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Health, income and individual characteristics: three microeconomic applications on elderly Europeans

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General introduction

The relationship between income, income inequalities and health is an issue which has been questioned by many disciplines such as economics, sociology and public health. Public policies to reduce health inequalities, based on redistribution in terms of income or based on reducing costs associated to the access of healthcare for the poorest, are present in many countries. These policies refer on the fact that there is a relationship between individual income and health status, such that their efficiency relies on causal effects between health and income. In this thesis, we study the economic and social determinants of differences in individual health outcomes among people aged 50 years old and over in Europe. The idea is to focus on the determinants which drive, induce and result in health inequalities. Health inequalities are the structural variation in health across a population. The first demographic works begin in the eighteenth century and shed light on important differences in health between individuals. For instance, [Villermé \[1830\]](#) shows that there are differences in mortality between the well-off and the least well-off neighborhoods of Paris. Since then, during the 20th and the 21st centuries, life span increased considerably (see figure 1). However, during the same period, differences in mortality between socio-economic groups did not disappear even though large improvements in medicine and medical technology have been done. These inequalities seem contradictory when considering the implementation of public health policies and redistribution. In 1945, a social protection system was set up in France and it was considered as the best health system in the world in 2000 ([WHO \[2000\]](#)). These observations led us to question the causes of these inequalities, and in particular, to study links between health outcomes and socio-economic and individual characteristics. Moreover, due to the increasing number of aged individuals in Europe, policy makers need appropriate information to curb these inequalities and to improve health status and health environment of these individuals. According to the United Nations, a society where the percentage of population over 65 years old is more than 7 percent is considered as an “ageing society” and an “aged society” is a society where this percentage exceeds 14 percent ([United Nations \[2015\]](#)). Over the decades, the proportion of elderly has substantially increased and many developed countries became either ageing or aged societies. As a result, this thesis focuses on a sample of

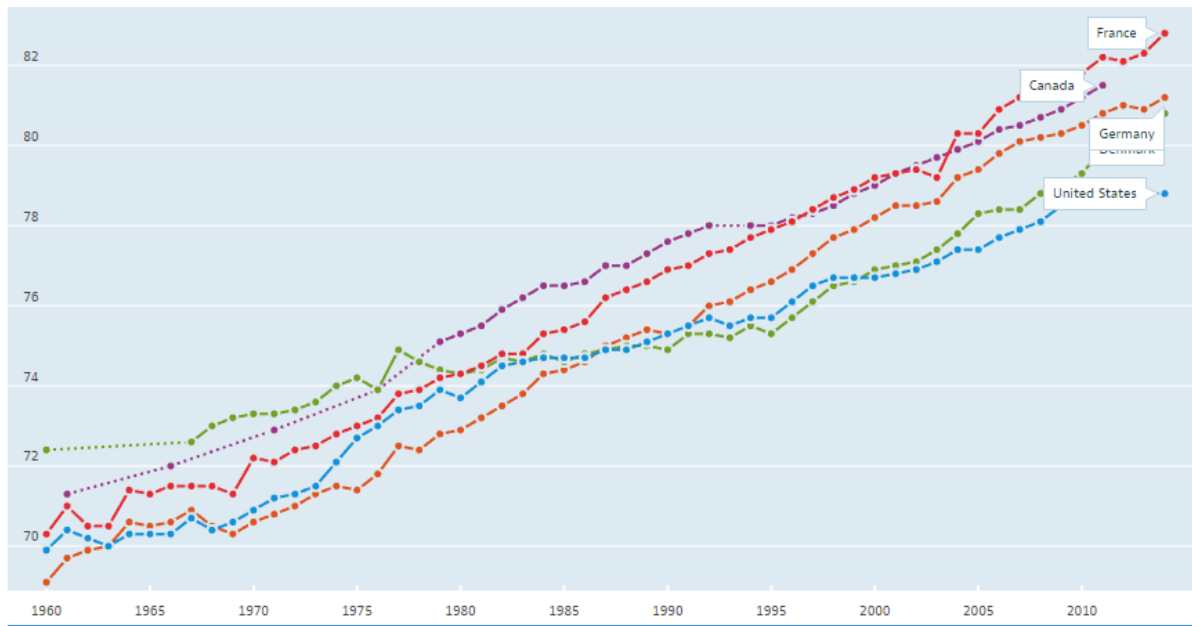


Figure 1 – Life expectancy at birth in years (1960 - 2015)

Source: OECD

individuals in their late middle age from the Survey of Health Ageing and Retirement in Europe (SHARE). SHARE is part of a context of an ageing population. It is the European Commission which has identified the need for scientific knowledge about ageing people in Europe since in 2050, one in three Europeans will be over 60 years old and one in ten will be over 85 years old.¹ Individuals over the age of 50 start to experience negative health shocks which create, at least partially, differences in health and income. Thus, governments need further investigation about health economics in order to implement accurate public policies among ageing people. The growing weight of the elderly in the population, combined with the fact that they are the ones who face an increasing number of health issues, justifies the deeper focus of scientific research on this part of the population. The elderly are often pointed to be the main reason for the steady rise in health spending that developed countries know for years. Over the last decades, governments in developed countries substantially increase the percentage of health expenditure in the GDP in order to improve health and access to health care (see figure 2).

1. Numbers estimated by the European Innovation Partnership on Active and Health Ageing project, which is part of the Health 2020.

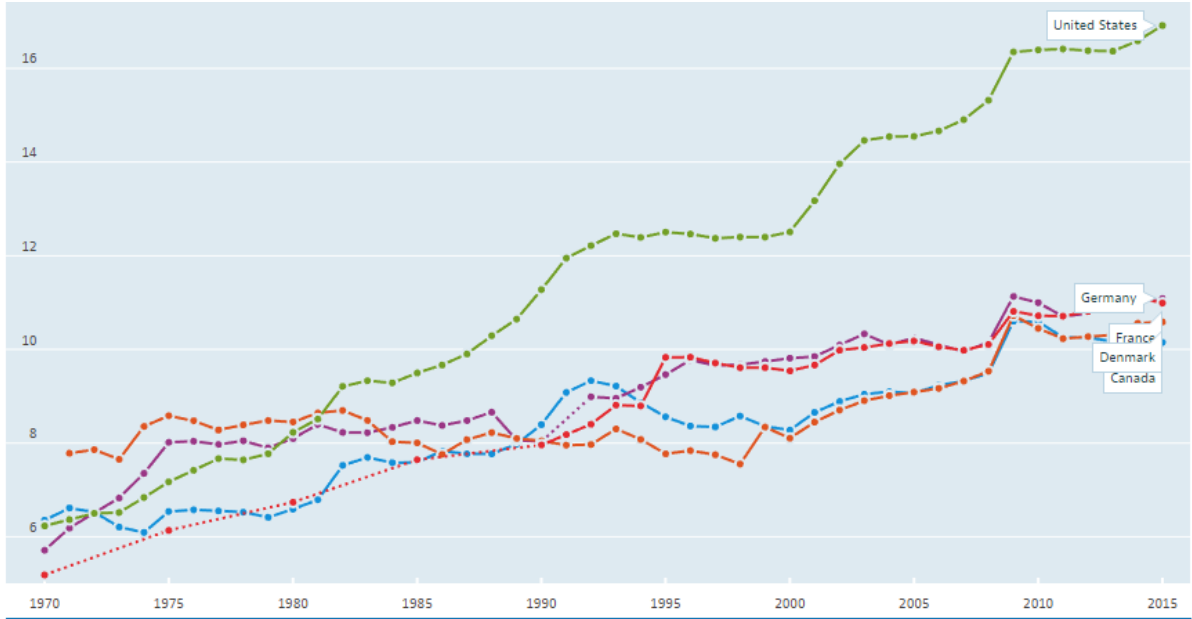


Figure 2 – Health expenditures, total, percentage of GDP (1970 - 2015)

Source: OECD

Health and income: what relationships?

The relationship between health and income goes back from a long time. In the twentieth century, researchers focus on cross-sectional relationship between mortality and economic level, specifically between national infant mortality rates and levels of income (Preston [1975]). For instance, Adelman [1963] finds that income and mortality rates are negatively correlated. Preston [1975] explains that additional income is invested in various domains (housing, hospitals), such that it decreases mortality. Moreover, when considering that growth of income is positively associated with gains in life expectancy, then high-income countries might have larger gains in life expectancy when compared to low-income countries which have slowly growing economies. Indeed, Preston [1975] is one of the first highlighting a concave relationship between health and income. He further states that “the obvious explanation for the non-linearity of the mortality/income relationship is that it reflects diminishing returns to increases in income”. As a result, from an early stage in the debate, it was argued that individuals who are better off financially tend to have better health, which is underlined by the existence of a positive relationship between socioeconomic status and health. This relationship has been studied many times and is now known as the health-income gradient. This term has been implemented in order to highlight the two-way mechanism between health and income which is difficult to disentangle. On one hand, the health-income gradient can be defined as the gradual evolution of health with income (or wealth). On the other hand, one might also think

of the reverse association stating that health status is important to be productive in the labor market, such that income is then positively affected. In other words, this gradient shows a positive correlation between health and income. In England, [Marmot et al. \[1991\]](#) focus on civil servants and find an inverse association between employment grade and prevalence of diseases, such that low income implies a higher risk of being sick. In the literature, [Smith \[1999\]](#) gives a very good example of the health-income gradient. In 1984, an American in excellent health had a median financial wealth of \$68,300 while an individual in poor health had a median wealth of \$39,200. The author observes that this gap increased over time. People with excellent health saw their median wealth increase from \$68,300 to \$99,300 between 1984 and 1989, while it decreased from \$39,200 to \$36,000 for the ones in poorer health. On the other hand, between 1979 and 1989, an individual with incomes below \$10,000 lived 6.6 years less than someone who earned more than \$25,000. [Cutler et al. \[2006\]](#) explain that there is a complex link between socioeconomic status and health which is more in favor of the latter causing the former. However, since their analysis is based on cross-sectional findings, it is difficult to highlight causal links.

Health and income being related is a well-established fact. Researchers then focus on the role of income inequalities in health inequalities. In this case, inequalities in health refer to the close relationship between health and membership in a group characterized by incomes, where income is an individual social determinant. As explained earlier, [Preston \[1975\]](#) highlights a non-linear relationship between health and income due to the concavity which is at play. However, income inequalities in a society might also be an important determinant of health status. Income-related health inequalities have gained much attention in recent years because they have increased in many developed countries during the past three decades. Socially based disparities in health status continue to plague even the most egalitarian societies despite public policies that aim to reduce or eliminate them. Indeed, in the last few years, policy makers aim at reducing health inequalities. They usually focus on access to healthcare given that the role of such policies is to improve health of lower income groups ([WHO \[2009\]](#)). However, health equity might also be favored by redistribution policies in terms of income ([Ettner \[1996\]](#); [Wagstaff et al. \[2003\]](#) or [Carrieri and Jones \[2016\]](#)). Early studies use aggregate data to test the correlation between income inequalities and health. Over the past decade, various works by Wilkinson show evidence of a relationship between income inequalities and life expectancy across a number of industrialized countries ([Wilkinson \[1992\]](#) or [Wilkinson \[1996\]](#)). While he reports correlation coefficients, a growing body of literature tests this hypothesis using regression frameworks. A link between income inequalities and health measures has been discerned at different levels (countries with [Waldmann \[1992\]](#), or across states, counties

and cities within nations with [Kaplan et al. \[1996\]](#); [Kawachi et al. \[1997\]](#)). Besides, some studies find an association between income distribution across U.S. states and state-level measures of alcohol consumption ([Marmot \[1997\]](#)) and smoking ([Kaplan et al. \[1996\]](#)). Moreover, independent of actual income levels, the distribution of income within countries and states has been linked to rates of mortality ([Kawachi and Kennedy \[1999\]](#)). One explanation is that underinvestment in public goods and the experience of inequalities are both greater in more stratified societies and that these, in turn, affect health. [Adeline and Delattre \[2017\]](#) find a negative relationship between income inequalities (either measured with a Theil index or a Gini coefficient) in a country and health status of individuals in this society.

The health-income gradient can be underlined by three hypotheses linking health with both income and income inequalities. The first one, called the Absolute Income Hypothesis, corresponds to the positive and concave relationship between income and health ([Preston \[1975\]](#)). The second hypothesis is a strong version of the Income Inequality Hypothesis and states that health status is driven by income inequalities within a geographical area. In this way, health status of all individuals is affected by an increase or a decrease in income inequalities. Finally, the last hypothesis linking health, income and inequalities is a weak version of the Income Inequality Hypothesis which asserts that income inequalities are a threat to individuals placed at the lower end of the income distribution. In this way, income inequalities have more negative impacts on low income people than on high income people. These hypotheses have been empirically tested, mainly in the United States or with a focus on one or two hypotheses only, and results are varied ([Carrieri and Jones \[2016\]](#); [Van Doorslaer et al. \[2004\]](#); [Hildebrand and Van Kerm \[2009\]](#)). Thus, there is a need to study these three hypotheses at the same time to have a better understanding of the relationships linking health to income and income inequalities, in order to draw some comparisons and conclusions.

Behind these hypotheses, there might be mechanisms through which income inequalities imply health inequalities. For public policies purpose, understanding the diversity of mechanisms is of interest. Indeed, understanding mechanisms by which income leads to variations in health outcomes help to understand how the latter could be improved. According to [Lynch and Kaplan \[1997\]](#), “social processes and policies that systematically underinvest in human, physical, health and social infrastructure” might in turn affect health status. Moreover, social environment, due to income inequalities, as perceived by individuals, might have direct consequences on health. Subsequently, [Kawachi and Kennedy \[1999\]](#) detail these relationships by highlighting three pathways linking income

inequalities to health. The first one is that disinvestment in human capital (at the individual level such as education) or social goods (at the aggregate level such as the percentage of health spending in the GDP) are linked to income inequalities. Disinvestment in such domains generate disparities in income afterwards such that interests of high income individuals diverge from low income ones, which in turn might favor health inequalities. The second pathway through which income inequalities favor health inequalities is the erosion of social capital, defined as “features of social organization that facilitate cooperation for mutual benefit”. In other words, social capital is interpreted as the set of collective resources an individual has access to (such as community solidarity). Income inequalities might result from a gap in expectations between low income people and high income individuals due to an increasing level of mistrust for instance. Finally, the last mechanism, through which income inequalities imply health inequalities, is due to social comparisons between individuals which might be stressful. Many communities have a common cultural model of the standard of living, such that when individuals do not succeed in achieving this cultural model of lifestyle, this might create income inequalities and thus health inequalities. This aspect can be identified using, as proxies, whether individuals face a stress period, whether they have been discriminated or their relative positions to others when it comes to school. Implementing adequate public policies to favor investments or social cohesion might thus reduce health inequalities. More recently, the World Health Organization (WHO)’s Commission on Social Determinants of Health also brings to the forefront these channels in a different way ([Solar and Irwin \[2010\]](#)). Nested mechanisms may be responsible for inequalities in the population health. Socioeconomic and policy contexts create inequalities due to socioeconomic resources, gender, race and ethnicity. These mechanisms are mainly “intra-individual” ([Montez et al. \[2017\]](#)). On the other hand, “extra-individual” factors shape population health into seven categories: governance, macroeconomic policies, social policies, public policies, culture and societal values, social capital or cohesion (as [Kawachi and Kennedy \[1999\]](#)), and the health system.² Focusing on the European ageing population, there are no available researches which study these mechanisms.

Does income impact health or does health impact income?

Social inequalities in health exist in all countries, regardless of insurance that regulates access to health care regime ([Potvin et al. \[2010\]](#)). In Europe, there are still huge differences among countries concerning either health or income. These differences were

2. The WHO refers to these mechanisms as the “socioeconomic and political contexts”.

higher in the past years than today thanks to the whole process of the European unification, which directly aims at removing these differences and promoting cohesion and integration into a single economic area for the member countries. According to the World Development Indicators, which are indicators at an aggregate level provided by the World Bank, over the period 1960-2012, average real GDP per capita increased by 140% in the European countries, life expectancy at birth increased by 15% (from 69.02 to 79.10) and infant mortality decreased by 89% (from 39.94 infant deaths per 1000 live births to 4.20). Thus, causal links between income and health are of importance. During the Report of the Commission on Macroeconomics and Health, the former WHO Director-General [Harlem Brundtland \[2001\]](#) explains that “a healthy population is a prerequisite for growth as much as a result of it”.

Correlation between health and income being demonstrated, as well as pathways through which income inequalities might result in health inequalities, the question is now on the direction of causal links between health and income: is health improved through increase in income, or, are healthier individuals more productive such that this increases income? Many studies focus on the correlation between the two since causal statements are a difficult task due to endogeneity issues and bidirectional causal links which might be at play. On one hand, relationship can run from income to health ([Apouey and Clark \[2015\]](#)). The idea is that higher income helps individuals access to goods and services which provide and enhance individual health status. On the other hand, causal link might run from health status to individual income. Indeed, poor health status may influence income, by reducing the ability to work ([Michaud and Van Soest \[2008\]](#)). Thus, it is important to highlight causal statements in order to implement public policies whose aim is to curb income-related health inequalities. [Deaton \[2002\]](#) explains that “policy cannot be intelligently conducted without an understanding of mechanisms; correlations are not enough. Income might cause health, health might cause income, or both might be correlated with other factors; indeed, all three possibilities might be operating simultaneously”. For instance, whether causal links run from income to health, imply that policy makers should favor public policies whose aim is to increase/favor income and redistribution. On the other hand, if causal links run from health to income, then governments should favor investments in health domains.

The dynamic interaction of changing humans in changing environments is not thought to be captured adequately by simple relationships among variables at a point in time, such that there might also be persistence in relationships. In order to highlight causal links between health and income, it is thus important to highlight temporal dynamics

between the two. To do so, researchers can focus on the first definition of the concept of causality which is derived from the seminal work of [Granger \[1969\]](#), where the definition is expressed in terms of predictability. The latter distinguishes lag causality from instantaneous one. Thus, Granger causal tests aim at investigating dynamic relationships between two variables. In this way, on one hand, individual health at time t (h_{it}) is a function of individual health in the past ($h_{i,t-1}$), income in the past ($inc_{i,t-1}$), and other individual socioeconomic variables at date t (X_{it}), such that:

$$h_{it} = f(h_{i,t-1}, inc_{i,t-1}, X_{it}) \quad (1)$$

As a result, income Granger-causes health if the whole history of income allows to predict the current value of health, compared to a situation where only the history of health is used. On the other hand, health Granger-causes income whenever individual income at date t (inc_{it}) is a function of the history of income ($inc_{i,t-1}$), health at the previous date ($h_{i,t-1}$) and individual socioeconomic variables (Y_{it}), such that:

$$inc_{it} = g(inc_{i,t-1}, h_{i,t-1}, Y_{it}) \quad (2)$$

This approach includes a phenomenon of persistence for individual income (*resp.* health status) in the relationship. In this way, testing and finding evidence of bidirectional Granger causal links between health and income means that there is a feedback between the two outcomes. Causal measurements are useful when it comes to the implementation of public policies. Particularly, Granger causality allows to identify whether an implemented policy was efficient.

The direction of the causality between health and income is considered as an important issue and this is debated among economists since the lack of a clear and true understanding is a shortcoming for policy makers who aim at narrowing health inequalities and thus improving health. The difficulty in disentangling causes and effects is due to endogeneity. Granger causality is defined by an auto-regressive form which might result in a biased estimation of the phenomenon of persistence (past income or past health) is correlated with the error term. Moreover, the Granger causality involves a delayed causality of income (*resp.* health) on health (*resp.* income) in a manner that disparities are created at all time. Again, the delayed explanatory variable might be endogenous when these are correlated with the error term. In the literature, authors use instrumental variables methods or exogenous shocks to investigate causal links between health and income, and to solve endogeneity issues. [Halliday \[2017\]](#) employs data from the Panel Study of Income and Dynamics (US) to investigate the Granger causal link running from income to health. He

implements a GMM procedure on a model in first-differences, and uses further lag variables as instruments. His results establish a causal link running from income to health in the case of married individuals. Then, [Michaud and Van Soest \[2008\]](#) use instrumental variables method to consider this relationship. They instrument health with the onset of critical health conditions (like cancer for severe condition or high blood pressure for mild condition). Using, the Health and Retirement Study with dynamic panel data models, they find strong evidence of causal effects of health of household members on household wealth.

