cavaco[a,b,*], Jean-Michel Etienne[c] and Ali Skalli[a]

[a] LEM, University Panthéon-Assas (Paris 2), 92, rue d'Assas 75270 Paris Cedex 06, France
[b] CCP, Aahrus School of Business, Prismet, Silkeborgvej 2, 8000 Aarhus C, Denmark
[c] ERMES, University Panthéon-Assas (Paris 2), 12, Place du Panthéon 75231 Paris Cedex 05, France

Abstract

It is well known that health and socio-economic status (SES) are linked through a bi-directional relationship. In addition, such a relationship takes place early in life and keeps on evolving over time so that both one's health and SES at a given point in time result from the cumulative effects of this spiral. Thus, only by simultaneously accounting for both pathways as well as for their dynamics would one be able to provide a clear picture of both the process of health accumulation and the dynamics of SES formation. We estimate a structural model where a variety of causal paths between different health dimensions and SES measures as well as their dynamics are simultaneously accounted for. This allows distinction between significant causal paths and insignificant ones, while accounting for endogeneity as well as for cofounders. We use the SOCIOLD survey where the targeted population is that of the older workforce (50 and older) from six EU countries (Denmark, Finland, France, Greece, the Netherlands and the UK). Our results show that (*i*) reverse causality is indeed a crucial issue: one's previous socio-economic status influences current health and previous health influences current socio-economic status, (*ii*) there are cumulative effects in the sense that both health and socio-economic statuses depend on their past values and (*iii*) the results are sensitive to whether simultaneity is explicitly accounted for or not.

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* Corresponding author: Postal address: LEM, University Panthéon-Assas (Paris 2), 92, rue d'Assas 75270 Paris Cedex 06, France; Tel.: +33 1 55 42 50 26; Fax: +33 1 55 42 50 24.
E-mail addresses: Sandra.Cavaco@u-paris2.fr (S. Cavaco), Jean-Michel.Etienne@u-paris2.fr (J.M. Etienne), skalli@u-paris2.fr (A. Skalli).

1. Introduction

Understanding the association between individual health and socioeconomic statuses is crucial for any analysis of the relationship between health and economic inequalities in general. But it is also very likely to shed light on a number of fundamental policy issues. Reducing socio-economic inequalities in health is indeed likely to result in an increase of individual well-being (van Praag and Ferrer-i-Carbonell, 2004; Veenhoven, 2007). In addition, a number of recent endogenous growth studies, both theoretical and empirical, suggest that health improvements are beneficial to economic growth (van Zon and Muysken, 2001; Bloom and Canning, 2003; Bloom et al., 2004; Gourdel et al., 2004). Actually, any improvement of the health of the population results in an increase of life expectancy which, in turn, positively influences well-being but also growth via its effect on population size. However, a tightly related issue is that of ageing which implies higher health expenditures (Fuchs, 1998) as well as an increasing burden on pension funding systems. In an endogenous growth framework, van Zon and Muysken (2001) simultaneously account for the productivity and the longevity growth effects of health and show that if the demand for health increases with the standard of living, then health and growth could become substitutes rather than complements. Actually, the ideal situation would be one where ageing is accompanied by a decline of the dependency ratio. Thus, although curative expenditures are indeed necessary in ageing societies, reducing health inequalities among members of the labour force could be seen not only as a preventive strategy but also as a means of slowing down the observed increase of the dependency ratio. One important reason why the slow down is likely to occur is the strong effect of health status on individual retirement decisions (Diamond and Hausman, 1984; Stern, 1996; Siddiqui, 1997; Mein et al., 2000; McGarry, 2002).

Equally important is the trade-off public authorities have to make as to the funding of the different sectors of the economy. Is it equally worth devoting more resources to health or to education, a sector which is also, if not more, contributive to economic growth?[2] This raises the issue of the competitiveness of the health sector[3] and makes the usual trade-off between efficiency and equity even more complicated. Illustrative enough with respect to this issue is the recurrent debate on how to promote equal access to health care while avoiding induced-demand problems or preventing health insurance contributions from creating market distortions via their effect on labour costs.

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[2] See Baumol (1967) for an early comparison of health and education as sources of growth.
[3] See for instance the European competitiveness report (European Commission, 2004, Chapter 3).

Since the seminal work by Grossman (1972), economists as well as epidemiologists agree upon the idea that the relationship between health and SES is bi-directional (Smith, 1999). The point of departure of Grossman's model is that the health stock individuals are endowed with deteriorates with age and that the rate of depreciation is exogenous. Thus, only by investing in health could they increase their health stock. As long as they are in the labour force, those who undertook the investment are more productive and are able to work more hours or more days. But they can also delay their exit from the labour market. Health has therefore an effect on individuals' SES. This is the so-called *selection* hypothesis which states that health status is an explanatory factor of individuals' position in the distribution of socio-economic statuses. Examples of empirical studies exploring the selection hypothesis include Diamond and Hausman (1984), Stern (1996), Siddiqui (1997), Bound et al. (1999), Fox and Goldblatt (1986), Marmot (1986), Currie and Madraen (1999), van de Mheen et al. (1999) among many others.

However, individuals' health statuses at any time as well as the investment they are able to undertake depend on their economic endowments. This is the so-called *causation* hypothesis which states that SES is also likely to influence health status. There are indeed a number of intermediate factors through which such an influence may take place. Examples are individuals' tastes and behaviour (Mackenbach et al.,1997; Stronks et al., 1997; Or, 2000; Muller, 2002; Osler et al., 2002; Shibuya et al., 2002; Sturm and Gresenz, 2002; Contoyannis and Jones, 2004), access to health care (Ayaniam et al.*,* 2000; O'Malley et al.*,* 2001), psycho-social factors such as the feelings of deprivation or loss of autonomy (Wilkinson, 1992; Marmot and Wilkinson, 1999; Berkman and Kawachi, 2000; Mellor and Milyo, 2001) or material living conditions as reflected by income (Ecob and Davey-Smith, 1999; Soobader and Le Clere, 1999; Fiscella and Franks, 2000; Deaton and Paxton, 2001), employment status (Or, 2000), working conditions (Askenazy, 2000), education (van Rossum et al., 2000; Everson et al., 2002), socio-demographic history (Grundy and Holt, 2000) or neighbourhood and living environment (Bosma et al.*,* 2001). Some authors have also explored the cumulative pattern of health accumulation along the life cycle (Wadsworth, 1986; Blane, 1999; Currie and Hyson, 1999; Case et al., 2002; Currie and Stabile, 2003; Case et al., 2005) while others have investigated the effect of childhood living conditions on adults' health (Elo and Preston, 1992; Blane et al., 1993; Kuh and Ben-Shlomo, 1997; Lynch et al., 1997; Lindeboom et al., 2006) or even intra-uterine socio-economic effects as well as genetic endowments (Smith and Kington, 1997; Behrman and Rosenzweig, 2004).

Two common features of this literature are worth highlighting. First, the dynamics of health accumulation over the life course is very seldom explicitly accounted for. Second, the coexistence of, and feedback effects between, the selection and causation mechanisms are never simultaneously considered. This is obviously a notable weakness as a basic corollary of Grossman's model is that health and socio-economic statuses continuously interact with each other over one's life course. It is this spiral which explains both health and socio-economic statuses of an individual at a given point of time and it is this spiral which researchers should investigate. Among the many advantages of such a strategy is the possibility it offers to infer age-specific and preventive policy recommendations.

The main reason why the various potential causal paths between health and socio-economic statuses are never simultaneously modelled is the difficulty one faces when attempting to unravel causal effects. Suppose one is interested in testing the selection hypothesis. S/he needs to observe some exogenous shock that influences socio-economic status only via its effect on health. A natural strategy could then consist in running experiences. Thomas et al. (2003) for instance observe the labour market outcome of a treated group of iron-deficient Indonesian citizens as compared to a control group having received no iron supplement. They provide strong evidence in favour of the selection hypothesis. Another strategy could rely on natural experiences. Arendt (2005) examines the effect of education on health in Denmark, the exogenous shock being simulated through the reform of compulsory schooling laws. Likewise, Lindhal (2005) explores the effect of income on health and mortality in Sweden, using lottery prizes as a source of exogenous variation. Both conclude in favour of the causation hypothesis.

An alternative approach is the one adopted by Adams et al. (2003) in their influential study. It consists of performing Granger-type causality tests using panel data. Suppose again one is interested in the selection hypothesis. The idea is that health has no causal effect on SES if the distribution of current socio-economic statuses, conditional on SES at time $t-1$ and socio-demographic characteristics at time $t-1$ is the same whether conditioned or not on health at time $t-1$. Hence testing is for the absence, not for the presence of a causal effect from health to SES. Using American data for the elderly (70 and older), they come with a rather weak evidence in favour of the selection hypothesis. They also show that socio-economic status has no direct causal effect on sudden onsets but does cause degenerative diseases such as mental

health problems and some chronic diseases.[4] One explanation the authors propose is that only partly does Medicare cover this type of pathology.

Though important, the study by Adams et al. (2003) has been criticized by a number of authors. For instance, Adda et al. (2003) have replicated their methodology using data from Great-Britain and from Sweden, including younger individuals as well. Their idea is that if Adams et al. (2003) explanation of the causation effect they highlight was correct, such an effect should not emerge from British and Swedish data as there is universal health insurance coverage in these two countries. Interestingly enough, their results plead in favour of the causation hypothesis. They interpret this result as being due to the effect of SES on health taking place early in one's life, a fact Adams et al. (2003) cannot explicitly take into account in an analysis focussing on the elderly. Likewise, Mealli and Rubin (2003) argue that SES at time $t-2$ is not accounted for while it is likely to influence both health and SES at time $t-1$. Such an effect cannot be captured through the sole conditioning on socio-demographic characteristics at time $t-1$. If such an effect is in place, then the measured effects are biased and the proposed tests, incorrect.

Actually, both criticisms could be avoided by adopting a structural form model rather than a reduced-form approach as is the case in Adams et al. (2003), but also in Arendt (2005) or Lindhal (2005). When modelling the selection hypothesis for instance, Adams et al. (2003) implicitly assume that past effects of health on SES are exhaustively accounted for by conditioning on past SES. Likewise, when testing the causation hypothesis, they assume that the past effects of health on SES are exhaustively taken into account via the conditioning on past health status.

It is this type of limitation which we aim at overcoming in this paper. Using older workforce data (50 and older) from six European countries, we estimate a structural model where a number of causal paths between health and SES are simultaneously and explicitly modelled. In addition, we use retrospective information on both health and SES to account for the dynamics of health accumulation and SES formation. Using Asymptotic Least Squares as a means of estimating our 5-equation structural model, we show that (*i*) both selection and causation effects are at play, (*ii*) there are indeed cumulative effects in the sense that both health and socio-economic statuses depend on their past values and (*iii*) the results are sensitive to whether simultaneity is explicitly accounted for or not.

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[4] The authors argue, however, that such a causal effect could be due to persisting confounders influencing both health and socio-economic status, e.g. genetic factors.

The paper is organized as follows. In section 2, we describe our data and empirical strategy. In section 3, we present the results and their interpretation. In section 4, we propose some concluding remarks.

2. Data and empirical model

2.1. Data

We use the so-called SOCIOLD survey which comprises 6 country-specific cross-sectional datasets, covering Denmark, Finland, France, Greece, the Netherlands and the United-Kingdom. The questionnaire has been designed by the partners of the EU-funded SOCIOLD research project and the survey conducted in 2004 by private interview companies in the different countries. Each of these circulated the questionnaire among internet users aged 50 or older, the targeted number of exploitable questionnaires being 500 in Finland and 1000 in each of the five remaining countries.[5]

The questionnaire was designed to help addressing those questions at the centre of the SOCIOLD project; namely:

➢ How does individual socio-economic and/or occupational status affect the physical and mental health and sense of well being of older individuals of working age?

➢ How does individual socio-economic and/or occupational status affect the ability of older workers to participate in the labour market?

Thus, the resulting data provide new information on a variety of dimensions of both health and SES. These include objective as well as subjective measures of respondents' health both at the time of interview and in the past so that a detailed description of both current health status and health history is available. Likewise, SES is described through a large number of indicators, including income, education, employment status, occupational status. Also useful is the available information about respondents' socio-economic family background as well as about their own socio-economic history. Moreover, for a number of life style indicators (smoking, alcohol consumption, dietary habits, physical activity, etc.) respondents are asked to describe their evolution over time. Eventually, a large number of childhood environment

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[5] Detailed descriptive statistics are given in Appendix 3, Table A3.1. In addition, for a number of socio-demographic variables, a comparison is also performed in Appendix 3, Tables A3.2 and A3.3 between SOCIOLD data and the sub-sample of individuals aged 50 and older from the 2001 wave of the European Community Household Panel (ECHP). Except for individuals' highest qualifications, no significant differences emerge from the comparison. It is well known, however, that educational information in the ECHP is very poor since it was not updated once sampled individuals entered the panel.

and living conditions indicators is collected, including housing conditions, school attendance, social and economic environment.

Given the model structure we aim at estimating, we believe the data available to us contain the relevant information except, perhaps, for genetic and intra-uterine information which is missing from the data.

2.2. The model

Our point of departure is the model, depicted in Fig. 1 below, which Adams et al. (2003) propose as a means of summarising the main causal paths between health and SES, discussed in the literature.
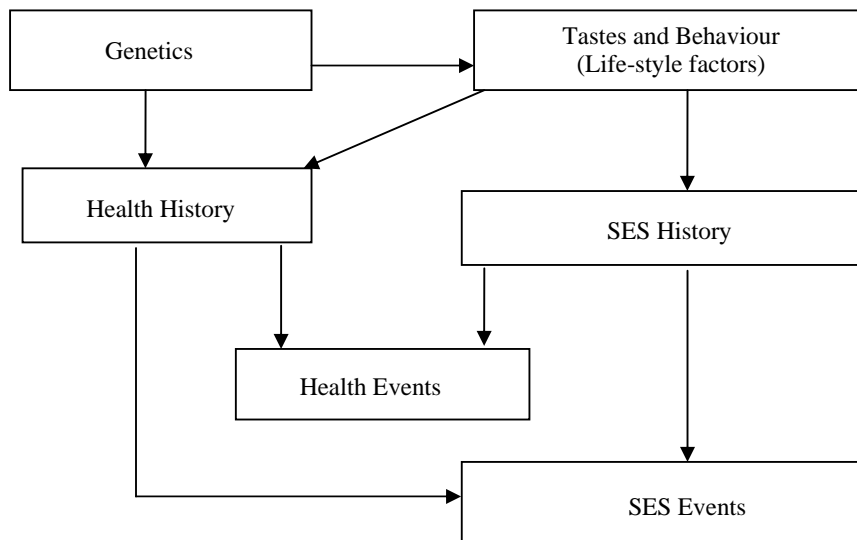
Fig. 1. Causal paths between socio-economic status and health status

Source : Adams et al. (2003), p.5.

An individual's life history is built from these period-by-period transitions. First, genetic factors might influence individuals' tastes and hence, their health accumulation behaviour either in terms of health care consumption or in terms of life style attitudes. But, as a part of individuals' initial endowments, they are also likely to condition their health histories.

Second, individuals' tastes and behaviour are also potential determinants of both their socio-economic and health histories. For instance, risk and time preferences might simultaneously determine individuals' willingness to invest in both health and education. Of course, the health events a person experiences might result either from her health history, her past SES, or

both. Poor health during childhood is likely to result in poor health during adult life. Likewise, poorly educated people are likely to be less aware than others of the importance of hygiene or of preventive medical care.

Eventually, high SES households have a better knowledge about how to maintain and improve their health and are able to devote more resources to health investments. However, health and SES history are also likely to have a direct influence on current SES. On the one hand, a poor health history might reduce one's earnings power. On the other hand, a poor SES history might result in both low earnings and wealth accumulation and then in relatively low current income.

We model these causal paths as a system of five simultaneous equations:

$$\begin{cases} Y_1 = \gamma_{13}Y_3 + \gamma_{14}Y_4 + X_1\beta_1 + u_1 \\ Y_2 = \gamma_{23}Y_3 + \gamma_{24}Y_4 + X_2\beta_2 + u_2 \\ Y_3 = \gamma_{35}Y_5 + X_3\beta_3 + u_3 \\ Y_4 = \gamma_{45}Y_5 + X_4\beta_4 + u_4 \\ Y_5 = X_5\beta_5 + u_5 \end{cases} \tag{1}$$

where Y_j, $j = 1, \cdots, 5$, are measures of socio-economic events (SESE), health events (HE), socio-economic history (SESH), health history (HH) and of tastes and behaviour (TB), respectively, X_j, $j = 1, \cdots, 5$, are vectors of exogenous explanatory variables, γ_{jh} and β_j, $j, h = 1, \cdots, 5$, are parameter vectors of conformable dimensions and u_j, $j = 1, \cdots, 5$, are error terms.

Our testing strategy consists in simultaneously estimating the above model. It has the following advantages:

- It simultaneously accounts for each of the causal paths that are highlighted in Fig. 1;

- It properly treats intermediate factors. For example, SESH appears in our model as being dependent of TB and as a determinant of SESE;

- Confounders are taken into account in two ways. First, the *X* vectors might include common exogenous observable variables which are then considered as determinants of more than one endogenous *Y* variable. Second, unobserved confounders are implicitly accounted for through the error structure of the simultaneous-equation model. Hence, the distributions of the *u* error terms are allowed to share common components;

- Reverse causality is explicitly modelled in the sense that SESH is treated as a determinant of HE whereas HH is treated as a determinant of SESE.

We now explicitly describe each equation of the above model structure.

2.2.1. Tastes and Behaviour

Ideally, the tastes and behaviour index should capture as many health-related individual attitudes as possible. Health is a consequence of various inputs including the adoption of healthy behaviours (dietary habits, exercise, etc.) and the avoidance of unhealthy ones (smoking, drinking, etc.). It is worth noting that not only are such attitudes likely to directly influence health status, but they also reflect individuals' time preferences and attitudes towards risk. For instance, Barsky et al. (1997) use experimental data and offer some evidence for the impact of risk attitudes on lifestyle choices. They find that risk tolerance is positively related to risky behaviours such as smoking and alcohol consumption.

Given the rather complex structure of the model above, we have had to face the difficult choice of an indicator of tastes and behaviour, despite the wealth of information available to us. Including further indicators would have meant equally extending the number of equations in the model. Constructing a synthetic variable would have probably led to a hardly interpretable measure. We have chosen individuals' body mass index (BMI)[6] when they were forty as an indicator of tastes and behaviour. We believe this variable reflects a variety of behavioural characteristics such as sedentary lifestyles, dietary habits and the like. A multiple correspondence analysis suggests BMI is indeed strongly correlated with such characteristics. Furthermore, it also contains some information on risk attitudes and time preferences, albeit very imperfectly (some smokers are slim). The BMI variable, which we denote Y_5, is modelled as a linear function of a set, X_5, of variables describing individuals' genetic factors and a set of demographic and early childhood characteristics which act as control variables. The error term, u_5, is assumed to be normally distributed.

Unfortunately, the data are neither designed to account for genetic factors nor do they contain any explicit information on these. We attempt to control for these by including a variable indicating whether one's father or mother or both has deceased due to some genetic disease.[7]

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[6] BMI is calculated by the index: weight in kg/(height in m)².
[7] In case one of, or both their parents have died, respondents had to indicate whether one of the following diseases was the cause of death: cardiovascular/heart disease, cerebro-vascular, respiratory, endocrine, musculo-skeletal, gastro intestinal, genito-urinary, malignant growth, benign growth, diabetes and/or high blood pressure.

The demographic characteristics include a gender dummy, age and its square, the number of children, two qualification dummies for compulsory and upper secondary education and three marital status dummies, the single being the omitted group.

Early childhood environment is captured through family home characteristics when the respondent was 14 years old. These include the number of rooms in the family home, the number of people who lived in the same family home and a set of dummies indicating whether the respondent had to share her/his bed, whether the house toilet was inside or outside the house, whether there was any crime or vandalism in the area and whether the respondent was brought up in a single parent household.