Moreover, instrumental variables techniques are also useful to estimate local average treatment effects ([Imbens and Angrist \[1994\]](#)). Whenever researchers seek to estimate effects of a policy on the entire population or on a subset (defined by predetermined characteristics), they should turn to randomized evaluations ([Duflo and Kremer \[2005\]](#)). Identification of program effects could be a difficult task but revolved with experimental researches which allow to highlight multiple channels of causality. Randomized evaluation methods consist in determining a program impact, mainly in developing countries, where policy makers want to understand benefits of programs. This can be considered as a public good because it offers efficient guidance to governments when they know which programs work. [Banerjee and Duflo \[2009\]](#) are in favor of cohesion between economists and governments in order to implement efficient randomized experiments. In this way, insights of researchers might guide policy makers. To do so, governments need to authorize the researcher to be a coexperimenter in a sense that he can redefine the question to be answered, according to his knowledge of previous experiments, results and theory. Even if all programs cannot be evaluated with randomized evaluations, [Duflo and Kremer \[2005\]](#) explain that the ones which target individuals are likely to be strong candidates. The idea behind program evaluations is to investigate and compare individuals who benefit from the program, from others who do not. Thus, one has to establish a reliable comparison group defined as individuals who would have outcomes similar to the ones exposed to the program in the absence of the program. Moreover [Glazerman et al. \[2002\]](#) explain that experimental methods might be a solution to omitted variable bias which is a major issue when non-experimental methods are used in developed countries. In health economics literature, [Giné et al. \[2010\]](#) implement a randomized controlled experiments to help smokers to quit. Treatment group is composed of Filipino smokers who invest money in a savings account for six months. After this time period, their savings are lost if they fail a urine test for nicotine. Randomly chosen smokers were 3 percentage points more likely to succeed the test and thus quit smoking. A known randomized controlled experiment in health economics is the one on moral hazard known as the RAND's Health

Insurance Experiment (HIE).³ This experiment was held between 1974 and 1981 and randomly assigned health insurance plans with different levels of cost sharing (from full coverage to almost no coverage) to many Americans (representative sample of households with adults under 62 years old). [Aron-Dine et al. \[2013\]](#) explain that results from this experiment are still considered as a reference to predict the impact of health insurance reforms on medical spending, as well as for designing actual insurance policies. Thus, randomized experiments are an alternative method to highlight causal links, and to highlight the importance and efficiency of public policies in smaller economic area before their implementation in broader areas.

However, randomized controlled experiments are criticized due to selection biases or attrition which arise. Indeed, individuals who are not affected by the public policy and the ones who participate are different such that it is difficult to establish a good comparison group. This issue results in pre-existing differences which must be controlled. Other alternative techniques are settled to obtain impacts of specific programs which aim at reducing the issues of selection bias and/or omitted variable.

First, the propensity score matching attempts to find a control group that is almost identical to the treatment group in terms of individual socioeconomic characteristics. In this way, non-treated individuals should have the same probability of being treated as individuals who are actually treated. For instance, [Jones and Richmond \[2006\]](#) use propensity score matching to highlight causal impact of risky behavior (alcohol consumption) on earnings on US citizens. They find that alcoholism contributes to loss in productivity which in turn decreases earnings.

Then at a more aggregate level, the difference-in-difference technique compares trends in the outcomes between regions which are impacted by the program and similar regions where the program is not settled. For instance, [Halla and Zweimüller \[2013\]](#) use a fixed-effects difference-in-differences approach to show that health shocks, measured by accidents occurring on the way to and from work, negatively impact employment and income for Austrian workers.

Finally, we can also suggest regression discontinuity design which uses discontinuities to identify effects of a public policy, by comparing individuals who are treated from the ones who are not but almost. Individuals are selected on the basis of socioeconomic characteristics such that some of them might respond to almost all characteristics. [Buser et al. \[2016\]](#)'s paper makes use of a regression discontinuity approach to identify the effects of

3. For more information, see website of RAND's Health Insurance Experiment (HIE): <https://www.rand.org/health/projects/hie.html>.

positive income changes on height and weight of young children in a developing country. They use change in the eligibility criteria for receipt of an unconditional cash transfer in Ecuador as a source of income variation. They find that children in families which receive the transfer are taller and weigh more.

In order to highlight causal links between health and income, other techniques are also available. Investigating impact of income uncertainty on health might be useful. Given an average income level, income uncertainty, which manifests itself in volatility of income, might have an impact on health outcomes. Modeling income processes to compute income variances can be useful to identify effects of income uncertainty on health. All over the life cycle, individuals face unexpected events which impact the level of income, either permanently or transiently. Differentiating between variances coming from permanent and transitory components of income is of importance because variances that do not have the same durability should have very distinct effects on health. In [Adeline et al. \[2018\]](#), they exploit this approach on a Canadian population, and, while controlling for income levels, find evidence that transitory income uncertainty deteriorates well-being. This suggests that public policies aiming to reduce income volatility coming from transitory shocks, can have positive and long-term effects on health outcomes of Canadians aged 50 and over.

In this way, estimation of the variances of transitory and permanent shocks comes from [Carroll and Samwick \[1997\]](#)'s methodology. Their methodology follows two main steps. First, the predictable growth from the income process is removed in order to compute a specific time series estimation of the variances of transitory and permanent components, the latter coming from exogenous income shocks. In this way, to obtain variances, the first step is to define the logarithm of permanent income as a random walk with a shock to permanent income, and a vector of individual characteristics. Then, the logarithm of current income is given by the permanent income plus a transitory shock. Permanent and transitory shocks are considered as Gaussian white noises uncorrelated with each other and uncorrelated at all lags. In this way, differences in income between d years are defined, such that, when we remove the predictable growth from the latter, we end up with the variance of the differences in income. These differences correspond to the sum of the variance of the permanent component of individual income and the variance of the transitory one. For each respondent, a series containing all possible differences in income is constructed to estimate both variances. In [Adeline et al. \[2018\]](#), this is done for each respondent while they were between 30 and 55 years old. As a second step, they estimate the effects of these components on mental health, life satisfaction and other health domains for Canadians aged 50 and over. In other words, they investigate the relation-

ship between health and income over the life cycle by considering income uncertainty, occurring during working-age (from 30 to 55 years old), and their effects on different health outcomes afterward (from 50 to 75 years old). Their results suggest that transient components of the variance of income (period of unemployment, illness, ...), occurring during a working-age period, deteriorate mental health, life satisfaction, and other health domains of Canadians aged 50 and over. Thus, public policies in Canada aiming to reduce income volatility, coming from transitory shocks, can have positive and long-term effects on mental health and well-being of the elderly.

Finally, when investigating causal links between health and income, simultaneity between the two outcomes might be at play, resulting in endogeneity issues as explained earlier. In this way, simultaneous equations approach is useful because it considers unobserved individual factors which may be common to both health and income. In the case of the health-income gradient, such factors might be physical maturity (some individuals are “physically stronger” than others due to their genetics) or intellectual capacity (some individuals have intellectual abilities which might in turn affect their income and health positively). These factors are considered in the error terms such that endogeneity issues arise. As a result, it is important to consider these correlations when studying the health-income relationship. Moreover, in order to consider heterogeneity in the individual effects of both equations, we can use a simultaneous equations model, estimated with Full Information Maximum Likelihood (FIML)⁴, such that health and income do not need to be specifically instrumented.

Simultaneous equations model is thus useful when considering two or more simultaneity issues. In order to constitute such model, each equation needs to have an economic meaning on their own, implying the autonomy requirement of such models. In other words, their meanings are derived from economic theory (Wooldridge [2015]). In this way, the equations need to satisfy both the order and the rank conditions (Wooldridge [2010]). The first condition implies that at least one exogenous variable is excluded from this equation, but included in the other equation(s) of the system. Then, the rank condition states that one equation, for instance composed of *one* endogenous variable, is identified if, and only if, the other equations of the system contain at least, for instance, *one* exogenous variable, and the latter should be excluded from the “first” equation (i.e., the one for which we focus on concerning the rank condition).

Implementing a FIML estimator to study the health-income gradient implies to consider

4. When health status is a qualitative variable, an estimation method like three stage least squares, which consider linear dependent outcomes, is not feasible.

simultaneously equations 1 and 2. In this method, error terms associated to both equations are assumed to be normally distributed and are decomposed into two terms: an individual time-invariant effect and an idiosyncratic error which is time and individual specific. Thus, a specific variance-covariance matrix for individual effects, in which we consider correlation among error terms of both equations, is defined (and we also have a specific variance-covariance matrix for idiosyncratic errors). Then, the associated likelihood needs to be computed in order to estimate the relationship. Whether the correlation among individual effects of both equations is statistically significant or not gives an insight on the necessity to conduct bidirectional simultaneous analyses or univariate ones.

Smoking, a risky behavior constituting a public health issue

In the health-income gradient, income is more generally defined by individual socio-demographic characteristics or by indices at the national level. In the same way, several outcomes can define health, such as self-perceived health status, mental health, physical health, well-being or life satisfaction. Policy makers and governments, whose aim is to curb inequalities, might also need to focus on risky behaviors of individuals such as smoking, drinking, or drug use. Indeed, by promoting prevention of these attitudes, governments will improve the general health of the population. Literature suggests that the risk of poor health is important when a person's socio-economic level is low. On a concrete example, the one of public policies against smoking, the relationship between income and health status characterized by the consumption of tobacco should be explored.

According to the WHO, smoking is the leading cause of premature death in the European Union (EU), responsible for nearly 700,000 deaths each year. The prevalence of smoking is an important component of the difference in mortality rates between countries. Higher smoking rates increase the health risks to smokers and non-smokers who are exposed to the effects of second-hand smoke. According to Eurostat, in 2014, 5.9% of Europeans aged 15 and over smoked at least 20 cigarettes a day and about 12.6% consumed less than 20. Governments therefore need appropriate public policies to reduce smoking among individuals and inform health damages that result from smoking.

Many factors can have an impact on the costs and benefits of smoking, and more precisely, individual heterogeneity. Thus, there is a need to shed more light on the impact of time and risk preferences, underlying income differences, on smoking, when the latter is considered as an economic decision. First, concerning time preferences, present-oriented

people do not take into account detrimental effects of smoking on health. On the other hand, future-oriented individuals integrate future health consequences of smoking when they choose to smoke. Moreover, an important question is on whether individuals know the risks and on whether they make decision to smoke in light of this information. The fact that individuals are cognizant of the risks is one of the key element of the rationality of choices. As a result, the adequacy of public risk perceptions is an essential component of the assessment of the rationality of smoking decisions. Furthermore, smoking is a costly and dangerous habit for health, such that we may wonder whether income plays a role in the consumption of this good. Indeed, individuals when choosing to smoke are going to maximize their utility function under a budget constraint. There are many possible pathways through which earnings can impact consumption of cigarettes. Smoking prevalence and the cumulative amount of tobacco differ across occupations and sectors, and thus across income. On the other side, economic costs of smoking might reflect income reductions associated with smoking on the labor market.

In order to assess risk and time preferences of smokers underlying the reasons for income differences in smoking behavior, researchers need to highlight causal links to implement adequate public policies. However, to do so, one has to be aware of multiple endogeneity issues in these relationships. The latter issues are due to simultaneity between (i) smoking and risk preferences on one hand, and (ii) smoking and income on the other hand. Indeed, concerning risk and smoking, [Viscusi \[1990\]](#) investigate risk preferences among smokers and show that the probability of lung cancer because of smoking is overestimated. [Viscusi and Hakes \[2008\]](#) find that higher risk beliefs decrease the probability of starting to smoke. More recently, [Pfeifer \[2012\]](#) highlights a higher demand for cigarettes for risk lovers, who are more likely to smoke. On the other hand, some authors investigate the reverse relationship in which smoking has an impact on risk preferences. [Viscusi \[1991\]](#) finds that risk perceptions are negatively correlated with smoking. [Lundborg and Lindgren \[2004\]](#) find that risk perceptions from lung cancer are overestimated by non-smokers and smokers, the latter being more likely to have higher risk perceptions. Then concerning smoking and income, smoking prevalence might be related to the job of individuals (and thus to income). In this sense, [Marsh and McKay \[1994\]](#) find that, in Britain, smoking prevalence is higher among individuals who have a smaller income. Similarly, [Hersch \[2000\]](#) explains that people in lower positions (like blue-collar workers) are more inclined into smoking. On the other hand, other studies ([Auld \[2005\]](#)) focus on the impact of smoking on wages and different theories might explain this link. For instance, there might be a self-selection of smokers into occupations which provide a better health coverage with lower wages in exchange ([Lokshin and Beegle \[2006\]](#)).

Thus there is a need to study these outcomes together. Simultaneous equations model, using a FIML estimator, might be useful since a three equations system can be settled:

$$\begin{cases} S_{it} = f(inc_{it}, RP_{it}, TP_{it}, X_{it}) \\ inc_{it} = g(S_{it}, Y_{it}) \\ RP_{it} = k(S_{it}, Z_{it}) \end{cases} \quad (3)$$

As explained earlier, in such model, whether the correlation among the error terms is significant justifies the model.⁵

To conclude, relationship between health and income is a subject in the spotlight for governments and policy makers whose aim is to reduce income-related health inequalities. The whole work of this thesis aims at trying to give answers to the following issues: How does socioeconomic status, and more specifically, income, relate to health? What are the pathways through which income inequalities imply health inequalities? Is it income which impact health or is it the inverse relationship? Do health and income simultaneously determine each other? Then, focusing on a public health issue: what roles do individual heterogeneities play in the decision to smoke? This thesis tries to answer these questions by working on a sample of elderly Europeans on the one hand, and by setting up original econometric methods on the other hand. This thesis is structured around three main axes: the relationship between health and income, the underlying causal impact of these two outcomes, and lastly, individual specificities in the choice of smoking, a health risk. All the analyses are performed using the Survey of Health, Ageing and Retirement in Europe (SHARE), a multidisciplinary and cross-national panel database of micro data on health, socio-economic status and social and family networks of more than 123,000 individuals aged 50 and over from many European countries and Israel.

In this way, the first chapter is entitled **“In-depth overview on the relationship between health and income: microeconomic evidence and pathways”**, in which the first part comes from the paper “Some microeconomic evidence on the relationship between health and income”, a joint article with Eric Delattre, published in *Health Economics Review* ([Adeline and Delattre \[2017\]](#)). In this first part, we intend to bring to the forefront correlations between health, income and income inequalities, in order to evaluate the effects of different income measures on individual health. We seek to identify

5. The appendix part of this Ph.D. dissertation, entitled **“Simultaneous equations model with non-linear and linear dependent variables on panel data”** is derived from a joint-work with Richard K. Moussa. It explains the computational details for the likelihood function of (i) a two equations system with both a linear and a non-linear outcomes; (ii) a three equations system with two continuous outcomes and one binary; (iii) a three equations system with one linear and two binary outcomes.

effects of individual income, as well as, effects of the individual's position on the income scale within a country, and then effects of income inequalities at the country level, on individual health. Main findings suggest that individual income is positively correlated with health of individuals, so that higher income would be associated with better health. Moreover, results show that an increase in income inequalities within a country would be associated with a decline in health status. However, the position of the individual in the salary hierarchy is not associated with individual health. Results are robust when considering another income inequality index or when considering that thresholds in the model are specific to individuals. Thus, this analysis allows to highlight the statistical associations of the health-income gradient. Moreover, in the second part of this chapter, we investigate pathways through which income inequalities induce health inequalities. Results suggest that, in order to decrease income-related health inequalities, governments should promote investments in human and social capital; and, reduce the psychosocial effects of comparisons between individuals.

Once these associations are emphasized, researchers need to focus on causal links in order to implement adequate public policies such as redistribution. Thus, in the second chapter, entitled “**Health and income: testing for causality on European elderly people**” and co-written with Eric Delattre, we investigate simultaneous causal effects from income to health and from health to income, while highlighting the Granger causality. We exploit the dynamic dimension of the SHARE survey to estimate a simultaneous equations model. Using a FIML estimator, we find evidence of persistent causal effects running from income to health, and from health to income. Thus, among Europeans aged 50 years old and over, there might exist individual unobserved factors common to both health and income. This chapter contributes to the health-income relationship and allows a better understanding of the direction of the causality in this literature.

Finally, the last analysis of this thesis, entitled “**Smoking habits, time and risk preferences: a comparative study over European elderly people**” and jointly done with Richard K. Moussa, focuses on a public health issue which is the tobacco consumption. This research aims at understanding the main determinants of tobacco consumption. Smoking decision is considered as an economic choice which depends on individual heterogeneity and a budget constraint, characterized by income. On one hand, benefits from smoking result from present satisfaction due to the addictive nature of this good. On the other hand, costs result from the price of cigarettes, and from the depreciation of the health capital resulting from smoking. Concerning heterogeneity, time preferences, and more specifically, future-oriented individuals correspond to people who value their health in the future. Concerning risk preferences, risk averse individuals do not want to

take the risk to alter their health by smoking. However, risk preferences and smoking might simultaneously determine each other because smokers might be more prone to take risks and thus be considered as risk lover individuals. In the same way, smoking and income might simultaneously determine each other because smoking might reduce the ability to work due to its harmful effects such that the latter reduce individual income. To overcome, simultaneity between both smoking and income on one hand, and smoking and risk preferences on the other hand, a simultaneous equations model using a FIML estimator is implemented. Results suggest that, for elderly Europeans, income has a negative and significant impact on smoking (however, smokers are, on average, richer than non-smokers). Similarly, concerning time preferences, individuals who are future-oriented consider negative impacts of smoking consumption on health. Finally, impacts of risk preferences depend on where individuals live, such that they might be either more likely to smoke, or, less likely to smoke.

Chapter 1

In-depth overview on the relationship between health and income: microeconomic evidence and pathways

Abstract

The health-income gradient states that income and health are positively related. This relationship is well established among the literature, however, researches also suggest that individual health may be affected by income inequalities within a society. On one hand, this chapter examines the association between income, income inequalities and health inequalities in Europe. On the other hand, the contribution of this chapter is to examine pathways through which income inequalities enhance health inequalities. Using the Survey of Health, Ageing and Retirement in Europe (SHARE), results suggest that (i) income has a positive and concave effect on health (Absolute Income Hypothesis); (ii) income inequalities in a country affect all members in a society (strong version of the Income Inequality Hypothesis). Concerning the latter hypothesis, which is the most complete relationship, our results further imply that (iii) governments should promote investments in human capital; (iv) resources an individual can put together (social capital) are important to narrow inequalities, and (v) individuals are very sensitive in following the common cultural model of the standard of living in a community (psychosocial effects of comparisons might deteriorate health when there are negatively detected). However, our study suggests that, when considering the position of the individual in the income distribution, as well as the interaction between income inequalities and these rankings, one cannot identify individuals the most affected by income inequalities (which should be the least well-off in a society according to the weak version of the Income Inequality Hypothesis).

Keywords: Health; income; inequalities; pathways; capital; Europe.

JEL Classification: D31; I00; I14; I31; O15.

1.1 Introduction

The last few years have seen unprecedented attention to an attempt by policy makers, policy advisers and international institutions to reduce health inequalities. To do so, they usually focus on the access to healthcare, given that such policies allow to improve the health of lower income groups (WHO [2009]; Potvin et al. [2010]). Improving equality of access to healthcare is however not the sole public policy which can favor health equality. In particular, it has been widely said that income and income inequalities are associated to health status; thus, any public policy which influences income and/or income inequalities might influence health. In this way, studying the relationship between income, income inequalities and health is interesting per se. With these elements in mind, this chapter confronts on an empirical basis three hypotheses. The first one, called the Absolute Income Hypothesis, was initially introduced by Preston [1975] and states that there is a positive and concave relationship between income and health.¹ Higher incomes can provide means for purchasing a better health status. The second one is the strong version of the Income Inequality Hypothesis and it asserts that the health status is determined by income inequalities within a society. Thus, the health of all individuals is affected by an increase or a decrease in income inequalities. The last one, a weak version of the Income Inequality Hypothesis, says that income inequalities are a threat to individuals placed at the lower end of the income distribution. This last hypothesis implies that income inequalities do not impact low income people and high income people in the same magnitude.