2.2.2. The History Equations

An individual's history is modelled through a set of two equations, the first one describing her health history, Y_4, and the second one, her socio-economic history, Y_3.

To depict an individual's health history, we rely on a probit model assuming there exists a latent variable the observed counterpart of which is the self-assessment by individuals of their own health ten years prior to the survey.[8] This is the only health history information that is available in the data.

It is often argued that subjective measures of health may lead to substantial under-estimation of the effects of general health. The criticism relies on the idea that individuals might use health to justify their decision not to work. That is, since health is one of the few legitimate reasons to be out of the labour force, individuals who face poor labour market opportunities rationalize their absence from the labour market by reporting poor health (Lindeboom and Kerkhofs, 2002). Another argument is that relative deprivation, due to income inequality for instance, might induce individuals to report lower health statuses. In addition, the distributions of subjective measures are in general difficult to compare across countries as they contain significant cultural and institutional components.

It should, however, be noted that in this paper's context, health history is measured through a variable measuring how individuals currently assess their health ten years earlier, not their current health. We believe that the above criticisms are less relevant for such a measure than for current self-assessed health.

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[8] Past self-assessed health is measured in the data through a 5-scale index, the lowest value indicating the poorest health status. The original variable has been translated into a binary indicator with value 1 for individuals reporting fair, good and very good health.

Socio-economic history is captured through the total number of months respondents have been unemployed over the last ten years. Previous studies have been concerned with the effect of unemployment on subjective well-being. They confirm the hypothesis that unemployment may have strong detrimental effects, particularly on mental health (Björklund and Eriksson, 1998). We assume there is a latent socio-economic history measure, Y_3, the observed counterpart of which is the number of unemployment months over the previous ten years. Since a large proportion of individuals reported no unemployment spells, the socio-economic history variable has to be treated as a left-censored dependent variable and be modelled in a tobit equation[9].

In fact, we also have data on individuals' employment status ten years prior to the survey (e.g. working, self-employed, unemployed versus non-employed). However, we prefer to use the information on unemployment spells over the last ten years for at least two reasons. First, the available employment status information is rather crude and is thus a very poor measure of socio-economic history. Second, the historical dimension is most likely to be better captured by a measure covering a 10 years span rather than by employment status in a given year.

Given the model structure described in Fig. 1, both health history and socio-economic history variables depend on tastes and behaviour. Fig. 1 shows that while genetic factors influence socio-economic history only via their effect on tastes and behaviour, they also have a direct effect on health history. For this reason, they are included in the left-hand side of the health history equation, not in the socio-economic history equation.

Both equations include a set of demographic control variables. Since the history equations relate to ten years prior to the survey, only non time-varying (gender and education) demographic variables are controlled for.

2.2.3. The Events Equations

The model includes two further equations describing individuals' health and socio-economic events, respectively.

It is assumed that there is a latent variable, Y_2, measuring respondents' current health conditions, the observed counterpart of which is current health status. Although the data contain information on subjective and objective measures of health, we have chosen to resort to the latter measure.

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[9] See descriptive statistics in Appendix 3, Table A3.1.

We construct an ADL-score as our health events variable. More specifically, individuals are asked whether, due to chronic physical health conditions, they need help with a number of activities of daily living. They indicate how difficult it is for them to bath or dress, to walk one block, to walk several blocks, to walk more than one mile, to bend, kneel or stoop, to climb one flight of stairs, to climb several flights of stairs, to lift or carry groceries, etc. Taking into account all the items, the ADL-score is stated on a 10-point scale, with 10 indicating the highest difficulty. As the health events index is a count dependent variable, we model it as a Poisson regression equation.

We further assume there is a latent variable, Y_1, measuring individuals' socio-economic events, the observed counterpart of which is current household income. Unfortunately, household income is provided in the data in the form of 13 income bands, which makes cross-country comparisons very difficult to perform. We have thus adopted the following 4-step strategy:

i. For each country, we estimate an ordered probit model the left-hand side of which is household income and the right hand side of which includes age and its square, a female dummy, three marital status dummies, the number of children under 16 and the number of persons between 17 and 64, a part-time dummy, a self-employment dummy and two further dummies for whether the respondent is unemployed or out of the labour force;

ii. For each respondent, we estimate the probability that s/he be in each of the 13 household income bands and infer her/his expected household income;

iii. Given the number of children and the number of adults in the household, we estimate expected equivalized household income;

iv. For each country, we identify the quintiles of the expected equivalized household income distribution within which each respondent falls.

The resulting household income variable is one where individuals are identified through the fifth of the distribution they belong to. It thus reflects the relative position of individuals in the household income distribution, not their absolute income.

According to Fig. 1, the main determinants of both health events and socio-economic events are health history as well as socio-economic history. But these equations include also the set of demographic characteristics: a gender dummy, age and its square, the two qualification

dummies and the three marital status dummies. These variables are thus common to the two exogenous vectors, X_1 and X_2, respectively.

2.3. Estimation Method

Given the data and the way the endogenous variables Y_j, $j=1,...,5,$ are measured, the system in Eq. (1) corresponds to a simultaneous equation model mixing a linear model (tastes and behaviour) with a probit model (health history), a tobit model (socio-economic history), a *Poisson* regression model (health events) and an ordered probit model (socio-economic history).

To estimate such a model structure, we resort to the method of non-linear Asymptotic Least Squares (ALS) which has been developed by Gouriéroux, Monfort and Trognon (1985) as an extension of so-called Amemiya's M-estimation method.[10] As far as we know, though it is rather widely used in other areas of economics (Duguet and Kabla, 1998; Crépon et al., 1998), the ALS method has never been used in the health inequality literature. Although Appendix 1 gives an overview of the method and highlights its main properties, we now briefly motivate our choice and describe the main steps of the empirical set-up.

As can be seen from Appendix 1, the ALS method yields Minimum Distance Estimators and is, by construction, suitable to estimate simultaneous-equation models where the dependent variables are not of the same type as is the case of system in Eq. (1). Compared to the Generalized Method of Moments (GMM), the ALS method has two advantages. First, although both methods imply a two-step estimation procedure, GMM requires the use of the whole data at each step whereas ALS's second step consists in retrieving the structural model parameters from auxiliary parameters to be estimated in the first step. In terms of computation, the ALS method is therefore less costly than GMM. The second reason is that the ALS method can be easily extended to handle more complex simultaneous-equation models. It thus provides a unified and tractable framework for the estimation of generalized limited dependent variables systems (Crepon et al., 1998).

Each of the two ALS estimation steps is thoroughly described in Appendix 1. The first one consists in estimating the reduced-form model parameters using the appropriate likelihood maximisation methods (M-estimation). The structural form parameters being associated to reduced-form ones through a set of so-called identification constraints, they can be inferred in

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[10] See also Gouriéroux and Monfort (1996).

the second step from the reduced-form parameter estimates obtained in the first step. Appendix 1 shows under which conditions both sets of estimates are consistent ones.

The complex structure of the model to be estimated as well as the asymptotic properties of ALS estimators, imply that reliability of the results is crucially dependent on sample size. This is why rather than conducting country-specific analyses, we pool the 6-country data sets and conduct our analyses while controlling for country fixed effects in each of the model equations.

One way to see whether our investment in this intensive technique is worthwhile is to compare our results to those obtained by estimating each equation separately. Such comparison is a means of assessing the biases which would result from neglecting either simultaneity or endogeneity in the model.

2.4. Identification

A brief look at the simultaneous equation model in (1) shows that it explicitly accounts for endogeneity of both health history and socio-economic history as well as of individuals' tastes and behaviour. Fig. 1 also shows that the only variable that could be considered as completely exogenous is that related to genetic factors. However, since it enters the health history equation as well as the tastes and behaviour equations, it cannot serve as a means of properly identifying the whole set of parameters in the model. In this model structure, genetic factors are assumed to be directly correlated to health history, not only via their effect on tastes and behaviour. They are thus correlated to health events and to socio-economic events since health history is a determinant of these.

Rather, we resort to other variables which we believe could reasonably be thought of as valid instruments. Appendix 2 presents the statistical tests we have performed to check this hypothesis. The results are in favour of our intuition.

To identify parameters γ_{35} and γ_{45} in the health history and in the socio-economic history equations, the instrument we include in the X_5 vector entering the tastes and behaviour equation is a dummy variable which is inferred from the following question :

Suppose that you were asked to stop smoking and in exchange for that you would be guaranteed that you get an extra period of life as an active person in reasonably good health. How long would the minimum of additional life period have to be for you to accept the offer?

One additional year,

Two additional years,
3-4 additional years,
5-6 additional years,
More than 6 additional years,
I would not accept the offer, irrespective of how long the period of additional years offered to me would be.

Those responding '*One additional year'* have been assigned value 1 while the others have been assigned zero.

Such variable could be seen as an indicator of individuals' time preference. Time preference refers to the rate at which people are willing to trade current benefit for future benefit. A higher rate of time preference will, *ceteris paribus*, result in a lower propensity to exercise, to observe no dietary restrictions, etc. Under this interpretation, one should expect those willing to give up smoking in exchange of a single extra year of life to adopt the safest health behaviour. Alternatively, it might be the case that individuals with poor life styles, including smoking, are able to measure the risks they face and thus to be more likely to be prepared to stop smoking. Under this interpretation, one should expect those willing to give up smoking to also have poor health behaviours.

However, we believe this indicator is very likely to be correlated with health history and events as well as with socio-economic history and events, but only via its "effect" on individuals' tastes and behaviour. It could thus be legitimately excluded from the first four equations of model (1).

To identify parameters γ_{14} and γ_{24} in the health events and in the socio-economic events equations, the instrument we include in the X_4 vector entering the health history equation is an indicator of individuals' initial health endowments. This was made possible from exploitation of the following question:

When you were less than 12 years of age, did you ever miss one month or more of school for health reasons?

Yes,
No,

The idea here is that initial health endowments are unlikely to affect health or socio-economic events if they have no influence on one's health history.