Various authors have studied the Absolute Income Hypothesis mainly in the United States, using different health measures, like self-perceived measures (Mackenbach et al. [2005]), life expectancy (Cutler et al. [2006]) and other health outcomes (Ettner [1996], Carrieri and Jones [2016]). Kennedy et al. [1998], Fiscella and Franks [2000], Wagstaff et al. [2003], and, Van Doorslaer et al. [2004] focus on the strong version of the Income Inequality Hypothesis and show that income inequalities in a society also matter in order to explain the average health status measured by self-perceived measures (mostly in the United States). Concerning the weak version of the Income Inequality Hypothesis, there are few empirical studies which investigate it, with the exception of Mellor and Milyo [2002] in the United States, Li and Zhu [2006] in China or Hildebrand and Van Kerm [2009] in Europe. Importantly, the strong version of Income Inequality Hypothesis and the weak version of Income Inequality Hypothesis are non-nested given that the weak version considers the rank of individuals and an interaction term between the rank and the

1. In this way, redistributing income from rich people to poor people will have an important and positive impact on the health of the poorer people, whereas the richer ones will experience a small decrease in their health.

income inequalities index whereas the strong version does not. Thus, both versions can be valid when income inequalities in a society are negatively associated to the health of all individuals, and more particularly the health of people ranked at the lower end of the income distribution. However, the authors previously mentioned focus mainly on one of the versions in the best case (mainly on data from the United States), without comparing them. This chapter aims at filling these gaps by looking at the three hypotheses, using the same European data, in order to give more insights about efficient public policies which should be implemented in Europe. Finally, studying these three hypotheses at the same time allows to highlight different mechanisms between health and income, especially pathways through which income inequalities imply health inequalities. As a result, once income-related health inequalities are demonstrated, we try to answer to the following question: how income inequalities affect individual health? Different pathways should be considered. Indeed, health might be impacted by income through a decrease in social spending by governments ([Lynch and Kaplan \[1997\]](#)) or by underinvestment in human or social capital. Moreover, income inequalities might affect negatively health through comparisons arisen between individuals. In this way, such mechanisms have been detailed by [Kawachi and Kennedy \[1999\]](#). They bring to the forefront three plausible mechanisms. The first channel through which income inequalities result in health inequalities is through disinvestment in human capital or in social goods. Differences in income may translate into differences in investments and social spending due to disparities in interest for individuals, such that health might differ across individuals. The second pathway is through the erosion of social capital, defined as the resources available to individuals or solidarity among them. The latter confers a sense of belonging to a community such that when solidarity differs among income groups, then health is altered. Finally, income inequalities result in unhealthiness through stressful social comparisons. Indeed, individuals compare themselves with others such that not achieving the average model of lifestyle, due to incomes for instance, implies a decrease in health status. These channels seem to be a key point in the analysis of the health-income gradient, however there are only few papers which investigate them. The majority of the studies only focus on either one or two channels and on older data ([Kaplan et al. \[1996\]](#); [Kawachi et al. \[1997\]](#); [Lynch and Kaplan \[1997\]](#); and more recently [Murayama et al. \[2012\]](#)). As a result, we put these questions back to the agenda and investigate these three pathways to have a complete understanding of income-related health inequalities.

In this chapter, we test the three above hypotheses with the Survey of Health, Ageing, and Retirement in Europe (SHARE), using mainly the fifth wave of this survey (2015 release), as well as the pooled version of the survey in robustness. We then investigate channels of income-related health inequalities, using the first three waves of this survey.

We use self-perceived health status as our health outcome. This subjective measure is sometimes criticized but similar to the ones used by [Mackenbach et al. \[2005\]](#), [Fiscella and Franks \[2000\]](#) and [Hildebrand and Van Kerm \[2009\]](#). Furthermore, some authors show that the latter is not biased ([Benitez-Silva et al. \[2004\]](#)). Lastly, even if this measure can be criticized because of interpersonal comparison issues, [Lindeboom and Van Doorslaer \[2004\]](#) prove that some econometric models tackle these problems.

The chapter is divided into two parts. The first one (section [1.2](#)) is about testing the hypotheses, while the second part (section [1.3](#)) concerns the mechanisms. In this way, the first part is organized as follows. Section [1.2.1](#) presents formally the three hypotheses that we test empirically. Section [1.2.2](#) describes the SHARE dataset, as well as the baseline econometric specification. In section [1.2.3](#) we present the results and some robustness checks. Then, the second part is organized as follows. In section [1.3.1](#), we give an insight of the pathways and the associated literature. Section [1.3.2](#) is dedicated to the data and section [1.3.3](#) describes the econometric specification and the analyses of the results. Finally, the last section (section [1.4](#)) concludes the chapter.

1.2 Microeconometric evidence on the health-income relationship

1.2.1 Literature review

Inequalities in health refer to the close relationship between health and membership in a group characterized by incomes, where income is an individual social determinant. This section formally presents the three hypotheses mentioned in the introduction, as well as some related literature. We should mention that, in this literature review, we transcribe terminology employed by authors which reflects causal relationships even if cross-sectional databases are used or some endogeneity might be at play.

The Absolute Income Hypothesis

From an early stage in the debate, the Absolute Income Hypothesis states that the relationship between health and income is positive and concave ([Preston \[1975\]](#)), meaning that people with higher incomes have better health outcomes, but income inequalities have no direct effect on health. As a result, the concavity of the relationship between individual income and health status is a necessary condition to assess the efficiency of redistributive policies, in which transferring a given amount of money from rich people to poor people will result in an improvement of the average health.

The individual-level relation between income and health is specified as follows:

$$h_i = \beta_0 + x_i\beta_1 + x_i^2\beta_2 + Z_i\gamma + \epsilon_i \quad (1.1)$$

where h_i represents the health status of individual i (objective or subjective measures); x_i is the income of individual i ; Z_i is a set of individual specific control variables²; and ϵ_i is the error term coming from differences in individual health. The concavity effect is legitimized if β_1 is positive, β_2 is negative, and $\frac{\partial h_i}{\partial x_i} > 0$.

A strong link between health and income has been demonstrated in a large number of empirical studies, and a concave relationship between the two is found. [Preston \[1975\]](#) explains that the impact of additional income on mortality is greater among the poor than richer people. [Ettner \[1996\]](#), using three US surveys, finds that increases in income improve mental and physical health but also increase alcohol consumption. Then, [Mackenbach et al. \[2005\]](#) show that a higher income is associated with better self-assessed health in Europe. Using mortality rates, [Cutler et al. \[2006\]](#) conclude the same thing in the United States. [Theodossiou and Zangelidis \[2009\]](#), using data on individuals aged between 50 and 65 from six European countries, find a positive but small effect of income on health. More recently, [Carrieri and Jones \[2016\]](#) analyze the effect of income on blood-based biomarkers and find a positive and concave effect of income on health.

The strong version of Income Inequality Hypothesis

Some researchers affirm that income inequalities in a society are equally important in determining individual health status. The key difference between the Absolute Income Hypothesis and the strong version of Income Inequality Hypothesis stems from the fact that the latter explicitly considers the effect of income inequalities on health while the former only takes into account the concavity assumption between health and income. [Mellor and Milyo \[2002\]](#) specifically define two versions of this hypothesis: the strong version and the weak version. The strong version of the Income Inequality Hypothesis implies that, whatever the level of income, the health of all individuals in a society is equivalently affected by income inequalities in this society. In this way, both the well-off and poor people are impacted by income inequalities. These may be a public bad for all members in a society since income inequalities are a threat to the health of all individuals. We can thus identify an individual effect (a micro part) which is assimilated to the Absolute Income Hypothesis and an aggregate effect (a macro part) which corresponds to the relationship between individual health and income inequalities in a society. Theoretically, the strong

2. Such as age, gender, number of years of education, marital status and the job situation. It can also contain countries dummies variables.

version of the Income Inequality Hypothesis is specified as follows:

$$h_{ij} = \beta_0 + x_i\beta_1 + x_i^2\beta_2 + \delta II_j + Z_i\gamma + \epsilon_{ij} \quad (1.2)$$

which is an expansion of equation (1.1) with the introduction of II_j as a measure of income inequalities in a society j (corresponding to the macro part explained above); where h_{ij} represents the health status of individual i in a society j .

This hypothesis has been empirically tested mainly on data from developed countries (principally in the United States). Tests have been conducted at both the individual level and the aggregate level. At the aggregate level, a number of studies try to demonstrate an association between income inequalities and public health and the results are contrasted (Kaplan et al. [1996]; Lynch et al. [1998]; Subramanian and Kawachi [2004]). At the individual level, Kawachi et al. [1997], Kennedy et al. [1998], and Fiscella and Franks [2000] all find a negative association between income inequalities and self-perceived health. However, Van Doorslaer et al. [2004] find no effect of income inequalities on an objective health measure, the McMaster health utility index, derived from the self-perceived health status. Finally, other authors test the impact of income inequalities on malnutrition (Wagstaff et al. [2003]) or health service use (Lindelow [2006]) and find contrasted results.

The strong version focuses on the direct ties between health and income inequalities. There are several potential pathways through which income inequalities might be negatively related to an individual's health. Kawachi and Kennedy [1999] summarize three plausible mechanisms linking income inequalities to health.³ The first one is that disinvestment in human capital is linked to income inequalities. In states with high income inequalities, educational outcomes are negatively impacted when a smaller proportion of the state budget is spent on education which creates differences in education and thus in income. High income disparities may translate into lower social spending because interests of richer persons begin to diverge from other people in societies where inequalities rise. Thus, reducing social spending turns into a decrease in life opportunities for poorer people and thus an increase in inequalities (see also Grossman [2015]). The second mechanism is that income inequalities lead to the erosion of the “features of social organization that facilitate cooperation for mutual benefit”. In other words, Kawachi and Kennedy [1999] interpret this mechanism as the erosion of the social capital, corresponding to the set of collective resources an individual can put together. This may be the access to public services, the feeling of security, the characteristics of the relatives or the community solidarity (Grignon et al. [2004]). Here we focus on the solidarity argument. This one is important for the maintenance of population health. Kawachi and Kennedy [1999] made

3. See part 1.3 for further explanation about these mechanisms and a detailed empirical investigation.

a study using the General Social Survey where each indicator of social capital (like the degree of mistrust or levels of perceived reciprocity) was correlated with lower mortality rates. An increasing level of mistrust between the members of a society was due to the development of the distance between the well-off's expectation and the ones of poorer people. Unfortunately, this result implies a growth of a latent social conflict. As a result, when health is associated to the erosion of social capital, this seems to be towards the transition of social policies which are detrimental to poor people, implying unequal political participation. A lower turnout at elections is perceived among states with low levels of interpersonal trust. These states are less likely to invest in policies that ensure the security of poorer people in a society. Finally, less generous states are likely to provide less hospitable environments for these individuals. The last mechanism is that income inequalities are correlated to unhealthiness through stressful social comparisons. In this case, a technique in anthropology called "cultural consensus analysis" is used to take into account the psychosocial effects of social comparisons. Indeed, many communities have a common cultural model of the standard of living. This technique involves interviewing people and observing if individuals succeed in achieving the cultural model of lifestyle. This aspect can be seen as the satisfaction individuals have with their life. However, it should be noticed and not forgiven that a possible endogeneity issue can appear with this mechanism connected to the life satisfaction of individuals.

The weak version of Income Inequality Hypothesis

The second version of the Income Inequality Hypothesis is the weak one. According to this hypothesis, people who are more likely to have poorer health are the ones who feel more economically disadvantaged than their peers in a reference group. As a result, it specifically suggests that only the least well-off are hurt by income inequalities in a society. The damaging effect of these inequalities on health decreases with a person's income rank. Indeed, for an individual, stress and depression leading to illness may be linked to the fact of having a low relative income when compared to another person (Cohen et al. [1997]). The main concern is thus on the difficulties that an individual may face when he is situated at the bottom of the social ladder. Theoretically, the weak version of the Income Inequality Hypothesis is specified as follows:

$$h_{ij} = \beta_0 + x_i\beta_1 + x_i^2\beta_2 + \delta II_j + \theta R_{ij} + \eta R_{ij} * II_j + Z_i\gamma + \epsilon_{ij} \quad (1.3)$$

which is an expansion of equation (1.2) where we introduce R_{ij} as a person's rank, and the interaction between inequalities and a person's rank ($R_{ij} * II_j$) to allow the effects of income inequalities to vary by the relative income level in a society. The interaction term

allows to know how income inequalities are related to people with lower levels of income, compared to other people. Therefore, this hypothesis suggests that the breadth of the difference between rich people and poor ones accounts for the health. When testing this equation, δ underlines the strong version of the Income Inequality Hypothesis whereas θ and η specifically refer to the weak version. Thus, if the three previous coefficients are significant and have the right signs, then both the strong and the weak version are correct, meaning that everybody's health is associated to income inequalities, and in particular people who are at the lower end of the income distribution. On the other hand, whether only δ (or θ and η respectively) is significant implies that only the strong version (*resp.* the weak version) is satisfied.

As explained in the introduction, only few researches focus on this hypothesis. [Mellor and Milyo \[2002\]](#) use data from the Current Population Survey and find no consistent association between income inequalities and individual health. On the other hand, [Li and Zhu \[2006\]](#), using data from China, find that income inequalities are detrimental for people who are at the lower end of the income hierarchy. Finally, [Hildebrand and Van Kerm \[2009\]](#) also test the hypothesis that income inequalities may affect only the least well-off in a society using the European Community Household Panel but find no evidence supporting it.

1.2.2 Survey of Health, Ageing and Retirement in Europe (SHARE) and method

The Survey of Health, Ageing and Retirement in Europe (SHARE) is a multidisciplinary and cross-national panel database of micro data on health, socio-economic status and social and family networks of more than 123,000 individuals aged 50 and over from many European countries and Israel ([Börsch-Supan et al. \[2013\]](#)). Since 2004, SHARE asks questions throughout Europe to a sample of households with at least one member who is 50 and older. These households are re-interviewed every two years in the panel. The SHARE survey was constructed in the different European countries under the leadership of Professor Axel Börsch-Supan. In addition, SHARE is harmonized with the Health and Retirement Study (in the United States - HRS) and the English Longitudinal Study of Ageing (UK - ELSA).

The first wave (2004-2005, 27,014 individuals) and the second one (2006-2007, 34,393 individuals) were used to collect data on health status, medical consumption, socio-economic status and living conditions. The 2008-2009 survey (Wave 3 - "SHARELIFE") was extended to life stories by collecting information on the history of the respondents. The number of participants increased from 12 countries in wave 1, to 15 (+ Ireland, Israel,

Poland and Czech Republic) in wave 2, and the third wave contains information about 14 countries. The fourth wave (2010-2011), is a return to the initial questionnaire of the first two waves. It collects data from 56,675 individuals in 16 European countries. Then, the fieldwork of the fifth wave of this survey was completed in 2013. The following countries are included in the scientific release of 2015: Austria, Belgium, Switzerland, Czech Republic, Germany, Denmark, Estonia, Spain, France, Israel, Italy, Luxembourg, Netherlands, Sweden, and Slovenia. This wave contains the responses of 63,626 individuals. We focus on the fifth wave ([Börsch-Supan \[2017e\]](#)) in order to have a great number of individuals who come from different countries.⁴ Moreover, in order to test and compare the three hypotheses linking health and income, one has to use the same set of observations (e.g. the fifth wave of the SHARE survey). We do not make our analysis using directly a pooled database since all the control explanatory variables are not available in every wave, which is a limitation of this database. However, we then focus on the pooled database, composed of waves one, two, four ([Börsch-Supan \[2017a,b,d\]](#)) and five, in order to make our results more robust (the third wave is not considered in the pooled database since it does not contain the same information as the other ones).

The advantage of the SHARE database is that it has many individual variables on health, socioeconomic status and income to perform this research. However, researchers should be also aware of the potential disadvantage of this database. Indeed, [Börsch-Supan et al. \[2013\]](#) explain that in some waves there are a relative low response rates and moderate levels of attrition (even though the overall response rate is high compared to other European and US surveys⁵) which are due to the economic crisis faced by some countries, implying a decrease in the participation rates. Due to this attrition, our main analyses focus on the fifth wave of this survey instead of the pooled database directly. Nonetheless, we present the results using the pooled database as a robustness test.

In this study, we want to underline the effects of income inequalities on health such that we need a measurement of income inequalities. The Gini coefficient, as well as the Theil index, are two well-known indexes which can be used.

Algebraically, the Gini coefficient is defined as half of the arithmetic average of the absolute differences between all pairs of incomes in a population, and then the total is normalized on mean income. If incomes in a population are distributed completely equally, the Gini value is zero, and if one person has all the incomes in a society, the Gini is one. The Gini

4. Please note that at the time of this part of the chapter, wave six of this survey was not released yet. This last wave is used in two last chapters, and then detailed in the latter.

5. After wave four was completed, the average retention rate over the year was 81 %.

coefficient can be illustrated through the Lorenz curve. However, the Gini coefficient does not take into account the income distribution since different Lorenz curves may correspond to the same Gini index.⁶ In other words, it does not distinguish between inequalities in low income group and high income ones. Formally, the Gini coefficient is:

$$Gini = \frac{2 \sum_i i y_i}{N \sum y_i} - \left(\frac{N+1}{N} \right) \quad (1.4)$$

with y_i representing the income of the population sorted and ranked, from the lowest decile group to the top decile group, and N representing the total population.

As a result, one of the solution is to use the Theil index which measures income inequalities. The Theil index is:

$$Theil = \frac{1}{N} \sum_i \frac{y_i}{\bar{y}} \ln\left(\frac{y_i}{\bar{y}}\right) \quad (1.5)$$

where \bar{y} is the mean income per person (or expenditure per capita). In order to normalize the Theil index to vary between zero and one, we divide it by $\ln(N)$.⁷ It measures a “distance” of the real population and the “ideal” egalitarian state where everyone have the same income.

Since the Gini coefficient does not take into account the income distribution, most of the following tables of results are displayed using the Theil index.⁸

In this part, the data used are from the fifth wave of the SHARE survey. This wave includes responses from 63,626 respondents aged 50 and over, living in 15 different countries. In one hand, the variable of interest is the health which is defined in the database as the self-perceived health status. Individuals are asked to classify their health using ordered qualitative labels from “poor” to “excellent”. Figure 1.1 characterizes the distribution of the health variable among individuals aged 50 and older by gender for all countries in wave five. As we can see the majority of inhabitants reports being in a good health. In the other hand, one of our main determinant of health is the income. This variable can be seen as a proxy for well-being, that is to say a factor which allows individuals to improve their living standards. In the database, it corresponds to the sum of individ-

6. For instance, if 50 percent of the population has no income and the other half has the same income, the Gini index is 0.5. The same result can be found with the following analysis which is less unequal. On one hand, 25 percent of total income is shared in the same way by 75 percent of the population, and on the other hand, the remaining 25 percent of the total income is divided by the remaining 25 percent of the population.

7. It is this normalized index that we use hereafter and that we name the Theil index.

8. However, all the analyses have been done using the Gini coefficient as well. Results are very similar.

ual imputed income for all household components. Figure 1.2 shows the distribution of income of people aged 50 and over in the fifth wave where the mean is about 36,000€. Moreover, the Income Inequality Hypothesis includes an indicator for the measurement of income inequalities (see figure 1.3). In this chapter, we use either the Gini index or the Theil index. The mean of the Gini index in Europe is 0.39 which corresponds to a rather egalitarian society. The mean of the Theil index in Europe is 0.33 which is also rather egalitarian. In this analysis, we include others variables such as age, marital status, education, job situation, dummies for countries and gender, and GDP of the countries (see tables 1.3, 1.4, 1.5 and 1.6 in the appendix for further information). Finally, the pooled data (waves 1, 2, 4 and 5) contains 181,708 observations, where each individual is present on average 2.9 years in the panel.