Likewise, to identify parameters γ_{13} and γ_{23} in the health events and in the socio-economic events equations, the instrument we include in the X_3 vector entering the socio-economic

history equation is an indicator of individuals' perception of the role luck plays in life. To be more specific, respondents in the survey are asked the following question:

Do you believe that in the long run people get the respect they deserve in this world?

Yes,
No,

Our assumption is that individuals who believe that the society they live in values effort and hard work are likely to have done as much as they could to improve their socio-economic status throughout their working lives and that both their current health and socio-economic statuses are influenced by the effect of such behaviour on their socio-economic history. Put differently, one's health and/or socio-economic statuses in the long run results from the cumulative effect of the efforts they have exerted throughout her/his life, not simply from short run attitudes.

3. Results

The data and empirical strategy presented in section 2 led to the results reported in Table 1 below which shows the estimated coefficients of the structural model (1). Each of the coefficients associated with endogenous variables, the γ coefficients, is indeed associated with a specific causal path and the main purpose of this study is to discriminate between significant causal paths and insignificant ones. Even though, the assessment of the degree of homogeneity of the population the significant paths hold for requires that all other variables that are controlled for be discussed first. We thus start by interpreting the explanatory power of the exogenous variables before we turn to interpreting the role of the endogenous ones.

An important feature displayed by Table 1 is the statistical significance of the three instrumental variables that are included in the model. This implies that as long as our assumptions regarding the correlation of these variables with the endogenous ones are valid, the γ parameters of the model are properly identified.[11] The signs on these instruments are also economically relevant and conform to the intuition. In column 1 (TB equation), the positive sign on the coefficient associated with the 'Lifesmoke' variable suggests that respondents whose BMI was relatively high when they were 40 are more likely to be willing to give up smoking for a 1-year extra period of life. While this contradicts the time preference interpretation, it is in line with the cumulated risk interpretation.[12]

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[11] See Appendix 2 for tests of the validity of instruments.
[12] Both these interpretations are discussed in section 2.4.

Likewise, column 2 shows that those who have had to miss school for one month or more for health reasons when they were less than 12 are less likely to report high past health scores. This suggests that health history is positively correlated with one's health status during childhood.

Eventually, the negative coefficient associated with the 'respect' variable means that individuals who believe that in the long run people get the respect they deserve in this world have had shorter unemployment durations during the last ten years. Such a result suggests that one's socio-economic history depends on her/his belief about how society rewards effort.

The other exogenous variables included in the model are country fixed effects as well as demographic characteristics.

Table 1: Structural form estimates

TB: BMI at 40 years old
HH: Health status in 1994
HE: ADL-score
SESH: Past unemployment duration
SESE: Household income quintiles

Equations	TB		HH		HE		SESH		SESE	
Variables	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE
Endogenous variables										
Tastes and Behaviour (TB)			-0,015	0,039			0,064	0,083		
Health History (HH)					-0,885***	0,061			0,021***	0,008
SES History (SESH)					0,139**	0,070			-0,037**	0,018
Parents' disease	0,518***	0,170	-0,116**	0,050						
Crime area	0,604*	0,324								
Number of persons/room	0,179	0,517								
Monoparental family	0,152	0,326								
Toilet inside	-0,151	0,235								
Bed sharing	-0,055	0,221								
Parents well-off	-0,546**	0,235								
Missed school			-0,385***	0,054						
Lifesmoke	0,479***	0,173								
Respect							-0,161**	0,081		

*** significant at 1%; ** significant at 5%; * significant at 10%.

Table 1 (continued): Structural form estimates

Equations	TB		HH		HE		SESH		SESE	
Variables	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE
Female	-0,587***	0,157	-0,132***	0,047	0,099***	0,020	0,090	0,106	-0,675***	0,031
Age					0,050***	0,020			0,020	0,059
Age²					0,019	0,029			-0,023	0,053
Upper secondary education	-0,340*	0,220	0,193***	0,057	-0,156***	0,021	-0,138	0,137	0,197***	0,043
Higher education	-0,692**	0,224	0,219***	0,062	-0,488***	0,053	-0,252*	0,148	0,556***	0,045
Married	-0,559**	0,294			0,042	0,039			0,412***	0,056
Divorced	-0,863***	0,340			-0,027	0,034			-0,009	0,067
Widowed	-0,259	0,445			-0,109***	0,044			0,215***	0,088
Country fixed effects[1]										
France	-0,680***	0,283	-0,179**	0,080	0,147***	0,036	0,146	0,166	-1,047***	0,055
Denmark	-0,150	0,313	-0,162**	0,075	0,118**	0,034	-0,052	0,164	-0,322***	0,056
Netherlands	-0,112	0,320	-0,375***	0,067	0,146	0,151	-1,896***	0,182	-0,639***	0,066
United-Kingdom	-0,385	0,288	-0,387***	0,072	0,180***	0,049	0,495***	0,151	-0,117**	0,051
Finland	0,097	0,351	-0,179**	0,092	-0,329***	0,054	-0,306	0,204	-0,626***	0,066
Constant	27,402***	8,396	1,408	1,039	0,210***	0,058	-0,562	1,103	Cuts	not reported
Number of observations : 5041										

*** significant at 1%; ** significant at 5%; * significant at 10%.

(1) Greece is the omitted country.

Country fixed effects in column 1 (TB equation) suggest individuals from different countries show similar propensities to invest in health. One exception though is France where BMI is significantly lower than in any other country. This confirms the so-called French paradox.[13] In contrast in column 2, the high significance of the negative coefficients on country dummies suggests that the Greeks have reported the highest past health scores. Whether this reflects real cross-country differences in health statuses or cultural differences influencing the degree of optimism in each country is hard to tell. Note, however, that country fixed effects in column 3 suggest Greece is also a country where ADL-scores are the lowest. Among the five remaining countries, only Finland does better. This suggests that inter-country differentials in the subjective and objective measures of health are quite comparable. Looking at column 4, it seems that while older workforce members from Denmark, Finland, France and Greece have on average been unemployed for similar numbers of months during the last ten years, their British (Dutch) counterparts have experienced more or longer (fewer or shorter) unemployment spells. Also interesting are the country-fixed effects highlighted in column 5 as they show that, *ceteris paribus*, it is in Greece where older workforce members are the most likely to have the highest positions in the equivalised household income distribution of their country.

Though neither household structure nor home characteristics during childhood are significant, the results in column 1 suggest childhood environment does influence BMI and thus the propensity to invest in own health. In particular, though the corresponding coefficient is significant at the 10% level only, those having grown up in an area of crime or vandalism report on average a higher BMI. In addition, those reporting their parents were well-off when they were children also report, *ceteris paribus*, significantly lower BMIs.

It is also worth noting from columns 1 and 2 that those whose father, mother or both died from some genetic disease tend to be fatter but also to report lower past health scores. Though the variable we use is rather poorly informative, these results confirm the idea of intergenerational transmission of health; that one's health depends on her/his parents'.

Let us now consider the effects of demographic variables. In line with the usual findings, our results show that males tend to be fatter than females. They also suggest that the more educated have a lower BMI. This confirms that the highly educated have a higher propensity to invest in health. Also, when compared to the single or the widowed, the married and the

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[13] The French paradox refers to the fact that although the French have a diet richer in saturated fat than the British for instance, the French mortality rate from heart disease is substantially lower. The most common explanation relies on the protective effect of wine drinking.

divorced seem to report lower BMIs. This probably reflects different dietary habits between these two groups.

Also in line with standard findings are the results in column 2 which show that females tend to report lower past health scores while the more educated tend to report higher ones than their least educated counterparts. These effects are confirmed by the results in column 3 with respect to the effects on current health. Again, females report the higher ADL-scores than their male counterparts whereas more education goes with lower ones. But columns 3 also exhibits results regarding age and marital status. As one would expect, it illustrates the positive correlation between age and ADL-scores. However, it also shows that among older workforce members, there are no significant differences in ADL-scores between the single, the married and the divorced; only the widowed enjoy, *ceteris paribus*, a better health status. There is nothing paradoxical in this result since age is already controlled for. What it probably says is that, *ceteris paribus*, those who survive to their partners are on average healthier.

Looking at column 3, we see that there are no significant gender differentials in the number of months of unemployment over the last ten years. In contrast, the effect of education is significant, albeit at the 10% level only. In line with the well-known negative effect of education on unemployment risk, it simply reflects the fact that the highly educated have experienced fewer months of unemployment over the last ten years.

Interesting results also appear in column 5 as to the effect of demographic characteristics on individuals' position in the household income distribution of their country. While there is *a priori* no reason why women would enjoy lower income than men within the same household, the significance of the negative coefficient on the female dummy suggests that women are less likely to belong to the highest fifths of the household equivalised income distribution. This probably suggests that women tend to live in larger size households or that male respondents tend to belong to wealthier households than their female counterparts. In contrast, age seems to have no significant effect on one's position in the income distribution. Given the relative homogeneity of our sample with respect to age (older workforce), this probably reflects the commonly observed peakedness of age-earnings profiles. The coefficients on educational dummies have the expected signs: more education goes with higher income. Eventually, compared to the single and the divorced, the married and the widowed enjoy higher positions in the income distribution. This is not surprising since in both cases, the partner or ex-partner contributes to household income in one way or another.

We now consider the structural coefficients of the endogenous variables of model (1). Looking at the first row of Table 1, we see that BMI has a significant influence on neither our health history indicator nor on our socio-economic history variable. This suggests that one's tastes and behaviour have no influence on her/his health and socio-economic history. This result should be treated with caution. Not only is BMI not an exhaustive measure of tastes and behaviour but it might also be the case that self-assessed past health and/or unemployment spells over the last previous years are not variables BMI would primarily affect. It is, however, worth noticing from Appendix 4, Table A4.1, that when simultaneity and/or endogeneity are not accounted for, BMI seems to have a rather strong influence on both self-assessed past health and on the number of unemployment months over the last ten years. Clearly, neglecting simultaneity and/or endogeneity would have led to misleading results, suggesting higher BMI has a significant negative effect on past health and on unemployment duration.

But row 2 of Table 1 shows health history has a highly significant effect on both current health status and current SES. This is a first element of the spiral mentioned above. First, it illustrates the dynamics of health accumulation in the sense that the higher the past health score one reports, the lowest is her/his ADL-score and the better is her/his current health status. Second, it provides evidence in favour of the selection hypothesis in the sense that one's past health status causes her/his current SES as measured by unemployment duration. Note that the significance of both health dynamics and selection-related causal paths is confirmed by Appendix 4, Table A4.1, where neither simultaneity nor endogeneity are accounted for. Actually, where the two sets of estimates differ is in the magnitude of the highlighted causal effects. Compared to Appendix 4, Table A4.1, Table 1 highlights a stronger effect of past health on current health but at the same time, a much weaker selection effect, the coefficient measuring the causal effect of past health on current SES is at least ten times smaller.

Finally, row 3 of Table 1 suggests socio-economic history causes both current health and current SES, again pleading in favour of the spiral effect. It says that the larger is the number of months an individual has been unemployed during the last ten years the lower is her/his position in the household income distribution. This illustrates the dynamics of SES formation. The other highlighted effect is that a longer unemployment spell results in a poorer current health status.[14] This is in favour of the causation hypothesis that SES causes health. Again,

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[14] This is obviously not in the spirit of Ruhm's (2000) finding that health improves during recessions.

these results are confirmed by those reported in Appendix 4, Table A4.1 although the estimated effects differ in magnitude. Table 1 suggests that neglecting simultaneity and endogeneity would have resulted in a downward biased causation effect and an upward bias in the SES dynamics effect.

4. Concluding remarks

In this paper, we have attempted to address an old question using an original methodology. While the literature suggests there is a large variety of causal paths underlying the relationship between health and socio-economic status, the question is that of how to discriminate between significant and insignificant ones. The task is difficult to perform since the relationship is complex. One has indeed to account for reverse causality, the presence of intermediate factors as well as of (unobserved) cofounders. While the conventional approach consists in general in treating the problem as a simple endogeneity or causality one, we argue that only in a structural model framework can one capture the subtleties of the processes of health accumulation and socio-economic status formation. These processes are indeed of a dynamic nature since health and socio-economic status start interacting with each other early in one's life and their effects cumulate over the life cycle. Thus, only by explicitly modelling these processes will one be able to understand both the health and socio-economic statuses of individuals at each stage of their lives.

It is often argued that income redistribution is a means of reducing health inequalities. But while this sounds trivial, a number of issues have to be considered. First, depending on their preferences, beneficiaries of extra-income will not necessarily devote their extra-resources to investing in health (Sen, 2002; Fleurbaey, 2007). Second, if preferences vary with age, then it is unlikely that redistributive policies show the same effectiveness depending on whether the beneficiaries are young or old persons. Another standard policy recommendation states that widening educational opportunities may result in narrower health inequalities. While this is certainly true, especially for young people and the forthcoming generations, it sheds no light on how to reduce health inequalities among currently old individuals whether in the labour force or out. What these few examples highlight is the need of age-specific policy recommendations. For instance, our data suggest that any improvement of the living environment of children will make it less likely that they suffer from obesity at adult age. This illustrates how important it is to account as much as possible for these dynamic effects when addressing the causality issues.

Our data suffer from a number of limitations which did not allow us to account for as many important aspects as we would have liked. Yet, despite their weaknesses, they illustrate well enough how important it is to simultaneously consider the effect of health on socio-economic status, the effect of socio-economic status on health together with the dynamic nature of these effects. Moroever, further questions still need to be addressed. For instance, how sensitive are the results to (*i*) considering different measures of socio-economic status as well as of health, including mental health, (*ii*) including, or focussing on, younger individuals, (*iii*) accounting for the effect of working conditions, (*iv*) accounting for parents' health as well as economic endowments? These questions are left for future research.

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Appendix 1: Estimation of model (1) using non-linear asymptotic least squares

The key idea of ALS is to estimate reduced form coefficients in each equation of the model separately and then infer from these auxiliary parameters the structural form parameters of the model using a minimum distance estimator. The intuition is the same as in the method of indirect least squares, although ALS is more general. We thus have two estimation issues: first, estimating the reduced form parameters and their joint covariance matrix; then estimating the structural form parameters in a consistent and efficient way. The first problem is solved by interpreting the maximum likelihood estimators of the reduced form equations as specific M-estimators. The second problem boils down to writing explicitly the relationships between the structural form parameters (parameters of interest) and the reduced form parameters (auxiliary parameters).

Reduced form estimation

Our structural model consists of five equations:

$$
\begin{cases}
Y_1 = \gamma_{13} Y_3 + \gamma_{14} Y_4 + X_1 \beta_1 + u_1 \\
Y_2 = \gamma_{23} Y_3 + \gamma_{24} Y_4 + X_2 \beta_2 + u_2 \\
Y_3 = \gamma_{35} Y_5 + X_3 \beta_3 + u_3 \\
Y_4 = \gamma_{45} Y_5 + X_4 \beta_4 + u_4 \\
Y_5 = X_5 \beta_5 + u_5
\end{cases}
\tag{1}
$$

where Y_j, $j = 1, \cdots, 5$, are measures of socio-economic events (SESE), health events (HE), socio-economic history (SESH), health history (HH) and of tastes and behaviour (TB), respectively, X_j, $j = 1, \cdots, 5$, are vectors of exogenous explanatory variables, γ_{jh} and β_j, $j, h = 1, \cdots, 5$, are parameter vectors of conformable dimensions and u_j, $j = 1, \cdots, 5$, are random disturbances. The five equations of the model are respectively estimated by an ordered probit model, a *Poisson* regression model, a Tobit model, a probit model and a linear model.

Once the reduced form of the model is written, the estimation problem becomes that of a series of single equations and the estimation method that is the most appropriate for each equation can be applied. One can thus write:

$$
\hat{\pi}_k \in \arg \max_{\pi_k} L_k(\pi_k), \qquad k = 1, \cdots, 5
$$

where π_k is the reduced form parameter in equation k and L_k, the corresponding likelihood function. The problem remains to estimate the joint covariance matrix of the $\hat{\gamma}_k$'s. We can solve it by considering that our estimators can also be defined globally by:

$$\hat{\pi} \in \arg\max_{\pi} L(\pi) \text{ with } \pi = \begin{pmatrix} \pi_1 \\ \vdots \\ \pi_5 \end{pmatrix} \text{ and } L(\pi) = \sum_{k=1}^{5} L_k(\gamma_k)$$

Maximising L with respect to π gives exactly the maximum likelihood estimators. This property arises from the separability of $L(\pi)$ with respect to the $\hat{\gamma}_k$'s. These estimators can be interpreted as M-estimators. Under the usual regularity conditions for M-estimators:

$$\sqrt{N}(\hat{\pi} - \pi) \xrightarrow[N \to +\infty]{} N(0, \Omega)$$

with asymptotic covariance matrix:

$$\Omega = J^{-1} I J^{-1}$$

where

$$I = \underset{X}{E}\underset{Y}{E}\left[\frac{\partial L}{\partial \pi}(\pi_0) \frac{\partial L}{\partial \pi'}(\pi_0) \right], \qquad J = \underset{X}{E}\underset{Y}{E}\left[-\frac{\partial^2 L}{\partial \pi \partial \pi'}(\pi_0) \right]$$

where π_0 denotes the true value of parameter π and the expectations are taken with respect to the distributions of the exogenous variables (index X) and of the endogenous variables (index Y).

The matrix I and the matrix J can be estimated by their sample counterparts:

$$\hat{I} = \frac{1}{N} \sum_{i=1}^{N} \frac{\partial L_i}{\partial \pi}(\hat{\pi}) \frac{\partial L_i}{\partial \pi'}(\hat{\pi}), \qquad \hat{J} = -\frac{1}{N} \sum_{i=1}^{N} \frac{\partial^2 L_i}{\partial \pi \partial \pi'}(\hat{\pi})$$

where N is the sample size. Note that since the objective function is separable, the off-diagonal terms (the cross derivatives) in $\hat{J}$ (and J) are zero.

Eventually,

$$\hat{\Omega} = \hat{J}^{-1} \hat{I} \hat{J}^{-1}$$

can be used as the estimator of the covariance matrix of the estimated reduced form parameters.

Through $\hat{\Omega}$ (and $\hat{I}$), the correlations between the perturbations of the different equations of the model are taken into account, without making any specific distributional assumptions on their joint distribution.

Let us write the reduced form:

$$\begin{cases} Y_1 = X\pi_1 + v_1 \\ Y_2 = X\pi_2 + v_2 \\ Y_3 = X\pi_3 + v_3 \\ Y_4 = X\pi_4 + v_4 \\ Y_5 = X\pi_5 + v_5 \end{cases} \tag{2}$$

where:

$$\underset{(N,k)}{X} = \begin{bmatrix} \underset{(N,k_1)}{X_1} \\ \underset{(N,k_2)}{X_2} \\ \underset{(N,k_3)}{X_3} \\ \underset{(N,k_4)}{X_4} \\ \underset{(N,k_5)}{X_5} \end{bmatrix}$$

Define exclusion matrices S_k, $k = 1, \cdots, 5$, consisting of 1's and 0's at the appropriate places, such that:

$$XS_k = X_k$$

Then, substituting of (2) into (1) and identifying, we get:

$$\begin{cases} \pi_1 = \gamma_{13}\pi_3 + \gamma_{14}\pi_4 + S_1\beta_1 \\ \pi_2 = \gamma_{23}\pi_3 + \gamma_{24}\pi_4 + S_2\beta_2 \\ \pi_3 = \gamma_{35}\pi_5 + S_3\beta_3 \\ \pi_4 = \gamma_{45}\pi_5 + S_4\beta_4 \\ \pi_5 = S_5\beta_5 \end{cases} \tag{3}$$

Amemiya suggested directly estimating the system (3) using regression methods. Let us use the following notations:

$$
\begin{aligned}
\hat{\Pi}_1 &= (\hat{\pi}_3, \hat{\pi}_4, S_1) & \theta_1' &= (\gamma_{13}, \gamma_{14}, \beta_1) \\
\hat{\Pi}_2 &= (\hat{\pi}_3, \hat{\pi}_4, S_2) & \theta_2' &= (\gamma_{13}, \gamma_{14}, \beta_2) \\
\hat{\Pi}_3 &= (\hat{\pi}_5, S_3) & \theta_3' &= (\gamma_{35}, \beta_3) \\
\hat{\Pi}_4 &= (\hat{\pi}_5, S_4) & \theta_4' &= (\gamma_{45}, \beta_4) \\
\hat{\Pi}_5 &= (S_5) & \theta_5' &= (\beta_5)
\end{aligned}
$$

and:

$$
\eta_k = (\hat{\pi}_k - \pi_k) - \sum_{m \neq k} (\hat{\pi}_m - \pi_m), \qquad k = 1, \cdots, 5
$$

We get:

$$
\begin{cases}
\pi_1 = \hat{\Pi}_1 \theta_1 + \eta_1 \\
\pi_2 = \hat{\Pi}_2 \theta_2 + \eta_2 \\
\pi_3 = \hat{\Pi}_3 \theta_3 + \eta_3 \\
\pi_4 = \hat{\Pi}_4 \theta_4 + \eta_4 \\
\pi_5 = \hat{\Pi}_5 \theta_5 + \eta_5
\end{cases}
$$

or, in matrix notations:

$$
\pi = \hat{\Pi} \theta + \eta .
$$

where $\eta = g(\theta, \hat{\Pi})$.