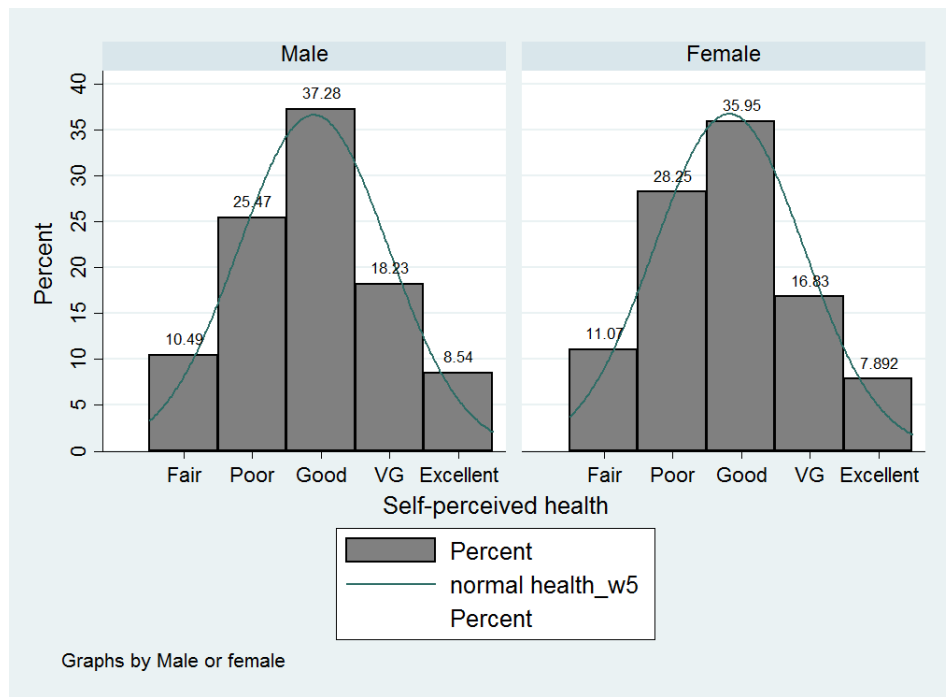


Figure 1.1 – Self-perceived health in Europe - Part 1

Source: SHARE - Wave 5

To model the association between self-perceived health and other socioeconomic status and test the hypotheses, we use an ordered probit specification. When the self-perceived health status outcome is denoted as h_i , the model can be stated as:

$$h_i = j \quad \text{iff} \quad \mu_{j-1} < h_i^* \leq \mu_j, \quad (1.6)$$

for $j = 1, 2, 3, 4, 5$

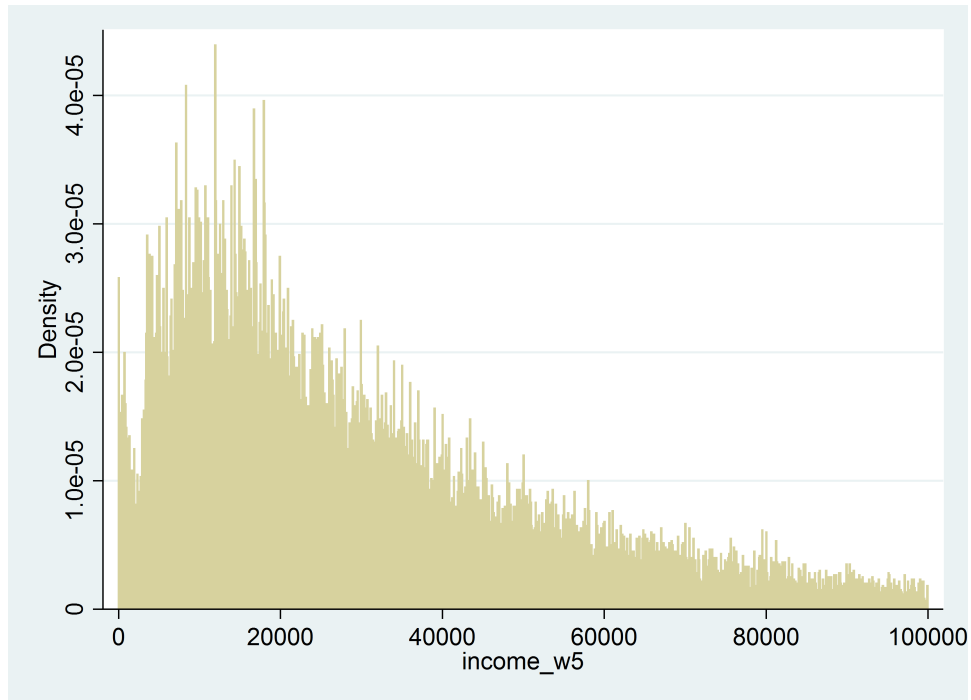


Figure 1.2 – Distribution of income in Europe - Part 1

Source: SHARE - Wave 5

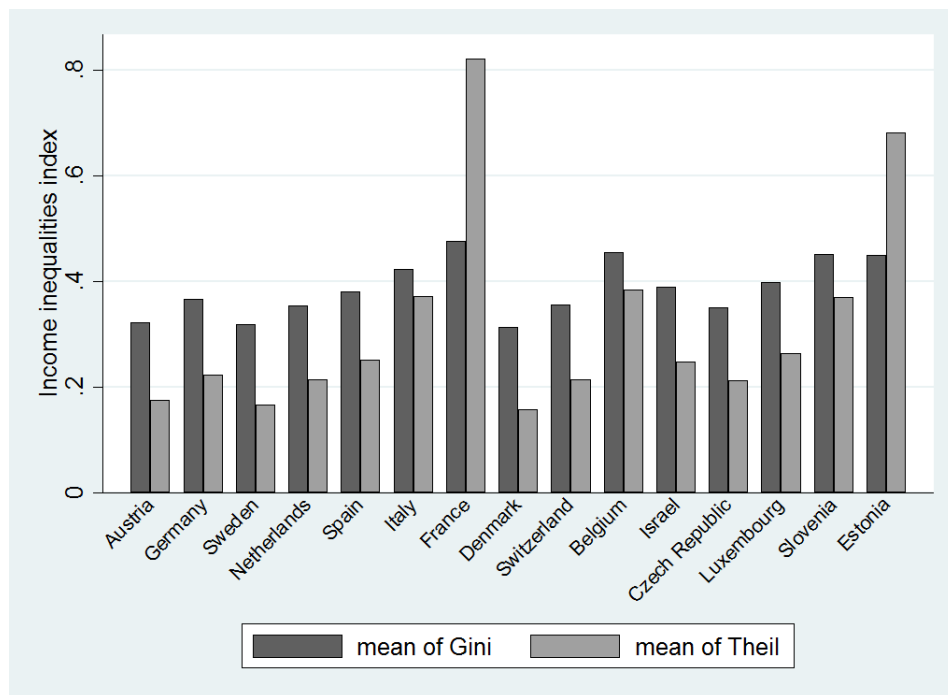


Figure 1.3 – Income inequalities indexes in Europe - Part 1

Source: SHARE - Wave 5

The latent variable specification of the model that we estimate can be written as:

$$h_i^* = x_i\beta + \epsilon_i \quad (1.7)$$

where h_i^* is a latent variable which underlies the self-reported health status⁹; x_i is a set of observed socioeconomic variables; and ϵ_i is an individual-specific error term, assumed to be normally distributed.

In this data, the latent outcome h_i^* is not observed. Instead, we observe an indicator of the category in which the latent indicator falls. As a result, the observed variable is equal to 1, 2, 3, 4 or 5 for “poor”, “fair”, “good”, “very good” or “excellent” with this probability:

$$P(y = j|x) = F(\mu_j - x_i\beta) - F(\mu_{j-1} - x_i\beta) \quad (1.8)$$

The interval decision rule is:

1. $h_i = 1$ if $h_i^* \leq \mu_1$;
2. $h_i = 2$ if $\mu_1 < h_i^* \leq \mu_2$;
3. $h_i = 3$ if $\mu_2 < h_i^* \leq \mu_3$;
4. $h_i = 4$ if $\mu_3 < h_i^* \leq \mu_4$;
5. $h_i = 5$ if $h_i^* > \mu_4$.

In this model, the threshold values $(\mu_1, \mu_2, \mu_3, \mu_4)$ are unknown. The value of the index necessary to shift from very good to excellent, for instance, is unknown. In theory, the threshold values are different for everyone.

1.2.3 Results

Economic results and discussion

Table 1.1 reports coefficient estimates for all hypotheses, when income inequalities are measured using the Theil index.¹⁰ The fifth wave of the survey gives access to 63,626 observations and we also display results of the pooled database for sake of robustness (see table 1.8 in the appendix section). Results in the first column report estimated coefficients for the Absolute Income Hypothesis (AIH) while results in columns two and three provide tests of both the strong version and the weak version of the Income Inequality Hypothesis (IIH).

9. Once h_i^* crosses a certain value you report poor, then fair, then good, then very good, then excellent health.

10. Results associated to the Gini coefficient are not provided here but they are very similar and available upon request.

Coefficients of individual income and income squared provide support for all the hypotheses that there is a positive and concave relationship between income and self-perceived health status. Indeed, coefficients associated to the income variable are all positive and significant and coefficients associated to the income squared variable are all negative and significant. This implies that higher income is related to a better health outcome. As a result, the Absolute Income Hypothesis is verified. Concerning income inequalities, coefficients on the Theil index in columns two and three are negative and significantly different from zero. This supports evidence of the strong version of Income Inequality Hypothesis stating that an increase in income inequalities is detrimental to all members of a society, i.e., income inequalities and health are negatively related. Indeed, concerning this index, zero represents an egalitarian state, thus the negative relationship between self-perceived health and the indicator of income inequalities is in line with health being better if the index is low. However, results in column three do not support the weak version of Income Inequality Hypothesis which states that inequalities are more detrimental to the least well-off in a society. Indeed, we introduce individual rank (by country) and an interaction term between the rank and the index of income inequalities to allow a variation between income level and the effect of income inequalities. In the specification, we choose to follow the framework of [Mellor and Milyo \[2002\]](#) who introduced interaction terms between the measurement of income inequalities and dummies variables based on quintiles of income (1 for the lowest income group and 5 for the highest, which is a proxy for the rank). In other words, interaction terms indicate the effect of aggregate income inequalities (at the country level) on self-perceived health status between individuals situated at different levels of the income distribution. Concerning the first two interaction terms ($II_j * Q1$ and $II_j * Q2$), they indicate the effect of aggregate income inequalities (at the country level) on self-perceived health status between the poorest individuals (situated at the lower end of the income distribution) and the richest ones (reference category corresponding to individuals situated at the top of the income distribution). These coefficients are positive and statistically significant, meaning that for the poorest individuals (compared to more well-off individuals), an increase in income inequalities in their country increases self-perceived health status, which is in contradiction with the weak version of the Income Inequality Hypothesis. Concerning the two other interaction terms (third and fourth quintiles, representing people at the middle and almost top of the income distribution), coefficients are not statistically significant meaning that middle and higher income people are not affected at all by an increase in income inequalities. This claim does not support the weak version because this hypothesis states that people at the lower end are the most affected by an increase in income inequalities compared to people at the top of the income distribution. As a result, higher income people should also be affected by income

inequalities (at a lower rate). Our qualitative results suggest that for low-income individuals, an increase in income inequalities in their country is positively related to report a better health status. Furthermore, for higher income individuals, an increase in income inequalities in their country is not related to report neither a better nor a lower health status. To conclude, our results do not support the weak version of Income Inequality Hypothesis, but it further invalidates this weak version because our qualitative results quite claim the opposite.

Regarding the mechanisms of [Kawachi and Kennedy \[1999\]](#), we choose to give a glimpse of empirical evidence, even if the latter is further examined in part 1.3 (table 1.1, column two). In this way, we characterize disinvestment in human capital (first mechanism) by the percentage of health expenditure in the GDP.¹¹ The coefficient associated is positively correlated to health meaning that when governments increase health spending, this has a positive effect on individual health. For the second mechanism, we want to illustrate the interaction between individuals to represent the erosion of social capital. As a result, we choose a variable from the SHARE survey: “received help from others”. The coefficient associated to this variable is negative and significant. We can explain this negative association by saying that people who are in bad health are the ones who receive help. In order to legitimize this explanation, we also explore the impact of the “reverse variable”: “given help to others”. In this case, the coefficient is positive and significant proving that people in good health offer their help. Then, the last mechanism is about social comparisons. The coefficient associated to this variable (“life satisfaction”) is positively linked to health which implies that when individuals are satisfied with their life, they also report having a good health.

In sum, our baseline specifications provide evidence of a statistically significant association between income, income inequalities and health since results are robust to model specifications.

11. Source: OECD website.

Table 1.1 – Results of the ordered probit model for wave 5 - Part 1

Variables	Absolute Income	IIH	
	Hypothesis	Strong Version	Weak Version
Income	1.84e-06*** (1.22e-07)	1.84e-06*** (1.20e-07)	1.89e-06*** (1.44e-07)
Income squared	-2.06e-13*** (1.55e-14)	-2.04e-13*** (1.50e-14)	-2.09e-13*** (1.73e-14)
<u>Quintiles of income</u>			
Quintile 1			-0.258*** (0.029)
Quintile 2			-0.201*** (0.028)
Quintile 3			-0.115*** (0.027)
Quintile 4			-0.053*** (0.026)
Quintile 5			Reference
Index of inequalities (II) - Theil		-0.403*** (0.024)	-0.838*** (0.049)
Interaction quintile 1 and II			0.115* (0.069)
Interaction quintile 2 and II			0.114* (0.068)
Interaction quintile 3 and II			0.023 (0.068)
Interaction quintile 4 and II			0.062 (0.068)
Interaction quintile 5 and II			Reference
<u>Co-variables</u>			
GDP		1.99e-06*** (4.53e-07)	0.0001*** (0.049)
Age	0.037*** (0.006)	0.019*** (0.006)	0.037*** (0.006)
Age squared	-0.0004*** (0.00004)	-0.0003*** (0.0004)	-0.0004*** (0.00004)
Years of education	0.034*** (0.001)	0.028*** (0.001)	0.026*** (0.001)
Gender = 1 if women	0.003 (0.009)	0.005 (0.009)	0.007 (0.009)
Married, living with spouse		Reference	
Registered partnership	-0.042 (0.035)	-0.006 (0.035)	0.058* (0.035)
Married, not living with spouse	-0.094** (0.039)	0.004 (0.039)	-0.076** (0.039)
Never married	-0.071*** (0.019)	0.023 (0.019)	0.023 (0.019)
Divorced	-0.045*** (0.015)	0.068*** (0.016)	0.032** (0.018)
Widowed	-0.024* (0.014)	0.055*** (0.015)	0.015 (0.014)
Retired		Reference	
Employed	0.253*** (0.014)	0.224*** (0.014)	0.246*** (0.014)
Unemployed	-0.212*** (0.028)	-0.103*** (0.028)	-0.176*** (0.028)
Permanently sick	-1.25*** (0.026)	-1.069*** (0.026)	-1.207*** (0.026)
Home-maker	-0.059*** (0.017)	-0.064*** (0.017)	-0.056*** (0.017)

Table 1.1 – Results of the ordered probit model for wave 5 - Part 1 (continued)

Variables	Absolute Income	IIH	
	Hypothesis	Strong Version	Weak Version
Other	−0.236*** (0.031)	−1.169*** (0.031)	−0.207*** (0.031)
<u>Mechanisms strong IIH</u>			
1 st : % Health expenditure in GDP		0.077*** (0.003)	
2 nd : Received help from others		−0.179*** (0.006)	
2 nd bis: Given help from others		0.001*** (0.0001)	
3 rd : Life satisfaction		0.216*** (0.003)	
<u>Cut-points</u>			
Cut-point μ_1	−0.474 (0.216)	0.899 (0.219)	−0.428 (0.215)
Cut-point μ_2	0.615 (0.216)	2.076 (0.219)	0.632 (0.215)
Cut-point μ_3	1.746 (0.216)	3.261 (0.219)	1.728 (0.215)
Cut-point μ_4	2.592 (0.216)	4.133 (0.219)	2.548 (0.215)
<u>Marginal effects at mean of absolute income, on</u>			
Pr(Poor health)	−2.84e-07*** (1.92e-08)	−2.58e-07*** (1.71e-08)	−3.02e-07*** (2.32e-08)
Pr(Fair health)	−3.06e-07*** (2.05e-08)	−2.97e-07*** (1.95e-08)	−3.24e-07*** (2.49e-08)
Pr(Good health)	8.80e-08*** (6.44e-09)	6.65e-08*** (4.97e-09)	9.56e-08*** (7.80e-09)
Pr(Very good health)	2.65e-07*** (1.78e-08)	2.55e-07*** (1.68e-08)	2.79e-07*** (2.14e-08)
Pr(Excellent health)	2.37e-07*** (1.59e-08)	2.34e-07*** (1.54e-08)	2.51e-07*** (1.92e-08)
Numb. of obs.		63,626	
For AIH, dummies for countries are included but not reported, and available upon request.			
***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are in parentheses, below the coefficients.			

Robustness checks

As a sake of robustness, we also make our entire analysis using the pooled database (see table 1.8 in the appendix section) and the results are very similar to the ones obtained with the fifth wave of the survey.

To give more support to the concavity assumption, we compute, for all three hypotheses, the marginal effects at mean¹² of income on the five health outcomes. Results, reported at the end of table 1.1, are all significant. On one hand, for the first two outcomes, income has a negative effect on the probability to report either a poor health or a fair health status. On the other hand, there is a positive effect of income on the probability to report being in a good, very good and excellent health (outcomes three to five). These results are obtained following the ordered probit regressions of the three hypotheses, where the quadratic effect of income is investigated (see equations 1.1, 1.2 and 1.3). These results do not validate the concavity assumption but they do show the increasing effect of income on self-perceived health status. We also plot the average marginal effect of income on each outcome for all individuals with a confidence interval, in order to give more support to the concavity effect in the three hypotheses (see figure 1.4 for the Absolute Income Hypothesis).¹³ We restrict ourselves to individuals who earn less than 200,000 € per year (which corresponds to more than 99% of the distribution, see table 1.5 in the appendix section for further information on the distribution of income). Graph 1.4a gives the impact of income on the probability to report a poor health. This impact is negative (y-axis is negative), meaning that when income raises, the probability decreases. In addition, the negative impact is stronger for the majority of the population than for individuals who earn very high incomes. In other words, for low incomes, in absolute terms, an additional increase in income has a larger impact on the probability of reporting a poor health than for very high income. This is a low support for the concavity assumption. Graph 1.4b gives the impact of income on the probability of reporting a fair health status. Conclusion are similar to the ones of graph 1.4a since the effect is negative. The slight decreases of the curve at the beginning does not impact the conclusion and can be related to large confidence intervals. Graph 1.4c gives the impact of income on the probability to report a good health status. For almost all the distribution, when income raises, the probability increases. Then, graphs 1.4d and 1.4e are more conclusive. Indeed, graph 1.4d gives the impact of income on the probability to have a very good health. For more than 99% of the income distribution, this impact is positive and decreasing, which might support the concavity assumption. Finally, graph 1.4e gives the impact of income on the probability

12. Marginal effects are computed focusing on the average individual of the database.

13. We do not include GRAPHS for the Income Inequality Hypothesis (both versions) since the results are very similar and do not change the main conclusion, but these are available upon request.

of reporting an excellent health status. As previously, when income increases, the probability to have an excellent health increases. However, when we look at people with very high incomes¹⁴, this impact is greater than for the majority of individuals.

Lastly, it is important to investigate the robustness of our results by taking into account the subjective nature of the self-perceived health status. Indeed, our baseline specification depends on a dependent variable which is subjective. Self-reported measures give information on individual health since people summarize all the health information they have from their practitioners (general practitioners and specialists) and from what they feel (Benítez-Silva et al. [2004]). However, the use of this measure might raise the problem of interpersonal comparisons between people aged 50 and over (“Is the way I consider “good health” the same as you consider this health commodity?”). Empirical studies on the relationship between health, income and income inequalities commonly use ordered probit models where the thresholds are constant by assumption. One limit is that it restricts the marginal probability effects. In fact, the distributional effects are restricted by the specific structure. Then, another limit is that an additional individual heterogeneity between individual realizations is not allowed by the distributional assumption. Thus, Boes and Winkelmann [2006] and Jones and Schurer [2011] both give a solution to these issues. Indeed, the generalized ordered probit model is based on a latent threshold where the thresholds themselves are linear function of the explanatory variables. In other words, previous thresholds of equation 1.8 are now computed by selecting individual characteristics so that they depend on covariates:

$$\mu_{ij} = \tilde{\mu}_j + x_i' \gamma_j \quad (1.9)$$

where γ_j is a vector of response specific parameters. We have:

$$\mu_{ij} = \mu_j \quad \forall_i \in C_j \quad (1.10)$$

where C_j is the class. With this model, the probabilities are:

$$P(y = j|x) = F(\tilde{\mu}_j - x_i \beta_j) - F(\tilde{\mu}_{j-1} - x_i \beta_{j-1}) \quad (1.11)$$

Now, the effects of covariates on the log-odds are category-specific and this model allows to have more heterogeneity across individuals. Results concerning the generalized ordered probit model are similar to the ones obtained from the ordered probit model. All the

14. In this case, people with very high incomes are individuals who earn more than 150,000€ per year, corresponding to less than 2% of the sample.