The basic idea of ALS is to use the estimate $\hat{\Pi}$ of π obtained in a first step and compute in a second step an estimate $\hat{\theta}$ of θ such that $g(\hat{\theta}, \hat{\Pi})$ is as 'close to zero' as possible. That is, we solve the program:

$$
\hat{\theta} = \arg \min_{\theta} g(\theta, \hat{\Pi})' \Psi^{-1} g(\theta, \hat{\Pi})
$$

where Ψ is a metric. Whatever Ψ is, $\hat{\theta}$ is consistent if $\hat{\Pi}$ is consistent, and it is asymptotically efficient for Ψ given by:

$$
\Psi^* = \underset{asymp}{V} \left[g(\theta, \hat{\Pi}) \right] = \frac{\partial g}{\partial \Pi'}(\theta, \hat{\Pi}) \Omega \frac{\partial g'}{\partial \Pi}(\theta, \hat{\Pi})
$$

where $\Omega = \underset{asymp}{V} \left[\hat{\Pi} \right]$ is the covariance matrix of $\hat{\Pi}$. Since we need an estimate of θ to estimate the optimal metric Ψ^*, we may estimate θ in two steps:

In the first step, we can use for Ψ the Euclidian metric and estimate θ as:

$$\hat{\theta} = \arg\min_{\theta} g(\theta, \hat{\Pi})' g(\theta, \hat{\Pi})$$

and then the asymptotic distribution of $\hat{\theta}$ is:

$$\sqrt{N}(\hat{\theta} - \theta) \xrightarrow[N \to +\infty]{} N(0, \Sigma)$$

with:

$$\Sigma = \left(\frac{\partial g'}{\partial \theta} \frac{\partial g}{\partial \theta'}\right)^{-1} \frac{\partial g'}{\partial \theta} \frac{\partial g}{\partial \Pi'} \Omega \frac{\partial g'}{\partial \Pi} \frac{\partial g}{\partial \theta'} \left(\frac{\partial g'}{\partial \theta} \frac{\partial g}{\partial \theta'}\right)^{-1}$$

Replacing θ, Π and Ω by their consistent estimates $\hat{\theta}$, $\hat{\Pi}$ and $\hat{\Omega}$, we are now able to compute an estimated $\hat{\Psi}*$ such that:

$$\hat{\Psi}* = \frac{\partial g}{\partial \Pi'}(\hat{\theta}, \hat{\Pi}) \hat{\Omega} \frac{\partial g'}{\partial \Pi}(\hat{\theta}, \hat{\Pi})$$

In the second step, we can then compute the corresponding optimal ALS estimator $\theta*$ such that:

$$\hat{\theta} = \arg\min_{\theta} g(\theta, \hat{\Pi})' \hat{\Psi}*^{-1} g(\theta, \hat{\Pi})$$

and the asymptotic distribution of $\theta*$ is:

$$\sqrt{N}(\theta* - \theta) \xrightarrow[N \to +\infty]{} N(0, \Sigma*)$$

where $\Sigma*$ can be estimated by:

$$\Sigma* = \left(\frac{\partial g'}{\partial \theta}(\hat{\theta}, \hat{\Pi}) \hat{\Psi}*^{-1} \frac{\partial g}{\partial \theta'}(\hat{\theta}, \hat{\Pi})\right)^{-1}.$$

Appendix 2: Testing for the validity of instruments in the model

The data we use in this paper are the outcome of a common questionnaire that has been circulated in the six participating countries; namely, Denmark, Finland, France, Greece, Netherlands and the UK. The questionnaire has been designed by the researchers involved in the EU-funded project, SOCIOLD.[15] Beside the core questionnaire, a number of questions have also been included regarding potential instruments for the identification of the major causal paths between health and socio-economic status that are discussed in the literature.

It is within this set of variables that we have looked for variables that may conform the exclusion restrictions which identification in our 5-equation model requires. However, given the different types of endogenous variables that intervene in the model, this selection process is difficult as standard Sargan-type tests are not appropriate in this context. This appendix section describes our selection strategy and statistically motivates the choice of the instruments that we have finally adopted.

The model structure (1) comprises five interdependent equations, some of which including two endogenous variables on their right-hand side. To test whether or not a potential instrument could be seen as a valid one, we simply consider each pair of interdependent equations in isolation. Consider a pair of such equations and write it as follows:

$$Y_j = X_j \beta_j + \gamma_{jh} Y_h + \gamma_{jk} Y_k + u_j$$
$$Y_k = X_k \beta_k + \alpha_k V_k + \gamma_{kr} Y_r + \gamma_{kl} Y_l + u_k$$

$$j \neq h \neq k \neq r \neq l.$$

where the Y's denote potentially endogenous variables, the X's are vectors of control variables, the u's are random disturbances and V_k is an instrumental variable which, under certain conditions, helps identifying γ_{jk}; that is the effect of Y_k on Y_j. Indeed, it is assumed that $E(Y_k u_j) \neq 0$ so that separate estimation of the first equation is likely to result in a biased estimate of the γ_{jk} parameter. Because of the complex nature of the model, we simply ignore the fact that Y_h, Y_r and Y_l might themselves be endogenous. The argument is that when testing for whether or not V_k is a valid instrument, the parameters of interest are α_k and γ_{jk}, not γ_{jh}, γ_{kr} or γ_{kl}. Of course, once instruments are shown to be valid, potential endogeneity

---

of every *Y* variable is accounted for in the simultaneous estimation framework described in section 2.

For V_k to be a valid instrument, it must fulfil the following requirements:

(i)   It could be legitimately excluded from the equation the left-hand side of which is Y_j. That is, it cannot be directly correlated with Y_j.

(ii)   If there should be any correlation between V_k and Y_j, then it should be exclusively due to the effect of V_k on Y_k.

In this paper, the three instruments that we use are dummy variables. However, the *Y* variables can be of any sort: continuous or discrete, quantitative or qualitative. Suppose Y_j is continuous or transformed into a continuous variable. Suppose also that we transform Y_k into a categorical variable. Then one means of assessing whether or not V_k conforms conditions (i) and (ii) is the following:

(i) V_k should be correlated with Y_k.

(ii)   If included in the first equation (the left-hand side of which is Y_j), V_k should have no significant effect on Y_j.

(iii)   Estimate $Y_j^s = X_j^s \beta_j^s + \gamma_{jh}^s Y_h^s + \eta_j^s$ using the sub-sample of individuals with $V_k = s$, $s = 0,1$. Evidence that V_k has no direct effect on Y_j could be provided by showing that, on average, η_j^0 is not statistically different from η_j^1.

We now proceed with each equation of model (1).

1. *Identifying the effect of tastes and behaviour on health history and on socio-economic history*

Let us consider first the three last equations of model (1). To identify the effect of tastes and behaviour on socio-economic history (γ_{35}) and on health history (γ_{45}), the instrument included in the TB equation is LIFESMOKE, which is a dummy variable taking value 1 for individuals willing to give up smoking in exchange of a single additional year as an extra period of life.

Simple mean calculations show that BMI at age 40 is on average 26.76 for the sub-sample of individuals with LIFESMOKE = 1 and 24.24 for those with LIFESMOKE = 0. Similar patterns emerge when one distinguishes between males and females or between countries. This suggests the LIFESMOKE indicator is "correlated" with BMI at age 40, our measure of tastes and behaviour. This is probably due to smokers with a relatively high BMI being aware of all the risk factors they face and are thus prepared to give up smoking in exchange of no more than one year of extra-life.

One means of showing that LIFESMOKE has no direct effect on socioeconomic history is to include it as an additional explanatory variable in the third equation of model (1). We have separately estimated such augmented socio-economic history equation using three different methods. Because socio-economic history is measured by the number of months of unemployment over the last ten years, we first treated the resulting equation as a count data model. But we have also estimated it using ordinary least squares. In both cases, we have alternatively estimated it using the whole sample of individuals and separating individuals according to whether their BMI at age 40 was below or above 25. In none of these cases has the LIFESMOKE variable turned out to have any significant effect on our socio-economic history measure.

Likewise, to see whether or not LIFESMOKE has any significant effect on health history, we have included it as an additional explanatory variable in the fourth equation of model (1). Given that the left-hand side variable of the resulting equation is of an ordered nature (self-assessed health ten years earlier), two versions of it have been estimated: a 5-outcome ordered probit (very good, good, fair, poor, very poor) as well as a linear version where the past health variable has been made continuous using z-scores. Again, both strategies have been implemented either using the whole sample of individuals or separating individuals according to whether their BMI at age 40 was below or above 25. For each estimated equation and for both sampling frameworks, the marginal probability that the coefficient on the LIFESMOKE variable be significant never reached the 10 percent level.

As a complementary approach, we have also estimated the socio-economic history equation (treating the number of unemployment months as a continuous variable), excluding the BMI variable from the right-hand side and separating the whole sample according to whether BMI at age 40 was above 25 or below. The idea is that if LIFESMOKE did not fulfil the exclusion restriction (had any direct effect on health history), then for slim individuals, for fatter ones or for both there would be a statistically significant difference between the residuals for

LIFESMOKE = 1 and for LIFESMOKE = 0. The same approach has been adopted regarding the health history equation with the ordered assessment of past health has been replaced by the corresponding z-scores. For BMI below 25 and above 25, respectively, Fig. A2.1 reports the mean residual for LIFESMOKE = 0, the lower and upper bounds of its 95% confidence interval as well as the mean residual for LIFESMOKE = 1. The residuals from the health history equation are reported on the left of the figure whereas those from the socio-economic history equation are reported on the right of the figure.

Fig. A2.1. Residuals from the health history and the socio-economic history equations (effect of tastes and behaviours)
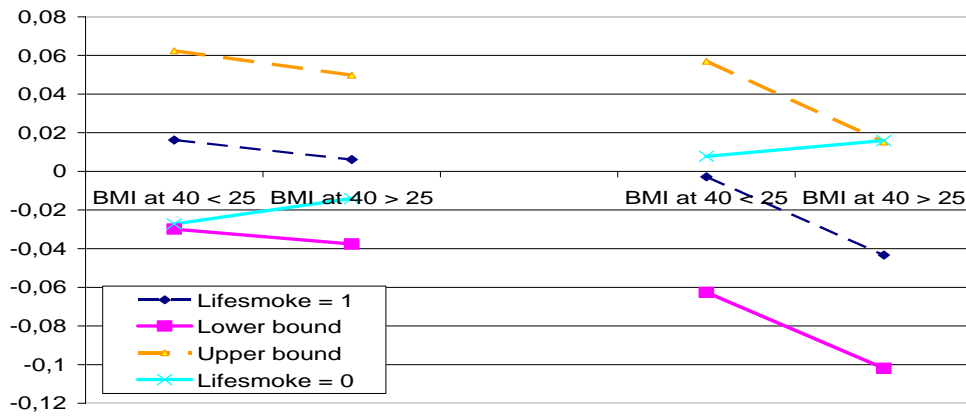
2. *Identifying the effect of health history on health events and on socio-economic events*

We now consider the first, the second and the fourth equations of model (1). To identify the effect of health history on socio-economic events (γ_{14}) and on health events (γ_{24}), the instrument included in the health history equation is a dummy for whether the individual missed school one month or more for health reasons when s/he was less than 12.

Table A2.1. Proportions of individuals reporting their health was good in 1994

Missed school for health reasons when less than 12	All	By gender		By country					
		Male	Female	DK	FI	FR	GR	NL	UK
YES	64.90	68.22	61.54	78.26	66.67	69.38	69.64	58.17	61.60
NO	80.35	81.61	79.13	82.11	81.86	81.73	85.24	74.37	76.23

Table A2.1 shows that for both males and females, those having had to miss school for health reasons in their childhood are less likely to have enjoyed good health 10 years earlier. This is descriptive evidence that health during childhood has an influence one one's health history. Thus, the remaining question is that of whether or not it has a direct effect on one's current socio-economic status and health.

We have first estimated an extended version of the first equation of model (1) where the missing school dummy has been added to the right-hand side. Two versions of the resulting equation have been estimated: one where individuals' current socio-economic status is measured by their position in the national household income distribution (an ordered probit model) and one in which it is measured by individuals' expected household income, corrected for cross-country differences in purchase power.[16] In none of these specifications has the dummy for missing school during childhood turned out to be significant. The same held when the whole separate regressions have been performed, distinguishing between individuals reporting their health was good ten years earlier and those reporting a bad past health status.

The same strategy has been adopted to test whether or not the missing school dummy has a direct effect on health events. It has indeed been included as an extra explanatory variable in the second equation of model (1) where, in addition, the left-hand side variable (ADL-scores) has been treated as a continuous variable. Again, whether one considers the whole sample or distinguishes between individuals reporting good past health and those reporting bad past health, the coefficient on the missing school variable show no statistical significance.

An additional test consisted in estimating the socio-economic events equation (with expected household income on its left-hand side), excluding the health history indicator and distinguishing between individuals having reported a good past health status and those having reported a poor one. Again, the idea is that if the missing school variable is to be legitimately excluded from the household income equation, then there should be no significant difference between the residuals whether individuals were previously healthy or not. The same approach has been adopted regarding the health events equation with ADL-scores being treated as a continuous variable. For previously healthy and previously unhealthy individuals, respectively, Fig. A2.2 reports the mean residual for the sub-sample of individuals having not missed school for health reasons when they were less than 12, the lower and upper bounds of its 95% confidence interval as well as the mean residual for the sub-sample of individuals having missed school for health reasons when they were less than 12. The residuals from the health events equation are reported on the left of the figure whereas those from the socio-economic events equation are reported on the right of the figure.

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[16] See section 2.2.3 for the way the expected income variable has been constructed.

Fig. A2.2. Residuals from the health events and the socio-economic events equations (effect of health history)
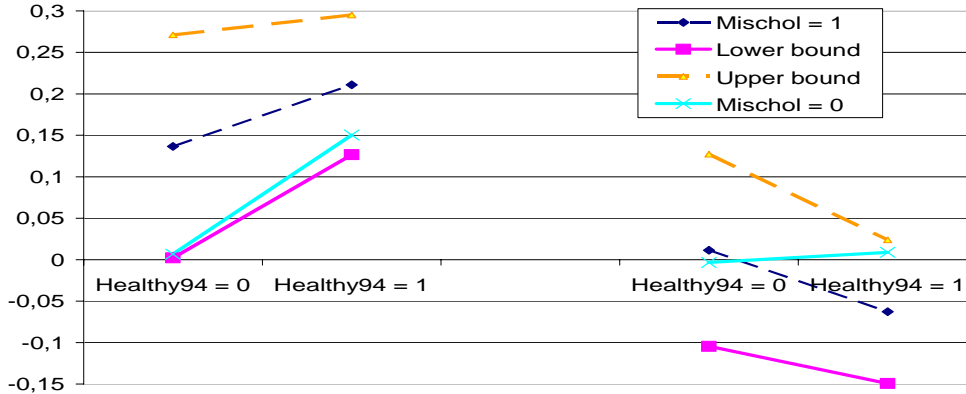
3. Identifying the effect of socio-economic history on health events and on socio-economic events.

To identify the effect of socio-economic history on socio-economic events (γ_{13}) and on health events (γ_{23}), the instrument included in the socio-economic history equation is a dummy variable for whether or not individuals believe that in the long run people get the respect they deserve in this world (we label it RESPECT). We thus consider the first, the second and the third equations of model (1) to see whether such instrument is valid.

Simple mean calculations show that the average unemployment duration is 6.43 month for the sub-sample of individuals with RESPECT = 1 and 7.23 month for those with RESPECT = 0. Again, similar patterns emerge when one distinguishes between males and females and between countries. This suggests that, on average, those who believe that in the long run people get the respect they deserve in this world, experience shorter unemployment durations.

Like in the previous sub-section, we consider two alternative versions of the first equation of model (1): one where individuals' current socio-economic status is measured by their position in the national household income distribution (an ordered probit model) and one in which it is measured by individuals' expected household income, corrected for cross-country differences in purchase power. Again, in none of the estimated specifications has the coefficient on the RESPECT dummy turned out to be significant. Note that this holds even when separate regressions are ran depending on whether individuals have experienced unemployment over the last ten years or not.

Taking the second equation of model (1), we now can check whether the RESPECT variable could also be legitimately excluded from the health events equation. We estimate the latter equation, treating its left-hand side variable (ADL-scores) as if it were continuous. The coefficient on the RESPECT variable is again insignificant whether one considers the whole sample or distinguishes between those having experienced unemployment over the last ten years and those who have not.

Now take the socio-economic events equation (using expected household income as the left-hand side), exclude the number of unemployment months from the right-hand side and separate the whole sample according to whether individuals have experienced some unemployment over the last ten years or no unemployment at all. For one to be allowed to exclude the RESPECT variable from the socio-economic events equation, the residuals from the estimated specification, there should be no significant difference between the residuals for RESPECT = 0 and for RESPECT = 1. We also proceed in the same way with the health events equation, treating ADL-scores as if they were continuous measures. For those having experienced some unemployment and those who have not, respectively, Fig. A2.3 reports the mean residual for RESPECT = 0, the lower and upper bounds of its 95% confidence interval as well as the mean residual for RESPECT = 1. The residuals from the health events equation are reported on the left of the figure whereas those from the socio-economic events equation are reported on the right of the figure.