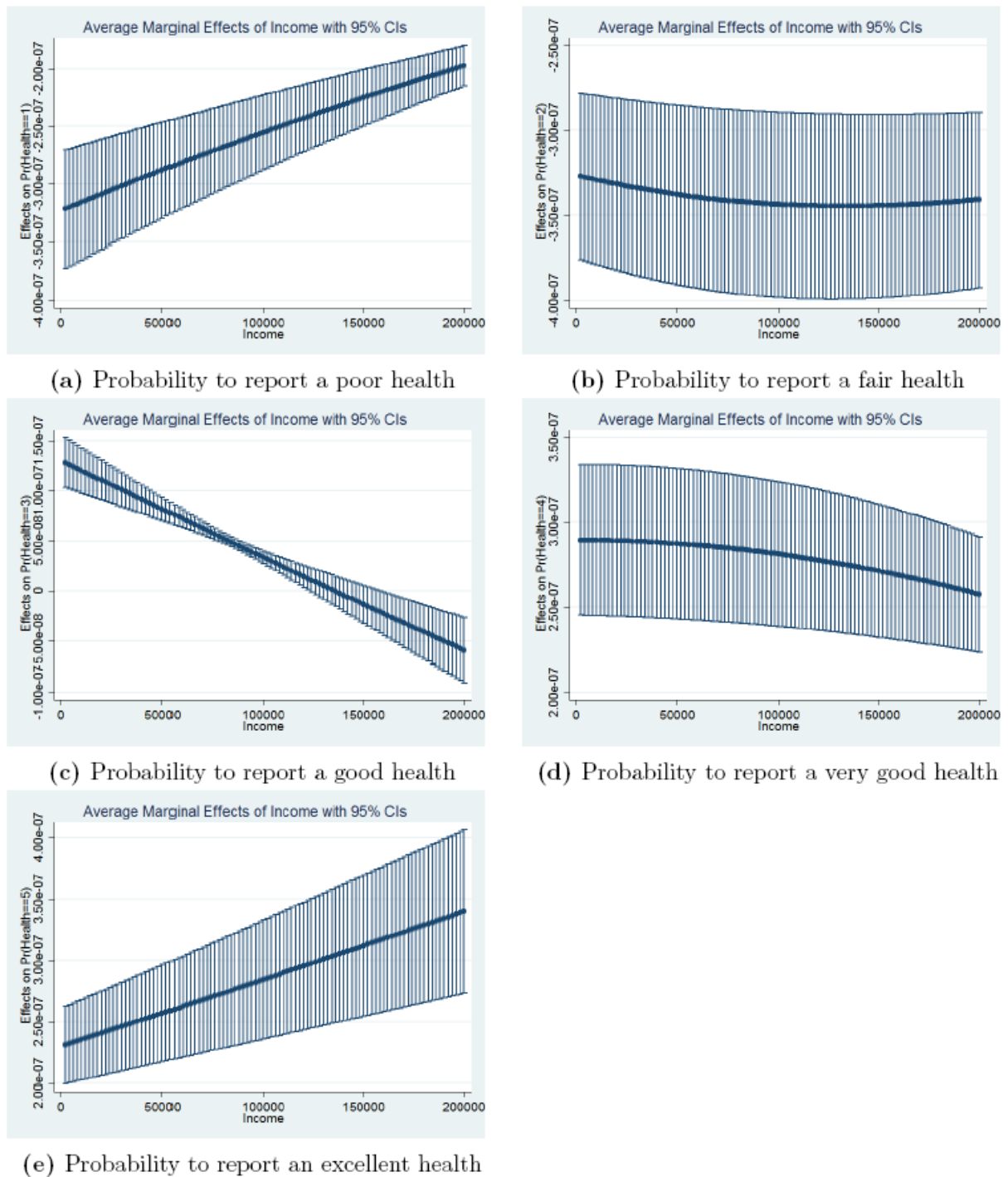


Figure 1.4 – Average marginal effects of income on health - Absolute Income Hypothesis

Source: SHARE - Wave 5

effects are estimated around each four cut-points (from poor to fair, fair to good, good to very good, and from very good to excellent). For all the hypotheses (Absolute Income Hypothesis - table 1.9; both versions of the Income Inequality Hypothesis - tables 1.10

and 1.11, in the appendix part), the coefficients associated to the variables of interest (income and income squared) do not significantly change when compared to the results of the ordered probit model. Results are consistent (either with the Theil index or the Gini coefficient for the Income Inequality Hypothesis) as this is proved in previous study (Lindeboom and Van Doorslaer [2004]). In fact, in the four cut-points, results are in accordance with the concavity assumption of income since coefficients are statistically significant. Moreover, the index of income inequalities is significantly negative which is in line with the strong version of the Income Inequality Hypothesis. Then, concerning the interaction terms, these are not significant for all quintile groups which do not justify the weak version of Income Inequality Hypothesis. Finally, adding some heterogeneity in this model and taking into account issues of interpersonal comparisons do not modify our previous results.

1.3 Pathways in income-related health inequalities

In the first part (section 1.2), we confront on an empirical basis three hypotheses linking income, income inequalities and health (Adeline and Delattre [2017]). The analyses (coefficient estimates, as well as average marginal effects) strongly support two hypotheses by showing that (i) income has a positive and concave effect on health (Absolute Income Hypothesis), and, (ii) income inequalities in a country affect all members in this country (strong version of the Income Inequality Hypothesis). The latter hypothesis is the strongest one because it highlights both the concave effect of income on health and the negative impacts of income inequalities in a country. Thus, not only individual income affects population health, but also societal pattern such as the distribution of income in a country (or in another aggregate level).

Overall, literature suggests a positive correlation between income and health (Cutler et al. [2011]) and a negative one between income inequalities and individual health. Now, the question is about mechanisms underlined by income-related health inequalities: how inequalities in the distribution of income affect individual health? Indeed, we need to understand channels of structural determinants of inequalities, pathways through which income distribution is linked to health status. In this part, we offer an analysis of these channels, specifically focusing on the strong version of Income Inequality Hypothesis. Understanding these mechanisms is an important pattern for policy makers or governments whose aim is to improve general health and curb health inequalities. Socioeconomic characteristics of individuals, comparison with their relatives, or other mechanisms at the state level might alter the level of income inequalities and thus, may also have an impact on health (either positive or negative). As a result, it is important to document such

health effects so that they may be considered within the context of economic decision. [Lynch and Kaplan \[1997\]](#) state that mechanisms through which inequalities impact health are twofold. First, there might be a pathway involving “social processes and policies that systematically underinvest in human, physical, health and social infrastructure”, then affecting health. Second, individual’s perception of the social environment, due to income inequalities, might have direct consequences on health. In the same way, [Kawachi and Kennedy \[1999\]](#) go further by differentiating three plausible mechanisms linking income inequalities to health: disinvestment in human capital, erosion of social capital and social comparisons. Even if these issues have been addressed, they should be put back on the agenda and tested on more recent data in order to implement adequate public policies. We focus on these specific pathways to have detailed insights on income-related health inequalities.

This deeper analysis of the strong version of the Income Inequality Hypothesis, is structured as follows. Section [1.3.1](#) describes three pathways by which income inequalities may involve health inequalities. Section [1.3.2](#) details the database used. Section [1.3.3](#) presents the econometric analysis and the results.

1.3.1 Literature review

For public policies purpose, understanding diversity of mechanisms is of interest. Indeed, understanding pathways by which income leads to variations in health outcomes help to understand how the latter could be improved. We focus on the three mechanisms highlighted by [Kawachi and Kennedy \[1999\]](#).

Disinvestment, the first mechanism

The first channel through which income inequalities result in health inequalities is through disinvestment in human capital (considered as a private good) or in social goods (corresponding to public goods). Empirically one should disentangle between aggregate investments done by governments or policy makers and individual ones done directly by an individual. However, in fact, both are related because in geographical areas with high income inequalities, differences in education impact income distribution when a small proportion of government budget is spent on education (see [Grossman \[2015\]](#)). Inequalities depend on the availability of public and private resources which are important in the reduction of negative health outcomes ([Lynch and Kaplan \[1997\]](#)).

At the aggregate level, [Kaplan et al. \[1996\]](#) show that income inequalities at the state level are correlated with indicators concerning investments in social goods. In societies

where inequalities rise, interests of high-income groups begin to diverge from other individuals, such that differences in income may, in turn, translate into lower social spending (public good). Thus, there is less investment of the state budget on education. This is also supported by [Krugman \[1996\]](#) who explains that this channel leads to disparities in interest for individuals when income gap widens: “a family at the 95th percentile pays a lot more in taxes than a family at the 50th, but it does not receive a correspondingly higher benefit from public services”. Individual perceptions of inequalities arise in an unequal environment where unequal distribution of resources, resulting from disinvestment in human, physical or social infrastructure (which can be used to investigate this mechanism), impact the level of physical and mental well-being of individuals. In other words, income inequalities are correlated with the level of government spending. [Mayer and Sarin \[2005\]](#) highlight a negative relationship by showing that some individuals who do not feel concerned by politics, might be less likely to vote. These individuals will not support redistributive policies such that social spending will be reduced. This will, in turn, increase inequalities. On the other hand, higher levels of income inequalities might motivate governments to increase investments through redistribution or greater demand for taxation. [Meltzer and Richard \[1981\]](#) explain that in a state with high income inequalities, the median voter might be in favor of redistributive policies¹⁵, implying taxation. Overall, this mechanism implies that the effect of income inequalities on health is attenuated when governments increase public spending.

Moreover, human capital might also be seen as a private good when it corresponds to skills, education, capacity and other attributes of an individual, which influence productivity and thus earnings potential (thus individual characteristics can be used as proxies to study this pathway). Indeed, according to the Organization for Economic Cooperation and Development (OECD), human capital is defined as: “the knowledge, skills, competencies and other attributes embodied in individuals or groups of individuals acquired during their life and used to produce goods, services or ideas in market circumstances”.¹⁶ This is why education attainment can correspond to human capital, and be related to income distribution. Thus, human capital investments can reduce income-related health inequalities.

15. [Galasso and Profeta \[2002\]](#) argue that individuals will support redistribution when they believe that inequalities result from structural disadvantages but not from a lack of individual effort.

16. Human capital reflects the value of human capacities. It could be invested in through education, training and enhanced benefits that lead to an improvement in the quality and level of production.

Social capital, the second mechanism

A second mechanism through which income inequalities impact health outcomes is via the erosion of the “features of social organization that facilitate cooperation for mutual benefit”. In other words, [Kawachi and Kennedy \[1999\]](#) interpret this mechanism as the erosion of the social capital. Social capital might refer to the set of collective resources an individual can put together, and also to the stock of investments, resources and networks that produce social cohesion, trust between citizens and a willingness to engage in community activities. According to [Coleman \[1990\]](#), social capital represents “a variety of different entities having two characteristics in common: they all consist of some aspect of social structure, and they facilitate certain actions of individuals who are within the structure”. This may be the access to public services, the feeling of security, the characteristics of the relatives or the community solidarity ([Grignon et al. \[2004\]](#)). Here we focus on the solidarity argument, i.e., which represents resources available to members of communities. [Kawachi et al. \[1997\]](#) report associations between mistrust, levels of perceived reciprocity or membership in voluntary associations (considered as a proxy for social capital) and mortality rates, giving evidence of an association between social capital and health indicators. They made a study using the General Social Survey where each indicator of social capital (like the degree of mistrust or levels of perceived reciprocity) was correlated with lower mortality rates. An increasing level of mistrust between the members of a society was due to the development of the distance between the well off’s expectation and the ones of poorer people. Unfortunately, this result implies a growth of a latent social conflict. As a result, health being affected by the erosion of social capital seems to be towards the transition of social policies which are detrimental to poor people, and might imply unequal political participation. A lower turnout at elections is perceived among states with low levels of interpersonal trust, such that they cannot ensure the security of poorer people in their society, meaning less hospitable environments for individuals. [Murayama et al. \[2012\]](#) review a number of published studies on social capital and health in which a general pattern applies: individual social capital appears to have positive effects on health outcomes. Social capital, in this work, may be considered as reciprocity between individuals which can make people willing to make resources available because of expectations of repayment. Thus, social participation provides opportunities to learn new skills and confers a sense of belonging to one’s community. In other words, solidarity influence health, directly by activating cognitive systems, and, indirectly by giving a sense of coherence and meaningfulness. As a result, social capital is important for the maintenance of population health.

Social comparisons, the last mechanism

A final channel is that income inequalities contribute to unhealthiness through stressful social comparisons. In anthropology, such analysis is called “cultural consensus analysis” and used to take into account psychosocial effects of social comparisons. Indeed, many communities have a common cultural model of the standard of living. This technique involves interviewing people and observing if they succeed in achieving the cultural model of lifestyle. Perception of stress, importance of social relation or self-esteem influence health according to [Wilkinson \[1992\]](#). [Lynch and Kaplan \[1997\]](#) explains that “it is this appraisal of relative well-being that may provide a psychosocially mediated link between income inequality and health status”. Differences in these effects lead to inequalities through health damaging psychosocial characteristics and behaviors. In health psychology literature, different theories are stated to explain how a climate of income inequalities might induce stress or detrimental health behaviors. [Bourdieu \[1984\]](#) demonstrates that tastes for art, music, food and reading material determine and influence how individuals embody and display their place in the social hierarchy. People integrate information about their environment which, in turn, shapes their perceptions of the relative position in the socioeconomic ladder. Socioeconomic gradient in health and income-related health inequalities are said to be two of the most prominent phenomena on the study of social determinants of health. Individuals are assumed to compete for their position in social hierarchies, and being unsuccessful in this competition might lead to stress which involves negative health outcomes.¹⁷ These correspond to the emergence of status seeking ([Marmot \[2004\]](#)) and to links between hierarchies and stress and illness. This competition depends on the perception of subjective socioeconomic characteristics. This subjectivity inherently involves social comparisons such that, in order to know and improve one’s own ranking in the social ladder, an individual needs to evaluate his peers. [Cohen et al. \[2008\]](#) explain that it is the subjective assessment of socioeconomic characteristics which predicts whether participants developed a common cold when exposed to a common cold virus, and not the objective indicators. Individuals integrate behaviors of others as benchmarks for evaluating their own behaviors, and usually shift their own to match the ones of the group. However, individuals might differ in their propensity to engage in social comparisons. Income comparisons are an important and easily measurable aspect of social comparisons. [Präg et al. \[2014\]](#) test this mechanism (specifically, income comparisons) in Europe. They investigate impacts of comparing one’s own health or health behaviors on the respective impacts on one’s health assessments and future health behaviors. Using income comparisons, they find that “individuals vary in their propensity to engage in

17. However, in the previous part, we do not find evidence of the impact of an individual’s rank in the income distribution on health.

social comparisons, and those with a higher propensity are also more likely to be affected by the outcomes of such comparisons”.

1.3.2 SHARE survey

Overview

Because we go deeper in our analysis of the health-income relationship, we still use the SHARE survey. As explained earlier, the 2008-2009 survey (Wave 3) “SHARELIFE” was extended to life stories by collecting information on the history of the respondents (Börsch-Supan [2017c]). We use this specific wave, SHARELIFE, which returns to the childhood of individuals interviewed in waves one et two, in order to investigate mechanisms described above. Thus, we will merge information obtained from wave three with current information of individuals in waves one and two. Using this database of individuals aged 50 and over, with its associated retrospective component about childhood of these individuals, allows to investigate the long-term effects. Indeed, we consider the whole life-cycle effects by studying factors at younger age and their impacts later in life. As a result, we are able to see the persistence of the mechanisms described above. Moreover, one should mention that this wave has not been used to study income-related health inequalities.

To be consistent, we investigate self-perceived health status, a subjective measure which has been proved to be a good predictor of an individual’s health, even if the majority of individuals report having a “good” health, which is relatively basic as an answer (see figure 1.5). Again, we use individual income from waves one and two, defined as the sum of individual imputed income for all household components. Figure 1.6 shows that highest incomes are in Switzerland, and Polish people have the lowest ones.

Proxies for the different pathways

The first mechanism, characterizing human capital and social goods, is investigated using three proxies. The first one is education because the more educated people are, the best their health should be. According to the number of years of schooling, majority of men reaches high-school (32.62%), whereas majority of women reaches primary education (29.85%). Then, we also include the number of books available in the household when individuals were ten years old. Figure 1.7 shows that the majority of our population only have access to few books.¹⁸ Access to literature at younger age increases human capital such that it should have a positive impact on earnings, and in turn, on health later in

18. This does not consider magazines, newspapers or school books.

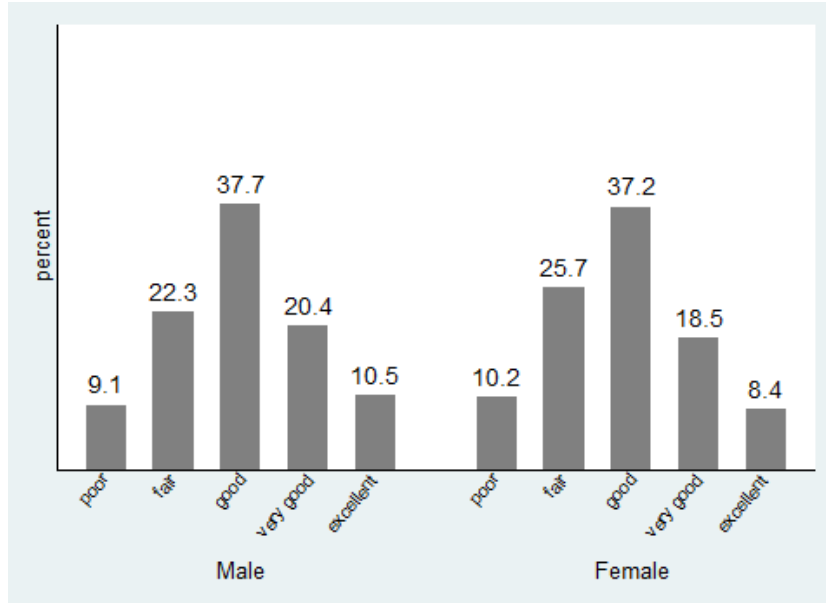


Figure 1.5 – Self-perceived health status in Europe - Part 2

Source: SHARE - Waves 1, 2 & 3

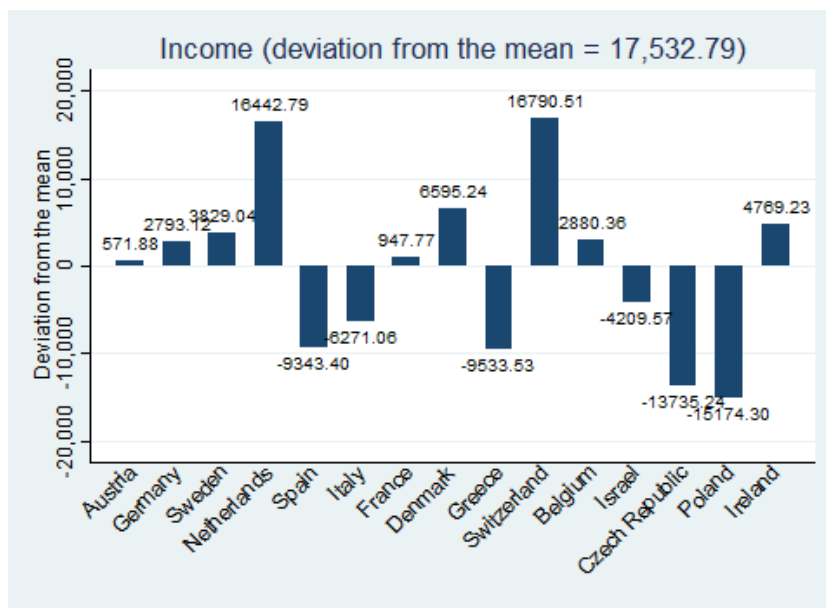


Figure 1.6 – Individual income, deviation from the mean (€) - Part 2

Source: SHARE - Waves 1 & 2

life. Finally, we also include percentage of health spending in the GDP for each country at each wave to characterize public goods, as explained previously in the first part (see figure 1.8). An increase in health spending should provide a more hospitable environment for individuals and thus improve their well-being and health.

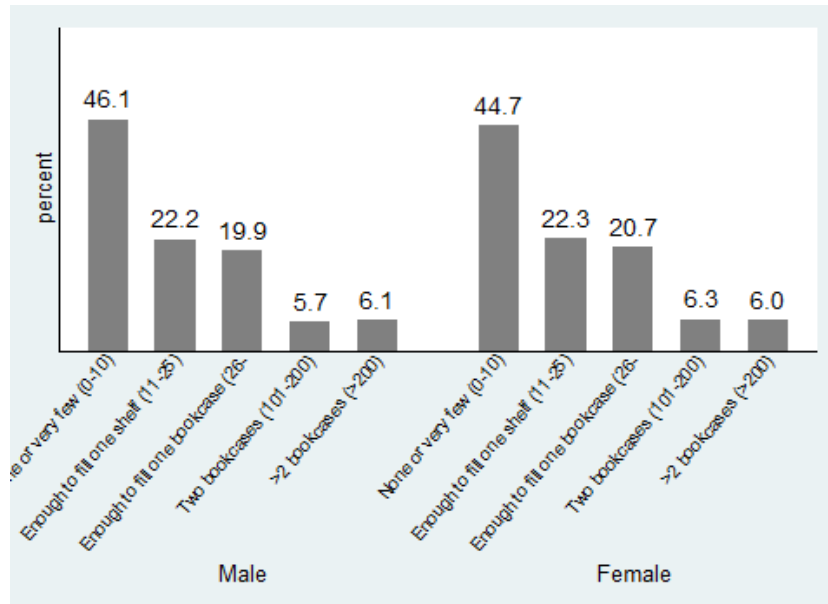


Figure 1.7 – Distribution of the number of books - Part 2

Source: SHARE - Wave 3

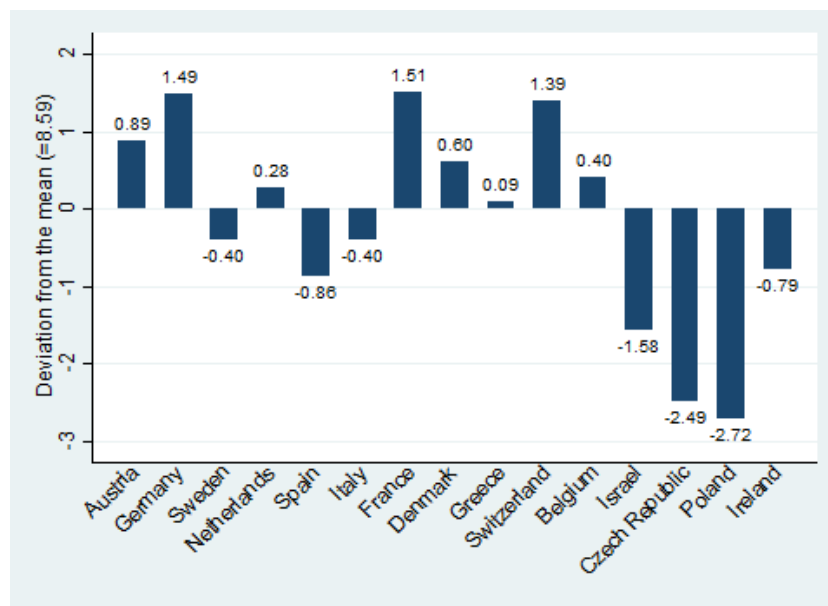


Figure 1.8 – Percentage of health spending in the GDP, deviation from the mean - Part 2

Source: OECD - From 2004 to 2007

Social capital, which corresponds to the second mechanism according to [Kawachi and Kennedy \[1999\]](#), is modeled using one variable from SHARELIFE. Erosion of social capital represents characteristics of the relative. Thus, the number of people living in the household when individuals were ten years old is used (five individuals on average). Being surrounding during childhood should have beneficial effects in building social capital. We

also investigate whether individuals ever received or gave help to people outside their household (as in the first part). This helps us to consider interactions and trust among individuals.

Finally, we find five proxies in SHARELIFE for the last mechanism corresponding to social comparisons. First, we consider the job of the member of the household who provided the majority of income when individuals were ten years old. Depending on jobs of breadwinners, earnings are different and this might create inequalities between individuals because of comparisons (individuals were not raised in equal “conditions”). This might impact health later through income inequalities when individuals follow a similar pattern from their breadwinner. Then, we consider the relative position to others when individuals were ten years old concerning their performance in both mathematics and languages at school (the majority responds having same performance at school). For an individual, being less good at school than his peers might have a negative effect on general well-being. This effect might have further consequences later in life on health. Lastly, we look at whether individuals faced a period of stress earlier in life (50.96% of individuals answer positively) and whether or not they have been discriminated (which concerns only 4.35% of them).¹⁹ Indeed, both elements, when experienced, have negative impacts on the cultural model of the standard of living in a society. These are negative for health of individuals.

1.3.3 Model and results

Econometric approach

In order to investigate mechanisms of income-related health inequalities, we focus on the strong version of the Income Inequality Hypothesis which is the hypothesis where our results are the strongest thanks to the concavity of income and to the negative impact of income inequalities in a country on individual health. We implement a dynamic ordered probit model to consider the panel dimension of the database. Our dependent variable is still self-perceived health status, now denoted as h_{it} . Thus, the latent variable specification of the model is the following:

$$\begin{aligned} h_{it}^* &= \beta_0 + x_{it}\beta_1 + x_{it}^2\beta_2 + II_{jt}\delta + X_{it}^1\alpha + X_{it}^2\theta + X_{it}^3\Omega + Z_{it}\Gamma + \epsilon_{it} \\ \epsilon_{it} &= \eta_i + \zeta_{it} \\ \forall i &= 1...N \quad \& \quad \forall t = 1...T_i \end{aligned} \tag{1.12}$$

19. Descriptive statistics are displayed in appendix section, table [1.7](#)

which is an extension of equation 1.2 (strong version of the Income Inequality Hypothesis) where we introduce proxies for the three different mechanisms (X_{it}^1 , X_{it}^2 , and X_{it}^3), in which T_i corresponds to the number of observations for an individual i .²⁰ Error terms are assumed to be normally distributed and can be decomposed into two terms, η_i an individual effect and ζ_{it} an idiosyncratic error.

X_{it}^1 is a set of variables for the first mechanism (human capital). It corresponds to educational background, number of books available in the place where individual i lived when he was 10, and the percentage of health spending in the GDP in each country. The second mechanism (social capital) is represented in the set of variables X_{it}^2 , using as information the number of people living in household when individual i was 10 and whether the individual ever received or gave help to someone in need. The set X_{it}^3 represents social comparisons (third mechanism) modeled with (i) job of the member of the household who provided the majority of income when individual i was 10; (ii) relative position of individual i to others when he was 10 concerning his performance in mathematics and languages at school; and, (iii) whether there were periods in which individual i were happier/stressed or being discriminated than during the rest of his life. Z_{it} is a set of variables of control such as age, age squared, gender, marital status, job status, groups of countries²¹ and self-perceived health status of individual i between 0 and 15 years old. We want to investigate income-related health inequalities such that we need to control the concavity of income in the estimates (x_{it} and x_{it}^2). Finally, we also include a measurement for income inequalities II_{jt} at date t in a country j , as included in the strong version of Income Inequality Hypothesis.

Analysis of the results

Table 1.2 shows coefficient estimates of equation 1.12 and table 1.12 indicates the associated average marginal effects on the probability to report each health outcome (from poor to excellent). Analyses are performed on a sample of 26,247 observations representing 17,933 individuals. As we want to investigate mechanisms through which income inequalities turn into health inequalities, we first need to focus on income. Income is positively related to self-perceived health status, in a concave way. The average marginal effect

20. h_{it}^* is a latent variable which underlies self-reported health status. Instead of observing the latent outcome, we observe an indicator of the category in which the latent indicator falls. As a result, the observed variable is equal to 1, 2, 3, 4 or 5 for “poor”, “fair”, “good”, “very good” or “excellent” with this probability $P(h_{it} = j | \mu, x_{it}, \eta_i) = \Phi(\mu_j - x_{it}\beta - \eta_i) - \Phi(\mu_{j-1} - x_{it}\beta - \eta_i)$, in which Φ is the standard normal cumulative distribution function.

21. Following the classification of the United Nations, northern Europe corresponds to Denmark, Ireland and Sweden; southern Europe is composed of Spain, Greece, Israel and Italy. Then, western Europe corresponds to France, Germany, the Netherlands, Belgium, Austria and Switzerland; whereas eastern Europe stands for Poland and Czech Republic.

implies that a 1% increase in income decreases the probability to report a fair health and increases the probability to report a very good health, which is in line with the literature. However, we do not find statistically significant effects for the others outcomes in terms of the average marginal effects. Focusing on the human capital argument, we use dummies for International Standard Classification of Education (ISCED) which indicates that having a lower grade level than high school is negatively related to self-perceived health status, whereas having a higher level is positively correlated to self-perceived health status. Having access to literature at younger age (number of books in household when the individual was 10) is also positively associated to self-perceived health status. Finally, individuals living in societies where governments invest a significant amount in health (relative to GDP) report being healthier. Average marginal effects associated to human capital tell the same story. An interesting result is the average marginal effect for individuals having access to more than 200 books when they were 10 years old. Having access to this large amount of literature at home as a child increases the probability to report being in excellent health later in life (compared with someone with only a limited access to books at younger age). Overall, an increase in human capital level improves health status such that it might decrease income inequalities as well. Concerning social capital, only variables about receiving or giving help have an impact on self-perceived health status. They illustrate interaction between individuals. Coefficient associated to the variable “received help from others” is negative and significant. This negative association means that people who are in bad health are the ones who receive help. In order, to legitimize this explanation, we also use the “reverse” variable: “given help to others”. In this case, the coefficient is positive and significant proving that people in good health offer their help. Furthermore, these effects are also legitimized with their associated average marginal effects. Finally, looking at the third mechanism on social comparisons, having stress periods or having being discriminated are negatively related to self-perceived health status. Indeed, according to the associated average marginal effects, having experienced one or the other earlier in life, increases the probability to report being in poor, fair or good health afterward, on one hand, and decreases the probability to be in very good or excellent health afterward, on the other hand. Moreover, focusing on the relative position to others in mathematics or in languages when individuals were 10 years old, coefficient estimates suggest that having a higher level increases self-perceived health status (compared to having the same level) whereas, having a lower level decreases health status. Thus, social comparisons, when badly experienced, have an effect on earnings later in life, such that it might, in turn, affect health status. We also investigate the impact of jobs of the breadwinner who provided majority of income when individuals were 10 years old. Unfortunately, no global pattern emerges from these results because there are a lot

of categories which are difficult to gather. However, results suggest that the absence of a main breadwinner in the household is negatively associated to self-perceived health status, such that, according to the average marginal effects, it increases the probability to report a poor or fair health and decreases the probability of reporting a very good or excellent health status (compared to a breadwinner working as a skilled agricultural).

Table 1.2 – Results of the ordered probit model - Part 2

Variables	Coefficients
Dependent variable:	Health _t
<u>Concavity</u>	
Income	1.40e-06*** (3.55e-07)
Income ²	−2.30e-13*** (7.71e-14)
<u>M1 - Human capital:</u>	
Education	
None	−0.590*** (0.072)
Primary	−0.353*** (0.034)
Secondary	−0.088** (0.034)
High-school	<i>Reference</i>
Tertiary	0.249*** (0.034)
Ph.D.	0.155 (0.183)
% Health spending	0.078*** (0.023)
Number of books in household when 10 y.o.	
0 to 10 books	<i>Reference</i>
11 to 25 books	0.113*** (0.031)
26 to 100 books	0.213*** (0.034)
101 to 200 books	0.183*** (0.053)
More than 200 books	0.282*** (0.056)
<u>M2: Social capital</u>	
People in household when 10 y.o	−0.004 (0.005)
Given help to others	0.177*** (0.021)
Received help from others	−0.405*** (0.025)
<u>M3: Social comparison</u>	
Stress period	−0.227*** (0.024)
Being discriminated	−0.254*** (0.055)
Job of breadwinner who provided majority of income when 10 y.o.	
Skilled agricultural or fishery worker	<i>Reference</i>
Manager	0.012 (0.061)
Professional	0.043 (0.066)
Technician	−0.012 (0.060)
Clerk	0.009 (0.046)
Sales worker	−0.007 (0.048)

Table 1.2 – Results of the ordered probit model - Part 2 (continued)

Variables	Coefficients
Craft or trades worker	−0.097*** (0.035)
Assembler	−0.108* (0.058)
Elementary occupation	−0.116*** (0.036)
Armed forced	−0.098 (0.094)
No main breadwinner	−0.204** (0.092)
Relative position to others in mathematics when 10 y.o	
Much better	0.079* (0.044)
Better	0.034 (0.030)
Same	<i>Reference</i>
Worse	0.017 (0.039)
Much worse	−0.226*** (0.082)
Relative position to others in languages when 10 y.o	
Much better	0.098** (0.045)
Better	0.049 (0.030)
Same	<i>Reference</i>
Worse	−0.090** (0.039)
Much worse	−0.019 (0.103)
<u>Covariates</u>	
Age	−0.013 (0.016)
Age ²	−0.0001 (0.0001)
Gini	0.027 (0.174)
GDP	0.0003*** (3.44e-06)
Marital status	
Married	<i>Reference</i>
Married, not living with spouse	0.026 (0.099)
Not married, living with partner	−0.059 (0.087)
Never married	−0.042 (0.046)
Divorced	−0.047 (0.041)
Widowed	−0.007 (0.033)
Current job status	
Retired	<i>Reference</i>
Employed	0.371*** (0.034)
Unemployed	−0.157** (0.063)

Table 1.2 – Results of the ordered probit model - Part 2 (continued)

Variables	Coefficients
Permanently sick	−1.372*** (0.060)
Homemaker	0.008 (0.035)
Other	0.072 (0.094)
Childhood health from 0 to 15 y.o.	
Excellent	<i>Reference</i>
Very good	−0.306*** (0.028)
Good	−0.625*** (0.032)
Fair	−0.817*** (0.052)
Poor	−0.936*** (0.081)
Varied a lot	0.419** (0.164)
Groups of countries¹	
Western Europe	<i>Reference</i>
Eastern Europe	−0.065 (0.125)
Northern Europe	0.473** (0.051)
Southern Europe	0.113** (0.049)
Time indicators	
Wave 1	<i>Reference</i>
Wave 2	−0.361*** (0.025)
μ_1	−2.455*** (0.619)
μ_2	−0.749 (0.618)
μ_3	1.076* (0.618)
μ_4	2.390*** (0.618)
Panel-level variance	1.344*** (0.047)
Numb. of obs.	26,247
Numb. of groups	17,933
***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are into parentheses below coefficients.	

1.4 Conclusion

In this study, we first underline the hypotheses which link health, income and income inequalities, and then we highlight mechanisms through which income inequalities involve health disparities. The aim of the first part of this chapter is to empirically investigate the evidence for the Absolute Income Hypothesis and both the strong and the weak versions of the Income Inequality Hypothesis, on one hand. On the other hand, the goal of the second part is to empirically investigate the impacts of human capital, social capital and social comparisons experienced during childhood, on health of people aged 50 and over in Europe. This study is one of the first analyzing the health-income relationship through different hypotheses at the same time, and investigating pathways of this relationship while considering the whole life-cycle effects, using the SHARE survey, as well as its retrospective component containing information on childhood of individuals.

Concerning the health-income relationship, we find evidence supporting the Absolute Income Hypothesis which states that people with higher incomes have better health outcomes. We also find evidence supporting the strong version of Income Inequality Hypothesis which argues that inequality affects all members in a society equivalently. In this hypothesis, we find that when there are high income inequalities in a country, people aged 50 and over feel less healthy. However, we do not find evidence supporting the weak version of Income Inequality Hypothesis which states that only the least well-off are hurt by income inequalities in a society. This hypothesis underlines the fact that income inequalities are more detrimental for the health of individuals with low incomes. Our qualitative results suggest that for low-income individuals, an increase in income inequalities in their country is positively related to report a better health status. Furthermore, for higher income individuals, an increase in income inequalities in their country is not related to report either a better or a lower health status. One limitation is the used of cross-sectional data without investigating possible endogeneity issues. Thus our results highlight statistical associations rather than causal effects. Finally, by implementing the generalized ordered probit, we control for potential problems of interpersonal comparisons and the results are very similar to the ones found with the ordered probit model.

Concerning, pathways through which income inequalities end up in health inequalities, we find evidence supporting the three mechanism, first highlighted by [Kawachi and Kennedy \[1999\]](#). Investments in human capital are positively and significantly related to self-perceived health status. Indeed, these investments are important to narrow inequalities in a society, and more specifically income inequalities. Reducing income inequalities, might in turn, reduce health disparities among individuals in a society. As a result, governments

should continue to promote education, access to culture and increase health investments. Moreover, focusing on social capital, defined as a set of collective resources an individual can put together, results suggest that there is solidarity between individuals because people in bad health seem to receive help from others whereas people in good health seem to offer their help when someone is in need. Then, social comparisons also seem to be important for health status. Effects of school difficulties during childhood accumulate over time, such that they are negatively associated with health after 50 years old. In addition, not feeling in accordance with the common standard way of life (being discriminated or stressed) is also negatively correlated with health. Tolerance and mutual aid are therefore two elements to put forward if we want to improve health of the population. Finally, because we considered information of individuals when they were 10 years old and the respective impacts on health at 50 years old and more, we give an insight on accumulated effects of what happened in childhood on health afterward.

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1.A Appendix A: descriptive statistics

1.A.1 First part

Table 1.3 – Descriptive statistics of the variables - Part 1

Variables	Mean	Std. Dev.	Min.	Max.
<u>Health</u>				
Self-perceived health status (N=63,626)	2.85	1.09	1	5
<u>Inequalities</u>				
Gini per country	0.39	0.05	0.31	0.48
Theil per country	0.33	0.19	0.16	0.82
<u>Other variables</u>				
Income	36,621.21	71,863.78	2	1.00e+07
GDP per country (2013 - Dollar US/capita)	39,726.43	11,543.57	26,160.08	92,781.41
Education (in years)	11.12	4.28	1	25
Age	67.12	10.06	50	103

Table 1.4 – Detailed descriptive statistics on health - Part 1

Health	Percentage of people
Poor (1)	10.81%
Fair (2)	27.01%
Good (3)	36.52 %
Very Good (4)	17.58%
Excellent (5)	8.18%

Table 1.5 – Detailed descriptive statistics on income - Part 1

Distribution	Income
5 %	3,828.99
25 %	12,446
50 %	24,659.55
75 %	46,200
95 %	103,897.2

Table 1.6 – Detailed descriptive statistics on the countries - Part 1

Country	% of people*	GDP - 2013** OECD	Indexes of inequality***	
			Theil index	Gini index
Austria	6.54%	45,132.54	0.176	0.322
Germany	8.71%	43,282.31	0.223	0.367
Sweden	7.06%	44,585.87	0.167	0.318
Netherlands	6.42%	46,749.31	0.215	0.354
Spain	9.75%	33,111.45	0.252	0.381
Italy	6.88%	34,836.43	0.373	0.423
France	6.86%	37,617.06	0.822	0.477
Denmark	6.37%	43,797.23	0.157	0.313
Switzerland	4.62%	56,896.91	0.214	0.355
Belgium	8.66%	41,863.94	0.384	0.454
Czech Republic	8.7%	28,962.64	0.212	0.351
Luxembourg	2.5%	92,781.4	0.264	0.397
Israel	3.56%	32,504.72	0.247	0.390
Slovenia	4.51%	28,675.43	0.369	0.451
Estonia	8.88%	26,160.08	0.681	0.449

*: Of each country in the full sample. **: Gross Domestic Product, Total dollar US/capita.