Fig. A2.3. Residuals from the health events and the socio-economic events equations (effect of socio-economic history)
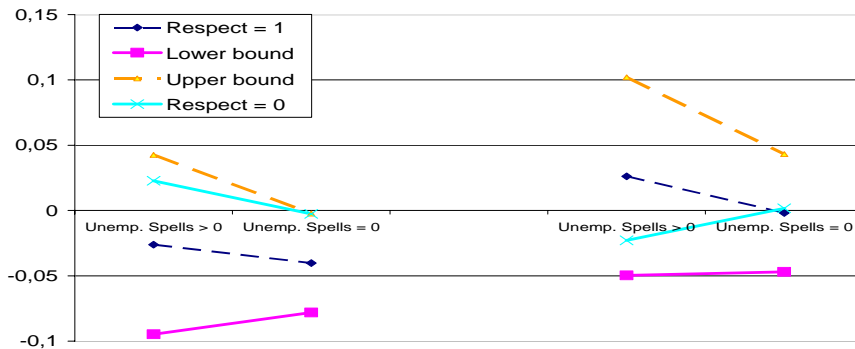
Appendix 3: Descriptive statistics

Table A3.1: Descriptive statistics of the variables per country

Variables	France	Denmark	Netherlands	United-Kingdom	Greece	Finland
Tastes & Behaviour (TB)						
BMI at 40	24.97	25.44	25.90	25.96	25.92	25.22
Health events (HE): ADL-score (%)						
0	25.52	22.52	25.80	21.95	33.91	48.10
1	18.25	27.54	15.70	19.59	26.74	20.04
2	14.16	14.94	12.70	12.89	11.91	10.76
3	11.07	9.70	9.90	10.14	6.16	8.44
4	7.78	7.02	7.70	5.51	4.30	3.80
5	8.28	4.68	5.70	6.00	3.53	3.16
6 and more	14.95	13.60	22.50	23.93	13.42	5.69
SES history (SESH)						
Non-censored observations (%)	28.91	30.99	10.40	48.33	32.79	28.17
Unemployment duration in months over 10 years	29.09	25.96	16.26	21.54	19.91	22.54
Health history (HH)						
Very good/good self-assessed status	79.76	81.72	71.00	72.44	84.36	80.80
Childhood environment						
Number of persons per room	1.28	1.27	1.35	1.05	0.56	1.54
Monoparental family (%)	13.06	9.03	9.37	14.96	14.19	12.24
Toilet inside (%)	78.86	91.30	92.89	86.91	42.06	67.30
Bed sharing (%)	18.34	3.12	24.92	12.40	42.16	7.81
Living in a crime area (%)	4.79	4.35	1.95	19.00	3.00	6.54
Parents' wealth is below average (%)	24.93	29.54	40.1	41.18	40.80	27.00
Parents' wealth is just about average (%)	51.74	52.67	51.20	47.06	48.50	59.00
Parents' wealth is above average (%)	23.33	17.79	8.70	11.76	10.70	14.00
Parents' disease (%)	21.64	18.95	31.20	31.89	17.26	22.36
Missed school for health problems (%)	15.95	10.26	20.70	25.89	5.59	6.96
Lifesmoke (%)	62.41	79.38	71.99	73.82	63.44	86.08
Respect (%)	46.86	42.03	40.30	43.50	62.76	50.42
Female (%)	52.94	55.52	53.35	41.63	56.24	38.82
Age	55.89	56.51	57.48	54.14	54.92	55.88
Single (%)	3.99	6.02	8.88	15.85[17]	6.36	7.79
Married (%)	67.89	75.27	76.42	64.61	76.48	74.83
Divorced (%)	23.63	14.92	9.80	13.98	7.37	12.74
Widowed (%)	4.49	3.79	4.90	5.56	9.79	3.64
Compulsory education level (%)	10.37	8.96	35.00	19.15	35.82	4.22
Uppers secondary education level (%)	37.79	33.44	31.90	43.09	39.66	16.24
Higher education level (%)	51.84	57.30	33.10	37.76	24.52	78.69
Observations	979	760	936	919	989	458

[17] In contrast to the other countries, the "single" category in the UK includes people living together but not married.

Table A3.2. SOCIOLD data compared to the sub-sample of individuals aged 50 or more from the 2001 wave of the ECHP: all individuals

Variables	Denmark		Finland		France		Greece		Netherlands		United Kingdom	
	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP
Salaried	0.69	0.61	0.77	0.44	0.52	0.43	0.30	0.30	0.42	0.52	0.52	0.60
Self employed	0.07	0.04	0.12	0.12	0.06	0.07	0.11	0.25	0.06	0.05	0.13	0.10
Unemployed	0.03	0.03	0.02	0.06	0.08	0.05	0.05	0.02	0.11	0.05	0.02	0.02
Retired	0.14	0.27	0.06	0.36	0.23	0.29	0.27	0.16	0.00	0.01	0.17	0.15
Domestic activities	0.01	0.02	0.01	0.01	0.03	0.16	0.00	0.26	0.14	0.28	0.04	0.06
Other activities	0.06	0.02	0.02	0.01	0.09	0.00	0.27	0.02	0.26	0.10	0.12	0.07
Female	0.56	0.52	0.39	0.50	0.53	0.53	0.56	0.52	0.53	0.51	0.43	0.54
Age	56.5	57.3	52.9	57.0	55.9	56.6	54.9	54.8	57.5	55.8	54.3	54.1
Married	0.75	0.74	0.78	0.75	0.68	0.80	0.76	0.88	0.76	0.80	0.73	0.74
Single	0.06	0.07	0.08	0.09	0.04	0.06	0.06	0.04	0.09	0.05	0.07	0.09
Widowed	0.04	0.05	0.02	0.06	0.05	0.06	0.10	0.06	0.05	0.05	0.04	0.04
Divorced	0.15	0.14	0.12	0.11	0.24	0.09	0.07	0.02	0.10	0.10	0.13	0.13
Born in the country	0.98	0.97	0.99	0.96	0.90	0.90	0.96	0.97	0.97	0.96	0.85	1.00
Higher education	0.57	0.28	0.79	0.29	0.52	0.20	0.24	0.12	0.33	0.00	0.36	0.41
Upper secondary	0.33	0.48	0.20	0.35	0.38	0.07	0.40	0.20	0.32	0.00	0.43	0.14
Compulsory schooling	0.09	0.24	0.01	0.37	0.10	0.73	0.36	0.68	0.35	1.00	0.20	0.45
Good/very good health	0.71	0.68	0.54	0.49	0.61	0.50	0.71	0.77	0.60	0.66	0.58	0.68
Observations	897	888	474	2 497	1 003	2 296	1 001	2 829	971	2 232	727	2 579

Table A3.3. SOCIOLD data compared to the sub-sample of individuals aged 50 or more from the 2001 wave of the ECHP: salaried workers

Variables	Denmark		Finland		France		Greece		Netherlands		United Kingdom	
	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP	Sociold	ECHP
Female	0.58	0.48	0.40	0.54	0.52	0.49	0.34	0.41	0.50	0.37	0.42	0.51
Age	55.8	55.6	52.3	52.0	54.5	53.8	51.8	52.0	56.0	54.1	53.5	52.5
Part-time	0.10	0.10	0.06	0.09	0.13	0.10	0.09	0.05	0.34	0.22	0.16	-
Private sector	0.44	0.52	0.51	0.53	0.60	0.58	0.69	0.58	0.53	0.65	0.59	0.68
Married	0.78	0.77	0.77	0.75	0.66	0.78	0.79	0.89	0.76	0.80	0.74	0.73
Single	0.05	0.07	0.08	0.09	0.04	0.07	0.08	0.05	0.09	0.06	0.07	0.09
Widowed	0.02	0.04	0.02	0.02	0.04	0.04	0.04	0.04	0.03	0.02	0.04	0.03
Divorced	0.14	0.12	0.12	0.14	0.26	0.12	0.09	0.03	0.11	0.12	0.14	0.15
Born in the country	0.98	0.99	0.99	0.96	0.89	0.91	0.94	0.96	0.97	0.98	0.84	1.00
Higher education	0.59	0.36	0.78	0.39	0.55	0.28	0.37	0.26	0.39	0.01	0.39	0.44
Upper secondary	0.34	0.48	0.21	0.38	0.37	0.06	0.40	0.24	0.29	0.00	0.40	0.14
Compulsory schooling	0.07	0.16	0.01	0.23	0.08	0.65	0.23	0.50	0.32	0.99	0.21	0.42
Good/very good health	0.88	0.80	0.61	0.61	0.76	0.55	0.78	0.89	0.65	0.76	0.56	0.68
Observations	681	543	423	1 104	573	999	418	858	469	1 149	476	1 542

Appendix 4: Model estimates ignoring both endogeneity and simultaneity

Table A4.1: Non simultaneous form estimates

Equations	TB (OLS)		HH (Probit)		HE (Poisson model)		SESH (Tobit)		SESE (Ordered probit)	
Variables	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE
Endogenous variables										
Tastes and Behaviour (TB)			-0.017***	0.003			0.017**	0.008		
Health History (HH)					-0.662***	0.029			0.249***	0.037
SES History (SESH)					0.083***	0.010			-0.151***	0.012
Parents' disease	0.521***	0.167	-0.113***	0.045						
Crime area	0.604*	0.343								
Number of persons/room	0.180	0.533								
Monoparental family	0.151	0.321								
Toilet inside	-0.150	0.254								
Bed sharing	-0.056	0.216								
Parents well off	-0.546**	0.239								
Missed school			-0.377***	0.053						
Lifesmoke	0.478***	0.172								
Respect							-0.234**	0.110		

*** significant at 1%; ** significant at 5%; * significant at 10%.

Table A4.1 (continued): Non simultaneous form estimates

Equations	TB (OLS)		HH (Probit)		HE (Poisson model)		SESH (Tobit)		SESE (Ordered probit)	
Variables	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE	Coeff	SE
Demographic characteristics										
Female	-0,589***	0,163	-0,132***	0,041	0,179***	0,029	0,108	0,122	-0,659***	0,031
Age					0,095*	0,058			0,004	0,054
Age²					-0,072	0,052			-0,011	0,049
Upper secondary education	-0,336	0,222	0,202***	0,056	-0,065*	0,037	-0,196	0,172	0,176***	0,038
Higher education	-0,687***	0,232	0,220***	0,056	-0,241***	0,038	-0,426***	0,176	0,543***	0,041
Married	-0,561	0,394			-0,125***	0,049			0,380***	0,053
Divorced	-0,863**	0,449			-0,027	0,058			0,012	0,065
Widowed	-0,259	0,547			0,144**	0,070			0,202***	0,086
Country fixed effects[1]										
France	-0,681***	0,265	-0,187***	0,070	0,275***	0,050	-0,029	0,193	-1,051***	0,053
Denmark	-0,151	0,347	-0,167**	0,074	0,229***	0,051	-0,086	0,209	-0,374***	0,051
Netherlands	-0,110	0,291	0,382***	0,066	0,375***	0,049	-2,946***	0,229	-0,683***	0,047
United-Kingdom	-0,385	0,281	-0,387***	0,070	0,423***	0,049	0,946***	0,188	-0,117**	0,051
Finland	0,193	0,317	-0,177**	0,090	-0,166**	0,075	-0,344	0,258	-0,609***	0,063
Constant	27,440***	7,947	1,477***	0,111	-1,838	1,618	-1,455***	0,328	Cuts	not reported
Number of observations : 5041										

*** significant at 1%; ** significant at 5%; * significant at 10%.

(1) Greece is the omitted country

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