***: Values.

1.A.2 Second part

Table 1.7 – Descriptive statistics of the variables of interests and some covariates - Part 2

Variables	Mean	Std. dev.	Min.	Max.	Nb. of obs.
<u>Self-perceived health status</u>	%				67,205
Poor	9.68				
Fair	24.17				
Good	37.42				
Very good	19.38				
Excellent	9.35				
Income	17,532.79	35,700.98	0	5,878,646	65,640
Gini	0.44	0.08	0.28	0.59	65,640
Age	65.19	10.16	50	109	69,416
Retired	49.99%				65,400
<u>Education</u>	%				65,640
None	5.16				
Primary	27.75				
Middle	17.3				
High school	30.86				
University	18.52				
Ph.D.	0.42				
<u>Number of books</u>	%				41,914

Table 1.7 – Descriptive statistics of variables of interests and some covariates - Part 2
(continued)

Variables	Mean	Std. dev.	Min.	Max.	Nb. of obs.
0-10	45.32				
11-25	22.24				
26-100	20.33				
101-200	6.04				
> 200	6.06				
# Individuals in household	5.66	2.75	0	180	42,082
Ind. who had a stress period	50.96%				41,407
Ind. who have been discriminated	4.35%				42,255
<u>Math level</u>	%				40,815
Much better	10.54				
Better	24.42				
Same	50.74				
Worse	11.75				
Much worse	2.56				
<u>Language level</u>	%				40,712
Much better	10.36				
Better	26.09				
Same	50.14				
Worse	11.75				
Much worse	1.65				
<u>Job of breadwinner</u>	%				41,360
Manager	4.48				
Professional	3.8				
Technician or associate prof.	4.21				
Clerk	5.59				
Service, shop or market sales	7.46				
Skilled agricultural or fishery	27.76				
Craft or related trades	20.62				
Plant/machine operator	4.68				
Elementary occupation	18.09				
Armed forces	1.65				
No main breadwinner	1.67				
% Health spending in the GDP	8.59	1.18	5.80	10.423	65,640
% of individuals who gave help	30.75				65,121
% of individuals who received help	23.54				45,875
<u>Childhood health status</u>	%				42,246
Poor	2.14				
Fair	5.76				
Good	22.78				
Very good	33.05				
Excellent	35.8				

1.B Appendix B: additional econometric results

1.B.1 First part

Table 1.8 – Results of the ordered probit model for the pooled database - Part 1

Variables	Absolute Income	IIH	
	Hypothesis	Strong Version	Weak Version
Income	1.41e-06*** (4.74e-08)	1.94e-06*** (4.34e-08)	1.16e-06*** (4.76e-08)
Income squared	-1.78e-13*** (1.14e-14)	-2.39e-13*** (1.13e-14)	-1.46e-13*** (1.12e-14)
<u>Quintiles of income</u>			
Quintile 1			-0.379*** (0.019)
Quintile 2			-0.288*** (0.019)
Quintile 3			-0.184*** (0.019)
Quintile 4			-0.115*** (0.018)
Quintile 5			<i>Reference</i>
Index of inequalities (II) - Theil		-0.473*** (0.018)	-0.567*** (0.038)
Interaction quintile 1 and II			0.121* (0.053)
Interaction quintile 2 and II			0.054 (0.053)
Interaction quintile 3 and II			-0.012 (0.052)
Interaction quintile 4 and II			0.053 (0.052)
Interaction quintile 5 and II			<i>Reference</i>
<u>Co-variables</u>			
GDP		0.0002*** (3.03e-07)	0.0002*** (3.06e-07)
Age	-0.014*** (0.003)	-0.018*** (0.003)	-0.015*** (0.003)
Age squared	-0.0001*** (0.00002)	-0.0001** (0.0002)	-0.0006*** (0.00002)
Years of education	0.021*** (0.001)	0.019*** (0.0005)	0.017*** (0.001)
Gender = 1 if women	-0.055*** (0.005)	-0.057*** (0.005)	-0.050*** (0.005)
Married, living with spouse		<i>Reference</i>	
Registered partnership	-0.060*** (0.017)	-0.030* (0.017)	-0.026 (0.017)
Married, not living with spouse	-0.098*** (0.009)	-0.087*** (0.009)	-0.091*** (0.009)
Never married	-0.127*** (0.014)	-0.108*** (0.013)	-0.027** (0.014)
Divorced	-0.079*** (0.011)	-0.062*** (0.011)	0.016 (0.011)
Widowed	-0.046*** (0.009)	-0.055*** (0.009)	0.026*** (0.009)
Wave 1	0.139*** (0.009)	0.431*** (0.009)	0.469*** (0.009)

Table 1.8 – Results of the ordered probit model for the pooled database - Part 1 (continued)

Variables	Absolute Income	IIH	
	Hypothesis	Strong Version	Weak Version
Wave 2	0.094*** (0.009)	0.247*** (0.009)	0.272*** (0.009)
Wave 4	−0.024*** (0.006)	−0.001 (0.006)	0.003 (0.006)
Wave 5		Reference	
<u>Cut-points</u>			
Cut-point μ_1	−2.494 (0.104)	−1.960 (0.104)	−1.976 (0.105)
Cut-point μ_2	−1.46 (0.104)	−0.952 (0.105)	−0.962 (0.105)
Cut-point μ_3	−0.378 (0.104)	0.106 (0.104)	0.102 (0.105)
Cut-point μ_4	0.455 (0.104)	0.919 (0.104)	0.919 (0.105)
For AIH, dummies for countries are included but not reported, and available upon request.			
***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are in parentheses, below the coefficients.			

Table 1.9 – Absolute Income Hypothesis - Generalized ordered probit (wave 5) - Part 1

Variables	Health outcomes			
	1 to 2	2 to 3	3 to 4	4 to 5
Income	1.99e-06*** (2.76e-07)	2.25e-06*** (2.00e-07)	3.68e-06*** (2.44e-07)	3.81e-06*** (4.44e-07)
Income squared	−2.11e-13*** (2.90e-14)	−7.96e-13*** (1.17e-13)	−3.26e-13*** (4.71e-13)	−5.41e-12*** (1.55e-12)
<u>Co-variables</u>				
Age	0.037*** (0.010)	0.037*** (0.008)	0.026*** (0.009)	0.029*** (0.012)
Age squared	−0.0004*** (0.0001)	−0.0004*** (0.0001)	−0.0004*** (0.0001)	−0.0003*** (0.0001)
Years of education	0.031*** (0.002)	0.038*** (0.001)	0.036*** (0.001)	0.024*** (0.002)
Gender = 1 if women	0.066*** (0.016)	−0.014 (0.012)	−0.005 (0.012)	−0.002** (0.016)
Married, living with spouse	<i>Reference</i>			
Registered partnership	−0.063 (0.069)	−0.093** (0.046)	0.029 (0.045)	−0.027 (0.057)
Married, not living with spouse	−0.251*** (0.062)	−0.112** (0.049)	−0.0001 (0.053)	0.118* (0.069)
Never married	−0.048 (0.032)	−0.068*** (0.024)	−0.038 (0.026)	−0.065* (0.035)
Divorced	−0.157*** (0.026)	−0.059*** (0.019)	0.050*** (0.021)	0.060** (0.027)
Widowed	−0.017 (0.021)	−0.026 (0.017)	0.002 (0.020)	−0.015 (0.029)
Retired	<i>Reference</i>			
Employed	0.398*** (0.029)	0.312*** (0.019)	0.203*** (0.019)	0.174*** (0.025)
Unemployed	−0.222*** (0.047)	−0.191*** (0.035)	−0.233*** (0.038)	−0.126** (0.053)
Permanently sick	−1.196*** (0.033)	−1.268*** (0.038)	−1.307*** (0.054)	−0.963*** (0.076)
Home-maker	−0.088*** (0.029)	−0.052** (0.022)	−0.047* (0.025)	−0.006 (0.035)
Other	−0.354*** (0.041)	−0.173*** (0.037)	−0.145*** (0.046)	−0.017 (0.064)
1 to 2: poor to fair; 2 to 3: fair to good; 3 to 4: good to very good; 4 to 5: very good to excellent.				
Dummies for countries are included but not reported, and available upon request.				
***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are in parentheses, below the coefficients.				

Table 1.10 – IIH, strong version - Generalized ordered probit (wave 5) - Part 1

Variables	Health outcomes			
	1 to 2	2 to 3	3 to 4	4 to 5
Income	1.75e-06*** (2.69e-07)	2.34e-06*** (1.97e-07)	3.89e-06*** (2.38e-07)	3.20e-06*** (4.42e-07)
Income squared	-1.89e-13*** (2.82e-14)	-8.28e-13*** (1.18e-13)	-3.75e-12*** (4.72e-13)	-5.18e-12*** (1.60e-12)
Index of inequalities (Theil)	-0.095** (0.041)	-0.369*** (0.031)	-0.739*** (0.035)	-0.475*** (0.048)
<u>Mechanisms</u>				
1st: % Health exp. in the GDP	0.059*** (0.005)	0.087*** (0.004)	0.073*** (0.004)	0.082*** (0.006)
2nd: Received help from others	-0.214*** (0.009)	-0.193*** (0.008)	-0.134*** (0.009)	-0.089*** (0.013)
2nd bis: Given help to others	0.001*** (0.0001)	0.001*** (0.0001)	0.001*** (0.0001)	0.001*** (0.0001)
3rd: Life satisfaction	0.195*** (0.004)	0.215*** (0.003)	0.239*** (0.004)	0.238*** (0.006)
<u>Co-variables</u>				
GDP	2.52e-06*** (8.66e-07)	1.41e-06** (6.04e-07)	-4.87e-07 (6.36e-07)	5.94e-07 (8.72e-07)
Age	0.019* (0.010)	0.004 (0.008)	0.013 (0.009)	0.019* (0.012)
Age squared	-0.0003*** (0.0001)	-0.0002*** (0.0001)	-0.0003*** (0.0001)	-0.0003*** (0.0001)
Years of education	0.025*** (0.002)	0.029*** (0.001)	0.028*** (0.001)	0.021*** (0.002)
Gender = 1 if women	0.069*** (0.016)	-0.018 (0.012)	-0.003 (0.012)	-0.0004 (0.016)
Married, living with spouse	<i>Reference</i>			
Registered partnership	-0.023 (0.071)	-0.053 (0.047)	0.034 (0.045)	0.014 (0.058)
Married, not living with spouse	-0.131** (0.065)	0.005 (0.051)	0.091* (0.054)	0.122* (0.072)
Never married	0.033 (0.034)	0.023 (0.025)	0.064** (0.027)	0.001 (0.036)
Divorced	-0.046* (0.028)	0.062*** (0.021)	0.166*** (0.022)	0.122*** (0.028)
Widowed	0.053** (0.023)	0.069*** (0.018)	0.076*** (0.022)	0.022 (0.031)
Retired	<i>Reference</i>			
Employed	0.344*** (0.030)	0.225*** (0.019)	0.177*** (0.019)	0.176*** (0.025)
Unemployed	-0.141*** (0.048)	-0.097*** (0.035)	-0.110*** (0.039)	0.012 (0.054)
Permanently sick	-1.016*** (0.034)	-1.121*** (0.034)	-1.098*** (0.056)	-0.744*** (0.084)
Home-maker	-0.074*** (0.029)	-0.033 (0.022)	-0.076*** (0.025)	-0.044 (0.035)
Other	-0.299*** (0.043)	-0.114*** (0.038)	-0.090* (0.048)	0.048 (0.067)

1 to 2: poor to fair; 2 to 3: fair to good; 3 to 4: good to very good; 4 to 5: very good to excellent.

***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are in parentheses, below the coefficients.

Table 1.11 – IIH, weak version - Generalized ordered probit (wave 5) - Part 1

Variables	Health outcomes			
	1 to 2	2 to 3	3 to 4	4 to 5
Income	1.97e-06*** (3.06e-07)	3.03e-06*** (2.43e-07)	5.92e-06*** (3.15e-07)	7.65e-06*** (6.10e-07)
Income squared	−2.09e-13*** (3.17e-14)	−1.14e-12*** (1.25e-13)	−6.03e-12*** (5.21e-13)	−1.60e-11*** (1.92e-12)
<u>Quintiles of income</u>				
Quintile 1	−0.145*** (0.055)	−0.195*** (0.039)	−0.003 (0.043)	0.070 (0.059)
Quintile 2	−0.099* (0.054)	−0.159*** (0.038)	−0.014 (0.039)	0.079 (0.059)
Quintile 3	−0.061 (0.054)	−0.043 (0.037)	0.018 (0.037)	0.025 (0.047)
Quintile 4	−0.012 (0.056)	−0.020 (0.036)	0.055 (0.034)	0.023 (0.043)
Quintile 5	Reference			
Index of inequalities (II) - Theil	−0.319*** (0.101)	−0.790*** (0.065)	−1.077*** (0.065)	−0.899*** (0.084)
Interaction quintile 1 and II	−0.204* (0.120)	0.079 (0.088)	−0.039 (0.107)	0.084 (0.147)
Interaction quintile 2 and II	−0.162 (0.123)	0.097 (0.087)	0.048 (0.101)	0.029 (0.138)
Interaction quintile 3 and II	−0.163 (0.125)	−0.048 (0.088)	−0.013 (0.098)	0.144 (0.129)
Interaction quintile 4 and II	−0.058 (0.132)	0.066 (0.088)	0.001 (0.093)	0.098 (0.124)
Interaction quintile 5 and II	Reference			
<u>Co-variables</u>				
GDP	0.0001*** (8.30e-07)	9.96e-06*** (6.31e-07)	3.83e-06*** (6.99e-07)	2.17e-06*** (9.91e-07)
Age	0.034*** (0.010)	0.023*** (0.008)	0.029*** (0.008)	0.034** (0.011)
Age squared	−0.0004*** (0.0001)	−0.0003*** (0.0001)	−0.0004*** (0.0001)	−0.0004*** (0.0001)
Years of education	0.025*** (0.002)	0.029*** (0.001)	0.028*** (0.001)	0.022*** (0.002)
Gender = 1 if women	0.066*** (0.015)	−0.016 (0.011)	0.0004 (0.012)	0.007 (0.016)
Married, living with spouse	Reference			
Registered partnership	0.053 (0.067)	0.023 (0.045)	0.075* (0.044)	0.049 (0.056)
Married, not living with spouse	−0.203*** (0.061)	−0.091* (0.049)	−0.014 (0.052)	0.052 (0.068)
Never married	0.034 (0.033)	0.014 (0.024)	0.042 (0.026)	−0.008 (0.035)
Divorced	−0.079*** (0.027)	0.009 (0.020)	0.107*** (0.021)	0.085*** (0.027)
Widowed	0.024 (0.022)	0.015 (0.018)	0.019 (0.021)	−0.015 (0.029)
Retired	Reference			
Employed	0.374*** (0.029)	0.251*** (0.019)	0.206*** (0.018)	0.188*** (0.024)
Unemployed	−0.188*** (0.046)	−0.169*** (0.034)	−0.221*** (0.038)	−0.128** (0.053)
Permanently sick	−1.162*** (0.032)	−1.262*** (0.033)	−1.245*** (0.054)	−0.923*** (0.080)
Home-maker	−0.062** (0.027)	−0.021 (0.021)	−0.081*** (0.024)	−0.069** (0.034)

Table 1.11 – IIH, weak version - Generalized ordered probit (continued) - Part 1

Variables	Health outcomes			
	1 to 2	2 to 3	3 to 4	4 to 5
Other	−0.317*** (0.041)	−0.152*** (0.037)	−0.148*** (0.046)	−0.017 (0.064)
1 to 2: poor to fair; 2 to 3: fair to good; 3 to 4: good to very good; 4 to 5: very good to excellent.				
***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are in parentheses, below the coefficients.				

1.B.2 Second part

Table 1.12 – Average marginal effects associated to the ordered probit model - Part 2

Variables	Average marginal effects				
Pr(Health _t)=	Poor	Fair	Good	Very good	Excellent
<u>Concavity</u>					
Income	−1.01e-07 (8.77e-08)	−1.68e-07** (6.84e-08)	−8.23e-09 (9.36e-08)	1.31e-07* (7.00e-08)	1.461e-07 (1.07e-07)
<u>M1 - Human capital:</u>					
% Health spending	−0.006*** (0.002)	−0.009*** (0.003)	−0.0005*** (0.0002)	0.007*** (0.002)	0.008*** (0.002)
Education					
None	0.050*** (0.007)	0.074*** (0.009)	−0.012*** (0.004)	−0.060*** (0.007)	−0.052*** (0.005)
Primary	0.027*** (0.003)	0.045*** (0.004)	−0.002** (0.001)	−0.036*** (0.004)	−0.034*** (0.003)
Secondary	0.006** (0.002)	0.011** (0.004)	0.001** (0.0003)	−0.009** (0.003)	−0.009** (0.004)
High-school			<i>Reference</i>		
Tertiary	−0.015*** (0.002)	−0.031*** (0.004)	−0.008*** (0.001)	0.024*** (0.003)	0.029*** (0.004)
Ph.D.	−0.009 (0.010)	−0.019 (0.023)	−0.004 (0.006)	0.015 (0.017)	0.0184 (0.022)
Number of books in household when 10 y.o.					
0 to 10 books			<i>Reference</i>		
11 to 25 books	−0.008*** (0.002)	−0.014*** (0.004)	−0.0002 (0.0002)	0.011*** (0.003)	0.011*** (0.003)
26 to 100 books	−0.015*** (0.002)	−0.026*** (0.004)	−0.002*** (0.001)	0.021*** (0.003)	0.022*** (0.004)
101 to 200 books	−0.013*** (0.004)	−0.023*** (0.007)	−0.001 (0.001)	0.018*** (0.005)	0.019*** (0.006)
More than 200 books	−0.019*** (0.004)	−0.035*** (0.007)	−0.003** (0.001)	0.027*** (0.005)	0.030*** (0.006)
<u>M2: Social capital</u>					
People in hh when 10 y.o	0.0003 (0.0003)	0.0005 (0.0006)	0.0003 (0.0003)	−0.0004 (0.0004)	−0.0005 (0.0005)
Given help to others	−0.013*** (0.002)	−0.021*** (0.003)	−0.001*** (0.0003)	0.017*** (0.002)	0.019*** (0.002)
Received help from others	0.029*** (0.002)	0.049*** (0.003)	0.002*** (0.001)	−0.038*** (0.002)	0.043*** (0.003)
<u>M3: Social comparison</u>					
Stress period	0.017*** (0.002)	0.027*** (0.003)	0.001*** (0.0004)	−0.021*** (0.002)	−0.024*** (0.003)

Table 1.12 – Average marginal effects - Part 2 (continued)

Variables	Average marginal effects				
Pr(Health _t)=	Poor	Fair	Good	Very good	Excellent
Being discriminated	0.019*** (0.004)	0.031*** (0.007)	0.002*** (0.0005)	−0.024*** (0.005)	−0.027*** (0.006)
Job of breadwinner who provided majority of income when 10 y.o.					
Skilled agricultural	<i>Reference</i>				
Manager	−0.001 (0.004)	−0.001 (0.007)	−0.0001 (0.0005)	0.001 (0.006)	0.001 (0.006)
Professional	−0.003 (0.005)	−0.005 (0.008)	−0.0004 (0.001)	0.004 (0.006)	0.005 (0.007)
Technician	0.001 (0.004)	0.001 (0.007)	0.0001 (0.0003)	−0.001 (0.006)	−0.001 (0.006)
Clerk	−0.001 (0.004)	−0.001 (0.007)	−0.0001 (0.004)	0.001 (0.005)	0.001 (0.006)
Sales worker	0.001 (0.004)	0.001 (0.006)	0.0001 (0.0003)	−0.001 (0.005)	−0.001 (0.005)
Craft or trades	0.007*** (0.003)	0.012*** (0.004)	0.0003** (0.0001)	−0.009*** (0.003)	−0.010*** (0.004)
Assembler	0.008* (0.005)	0.013* (0.007)	0.0001 (0.0003)	−0.010* (0.006)	−0.011* (0.006)
Elementary occ.	0.009*** (0.003)	0.014*** (0.004)	0.0002 (0.0002)	−0.011*** (0.003)	−0.012*** (0.004)
Armed forced	0.007 (0.007)	0.012 (0.011)	0.0001 (0.0005)	−0.009 (0.009)	−0.009 (0.009)
No main breadwinner	0.016** (0.008)	0.024** (0.011)	−0.001 (0.002)	−0.019** (0.009)	−0.020** (0.008)
Relative position to others in mathematics when 10 y.o					
Much better	−0.006* (0.003)	−0.009* (0.005)	−0.0007 (0.0006)	0.007* (0.004)	0.008* (0.005)
Better	−0.003 (0.003)	−0.004 (0.004)	−0.0002 (0.0002)	0.003 (0.003)	0.00 (0.003)
Same	<i>Reference</i>				
Worse	−0.001 (0.003)	−0.002 (0.005)	−0.0001 (0.0002)	0.002 (0.004)	0.002 (0.004)
Much worse	0.018** (0.007)	0.027*** (0.009)	−0.002 (0.002)	−0.022*** (0.008)	−0.022*** (0.007)
Relative position to others in languages when 10 y.o					
Much better	−0.007** (0.003)	−0.012** (0.005)	−0.001 (0.0007)	0.009** (0.004)	0.011** (0.005)
Better	−0.004 (0.002)	−0.006 (0.004)	−0.0004 (0.0003)	0.005 (0.003)	0.005 (0.003)
Same	<i>Reference</i>				
Worse	0.007** (0.003)	0.011** (0.005)	−0.00004 (0.0002)	−0.009** (0.004)	−0.009** (0.004)
Much worse	0.001 (0.008)	0.002 (0.005)	0.0001 (0.0003)	−0.002 (0.009)	−0.002 (0.011)
<u>Co-variables</u>					
Age	0.002*** (0.0001)	0.003*** (0.0002)	0.00007 (0.0001)	−0.002*** (0.0002)	−0.002*** (0.0002)
Gini	−0.002 (0.013)	−0.003 (0.021)	−0.0002 (0.001)	0.003 (0.016)	0.003 (0.018)
GDP	−2.63e-06*** (3.44e-07)	−4.36e-06*** (4.11e-07)	−2.23e-07*** (6.65e-08)	3.41e-06*** (3.13e-07)	3.80e-06*** (3.63e-07)
Marital status					
Married	<i>Reference</i>				
Married, not living with sp.	0.004 (0.007)	0.007 (0.010)	0.0002** (0.0001)	−0.006 (0.008)	−0.006 (0.009)

Table 1.12 – Average marginal effects - Part 2 (continued)

Variables	Average marginal effects				
Pr(Health _t)=	Poor	Fair	Good	Very good	Excellent
Living with partner	−0.002 (0.007)	−0.003 (0.012)	−0.0002 (0.001)	0.002 (0.009)	0.003 (0.011)
Never married	0.003 (0.003)	0.005 (0.006)	0.0002 (0.0002)	−0.004 (0.004)	−0.004 (0.005)
Divorced	0.003 (0.003)	0.006 (0.005)	0.0002 (0.0001)	−0.004 (0.004)	−0.005 (0.004)
Widowed	0.001 (0.002)	0.001 (0.004)	0.0001 (0.0002)	−0.001 (0.003)	−0.001 (0.003)
Current job status					
Retired			<i>Reference</i>		
Employed	−0.023*** (0.002)	−0.048*** (0.005)	−0.008*** (0.001)	0.037*** (0.004)	0.042*** (0.004)
Unemployed	0.012** (0.005)	0.020** (0.008)	−0.002 (0.001)	0.016** (0.007)	−0.015*** (0.006)
Permanently sick	0.172*** (0.010)	0.137*** (0.004)	−0.105*** (0.008)	−0.127*** (0.005)	−0.077*** (0.003)
Homemaker	−0.001 (0.003)	−0.001 (0.005)	8.59e-06 (0.00003)	0.001 (0.004)	0.001 (0.003)
Other	−0.005 (0.006)	−0.009 (0.012)	−0.0002 (0.001)	0.007 (0.009)	0.007 (0.009)
Childhood health from 0 to 15 y.o.					
Excellent			<i>Reference</i>		
Very good	0.019*** (0.002)	0.038*** (0.004)	0.008 *** (0.0009)	−0.029*** (0.003)	−0.036*** (0.003)
Good	0.045*** (0.003)	0.077*** (0.004)	0.004*** (0.001)	−0.061*** (0.003)	0.065*** (0.003)
Fair	0.064*** (0.005)	0.100*** (0.006)	−0.004 (0.003)	−0.080*** (0.005)	−0.079*** (0.004)
Poor	0.077*** (0.009)	0.113*** (0.009)	−0.011** (0.005)	−0.092*** (0.008)	−0.088*** (0.005)
Varied a lot	0.027** (0.013)	0.052** (0.020)	0.008*** (0.001)	−0.040** (0.016)	−0.047*** (0.016)
Groups of countries					
Western Europe			<i>Reference</i>		
Eastern Europe	0.005 (0.009)	0.008 (0.015)	0.0002* (0.0001)	−0.006 (0.012)	−0.007 (0.012)
Northern Europe	−0.029*** (0.003)	−0.058*** (0.006)	−0.012*** (0.002)	0.044*** (0.005)	0.055*** (0.006)
Southern Europe	−0.008** (0.003)	−0.014** (0.006)	−0.0009* (0.0006)	0.011** (0.005)	0.012** (0.005)
Time					
Wave 1			<i>Reference</i>		
Wave 2	0.026*** (0.002)	0.044*** (0.003)	0.003*** (0.0006)	−0.034*** (0.002)	−0.039*** (0.003)
Numb. of obs.	26,247				
Numb. of groups	17,933				

***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are into parentheses below the coefficients.

Chapter 2

Health and income: testing for
causality on European elderly people

Abstract

Socio-economic status and health status are positively related which is known as the health-income gradient. However, one must be careful in considering the causal impact of income on health, since the reverse causality might be at play. Income inequalities are an important factor in health inequality such that policy makers who aim at improving general health or narrowing health inequalities using public policies, need to understand the sources and the true direction of the causality between income and health. We thus investigate bivariate causal effects from income to health and from health to income by highlighting the Granger causality. Using the Survey of Health, Ageing and Retirement in Europe (SHARE), we find evidence of persistent causal effects running from income to health and from health to income. Results, using a Full Information Maximum Likelihood estimator (FIML), suggest that considering a simultaneous equations approach is required because there are individual unobserved factors common to both equations (statistically significant correlation between the two equations).

Keywords: Granger causality; income; simultaneity; self-assessed health; FIML.

JEL Classification: C32; C33; D31; I10; J14.

2.1 Introduction

A topic at the center of health economics is the relationship between health and individual income, with the consensus view among researchers being that higher socioeconomic status is associated with better health (Preston [1975]). This relationship has been reviewed using many health outcomes in different countries (e.g. Van Doorslaer et al. [1997] using self-assessed health). While this relationship appears to be well-known, this is not the case concerning its causal interpretation. There are many possible pathways through which earnings can impact health. Indeed, there is a causal relationship between socioeconomic status, or more specifically income, and health of the former on the latter (Frijters et al. [2005]; Apouey and Clark [2015]). However, we can also think of the reverse association, for instance stating that poor health status may influence income, by reducing the ability to work (Michaud and Van Soest [2008]). This lack of a clear understanding of causality and the direction of the causal effects is an important omission. Since income inequalities are an important factor in health inequalities (e.g. Carrieri and Jones [2016]), policy makers who aim at improving general health or narrowing health inequalities in a society, need to understand the sources and the true direction of the causality between income and health. The difficulty in disentangling cause and effect is due to endogeneity, more specifically whenever health and income mutually determine one another, there are simultaneity issues. Since simultaneous causality in both directions may exist, testing causal impacts require considering on one hand, the impact of income on health, and on the other, the impact of health on income. Different econometric methods have been used to fix this issue such as instrumental variables method or exogenous income shocks and health shocks, but without finding a common consensus about the direction of the causality (from income to health according to Halliday [2017] or from health to wealth according to Michaud and Van Soest [2008]). While these studies disagree about the direction of the causality between health and income, they provide interesting insights. However, majority of these studies do not adequately consider heterogeneity due to individual fixed effects that may be associated with both income and health. The two previous cited studies address this concern by employing dynamic panel techniques to investigate causality. Nevertheless, this analysis deepens the link between health and income and is different from the latter since we explicitly bring to the forefront the Granger causality while taking into account other information and concerns. Indeed, on one hand, Halliday [2017]’s study differs from ours in two points. First, he only considers the impact of income on health, while we consider this relationship, as well as the impact of health on income in order to highlight bidirectional causal links. Second, our database contains more information (specifically information on morbidity indicators) so that we can investigate more control variables in the estimates to make robust links. On the other hand,

[Michaud and Van Soest \[2008\]](#) work on the Health and Retirement Study, a population of U.S. couples aged 50 and older, a similar population than ours, but focus on wealth. However, instead of considering two univariate relationships (one considering the impact of income on health and the other one considering the impact of health on wealth), we implement a simultaneous equations approach to consider the possible existence of individual unobserved factors common to both equations. Thus, we tackle endogeneity issues using a specific structure for the error terms.

This chapter contributes to these subjects by bringing the Granger causality to the forefront. We use the European SHARE dataset, where the temporal dimension of the data is employed to evaluate and predict changes in self-perceived health status according to income, and the reverse association. Full Information Maximum Likelihood estimator is implemented, using a simultaneous equations model to investigate bivariate causality between health and income, on European elderly people.

In section [2.2](#) we present the theoretical framework of the causal relationship between income and health. Section [2.3](#) describes the Survey of Health, Ageing and Retirement in Europe. In section [2.4](#) we detail the econometric framework, as well as the results. Section [2.5](#) concludes the chapter.

2.2 The causal relationship

The relationship between self-perceived health status and individual income is heavily documented in health economics. Self-perceived health status assesses the general perceived health of an individual. It represents an important predictor of an individual's health since it combines different elements that an individual knows about his own health. This subjective measure also integrates factors which are not always considered by health professionals such as individuals' beliefs and attitudes towards health commodity for instance. Thus, this subjective indicator is a good predictor of people's actual health status ([Benitez-Silva et al. \[2004\]](#); [DeSalvo et al. \[2005\]](#) ; [Bond et al. \[2006\]](#)). Recent studies modeling the dynamics of health-income relationship question the existence of a causal effect of income, or other socioeconomic status, on health (see for instance, recent studies by [Kim and Ruhm \[2012\]](#); [Apouey and Clark \[2015\]](#) or [Halliday \[2017\]](#)). Direction of causality is considered to be an important issue much debated among economists, since the lack of a clear and true understanding constitutes a major shortcoming for policy makers, who aim to narrow health inequalities and improve health. In this chapter, we investigate the direction of the causality by tackling the question of what happens to a person's health (*resp.* income) when they experience a variation in their income (*resp.*

health). In the literature, some papers use instrumental variables methods or exogenous income shocks to investigate a causal link from income to health, and solve income endogeneity issues. Concerning instrumental variables method, authors investigate different kind of instruments and the majority find that income has a positive and significant effect on health (Ettner [1996]; Economou and Theodossiou [2011]; Halliday [2017]). Indeed, Ettner [1996] examines the effect of income on different health proxies, such as self-assessed health, daily activity limitations, proxies for alcohol abuse and others. She uses cross-sectional data from a number of US surveys collected in the 1980's. Depending on the health outcome, she uses ordered probit, probit or two-part models. The problem of reverse causality is addressed using parental education, work experience, spousal characteristics and unemployment rate as instruments. In each case, Ettner finds that income still has a significant impact on health. Economou and Theodossiou [2011] use European data and control for income endogeneity using inheritance, children's education and art collection as instruments. Results indicate a strong and positive relationship between household income and health. However, the use of cross-sectional data weakens the causal statement. More recently, Halliday [2017] employs data from the Panel Study of Income and Dynamics (US) to investigate the causal link of income on health. He implements a GMM procedure on a model in first-differences, and uses further lag variables as instruments. His results establish a causal link running from income to health in the case of married individuals. However, Michaud and Van Soest [2008] do not find a significant impact of wealth on health, using inheritances as instrument for wealth. They investigate the pathways of the health-wealth gradient using six waves of the Health and Retirement Study, implemented in a GMM framework. On the other hand, exogenous increases in income are investigated to identify a causal effect of wealth or income on health. These exogenous shocks result from lottery winnings (Lindahl [2005]; Gardner and Oswald [2007]; Apouey and Clark [2015]), inheritances (Meer et al. [2003]; Kim and Ruhm [2012]) or other economic changes (Frijters et al. [2005]; Adda et al. [2009]; McInerney et al. [2013]). Findings from these studies suggest that lottery wins have a positive effect on mental health. Indeed, Lindahl [2005] uses Swedish longitudinal data to account for the health-income relationship. In this paper, lottery prizes are used to provide exogenous variations in income. Lindahl focuses on different aspects of health and the results are varied. He finds that lottery winnings have a positive impact on mental health and imply lower body mass index.¹ Gardner and Oswald [2007] explore the causality issue using medium-sized lottery wins (£1000+) as their instrument.² They find that mental health is positively affected by income. Apouey and Clark [2015] study the exogenous impact of

1. However, lottery winnings have no effect on other physical health problems.

2. They use medium-sized lottery wins because individuals who get no win are almost indistinguishable from individuals with a small win in the database used.

income on different health outcomes with English data, using lottery winnings. They find that positive income shocks do not have a significant effect on general health, but have an effect on mental health. Nonetheless, inheritances do not have a significant effect on health. [Meer et al. \[2003\]](#), on American data, use the amount of inheritances and gifts received over the last five years (amounts larger than US \$10,000). Results suggest that wealth does not have a significant effect on health. The validity of inheritance information is also open to debate, as noted by the authors. [Kim and Ruhm \[2012\]](#), using eight waves of the Health and Retirement Survey, find that bequests (larger or equal to US \$10,000) do not have a significant impact on health. Finally, variations in income due to changes in the economic environment suggest that health is positively impacted by exogenous income shocks. [Frijters et al. \[2005\]](#) analyze German data and their instrumental method is to use an exogenous change in income due to the fall of the Berlin wall. In other words, they investigate whether there was a causal effect of income changes on the health satisfaction of East and West Germans in the years following reunification. Results suggest a positive impact of income on health. [Adda et al. \[2009\]](#) model income and health as a stochastic process evolving over the life cycle, created using a synthetic cohort dataset which is based on successive years of micro data from several English cross-sectional surveys. They exploit the fact that, at the cohort level, over the eighties and nineties, there were sizable changes in income, mainly due to changes in the macroeconomic environment. According to their results, income variations have little effect on health, but affect health behaviors and mortality. [McInerney et al. \[2013\]](#) use exogenous variation in the interview dates of the 2008 Health and Retirement Survey to assess wealth losses' impacts on mental health. They find that feeling of depression and use of antidepressant drugs increase after the 2008 stock market crash.

Concerning literature on the impacts of health on income, there are less researches. The main idea is that having a bad health may reduce the ability to work efficiently such that it has a negative effect on health. Moreover, poor health can also be associated to important medical expenditures such that it might imply a decrease in income. [Grossman \[1972b\]](#)'s model of health production is a good starting-point of how health is a factor because it allows to understand that health may be seen as a stock, and income might be related to saving motives. [Smith \[1999\]](#) explains that “arithmetically, savings may fall as current health deteriorates because it reduces current period income or increases either consumption or out-of-pocket medical expenses”. Moreover, income might be affected by the onset of health events which might reduce the amount of labor supplied. As a result, we can consider that health is a form of human capital. Other things being equal, we expect healthier people to be more productive, and more productive workers tend to earn higher wages and work more. Using exogenous health shocks, this result is supported by

Wagstaff [2007] and Halla and Zweimüller [2013]. Using a Vietnamese database, Wagstaff [2007] finds that some health shocks (particularly the death of a working-age household member) have a negative impact on earned income. Results also suggest that health shocks have more impact on incomes of urban households than of rural ones. Then, Halla and Zweimüller [2013] compare workers who get in an accident on the way to work with workers who do not (considered as health shocks) in order to implement a quasi-experimental experience. Using a fixed effects difference-in-differences approach on Austrian data, they show a persistent negative causal effect of health shocks on employment and earnings. However, this negative impact of health shocks on income is not always found in studies. For instance, Charles [2003], using the Panel Study of Income Dynamics, studies the dynamic effects of a disability on earnings and finds that earnings have already dropped one year before the onset of the disability. On the other hand, Michaud and Van Soest [2008] use instrumental variables method to consider this relationship. They instrument health with the onset of critical health conditions (like cancer for severe condition or high blood pressure for mild condition). They find strong evidence of causal effects from health of household members on household wealth.

Moreover, we should be aware that in the causal relationship from income to health³, there are likely to be effects which need to be controlled. In figure 2.1, we notice that health status is a decreasing function of age.⁴ When people get older, they tend to consider themselves as being less healthy. Changes in health status are thus partly due to the age. As a result, researchers need to control for this factor if they want to establish a causal link between income and health. Indeed, self-rated health assimilates morbidity, which in turn depends on diagnosed health problems, interactions with health professionals, as well as diseases (Tubeuf et al. [2008]). Traditional measures of morbidity provide important information about levels of health. Morbidity corresponds to the incidence of diseases. It seems that morbidity is a good predictor of the self-assessment of health status, and this is why we control for its effect in the health-income relationship. We model the latter thanks to indicators characterized by chronic illnesses and disability. The last impact we need to be careful about is technological progress. Examining trends and patterns in mortality helps to explain changes and differences in health status, permitting evaluation of health strategies. Hoeymans et al. [2014] argue that technological applications arise in prevention, treatment and care. Benefits range from improved diagnostic skills to regenerative medicine facilitating the independent living. For example, research enables more targeted prescription of medicines, and sensor technology enables instruments

3. Concerning the empirical counterpart of the reverse association (from health to income), we consider an improved version of the Mincer [1974]’s equation.

4. Figure 2.1 comes from the Survey of Health Ageing and Retirement in Europe, which contains five waves (each two years, from 2004 to 2015). See section 4.1 for further information.

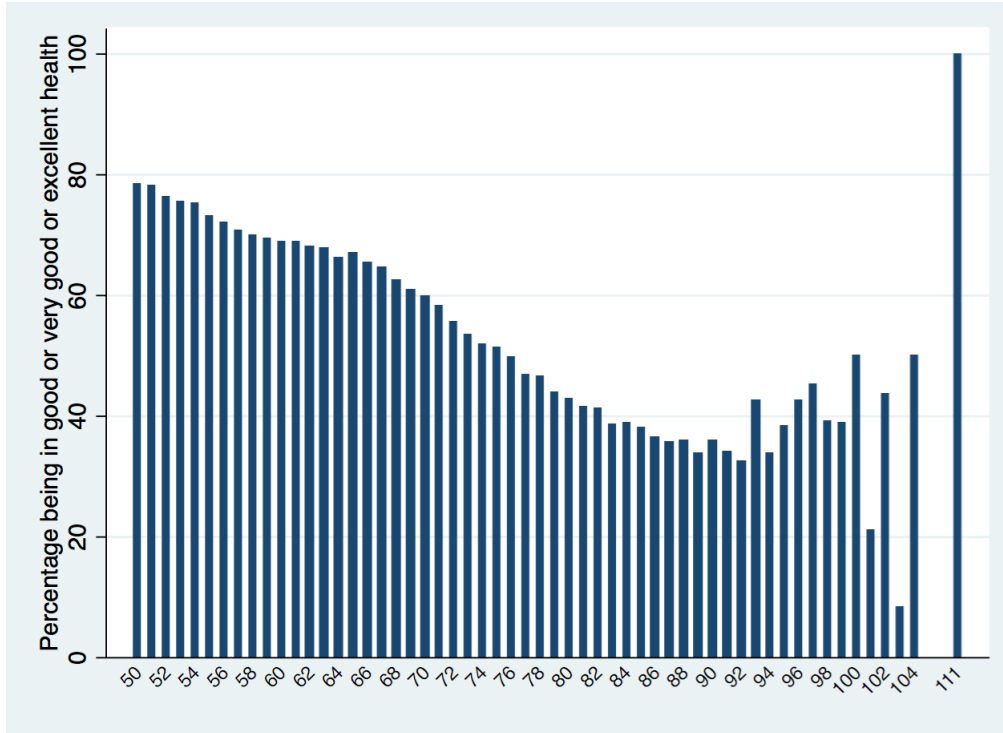


Figure 2.1 – Health of individuals by age

Source: SHARE

that monitor health status and home automation devices. As a result, one anticipates that self-perceived health status will increase across the board in the future, thanks to technological and societal trends allowing an improvement in medical care. Empirically, technological trends can be modeled in two ways: using longevity as a proxy (which is linked to the improvement of medicine); and using any variable which is homogeneous across individuals in a given year. Concerning the latter way to model technological trends, we suppose that everybody is affected in the same way by these trends.

One should notice that when talking about causality in social sciences, experimental studies might be useful. However, in this research we do not make use of these methods because we think that it refers to a different approach and thus story. Moreover, we do not have the means necessary to develop such methods. However, since we have access to a rich panel database, we can investigate causal links between income and health.

2.3 SHARE survey

All waves of the Survey of Health, Ageing and Retirement in Europe (SHARE), a multidisciplinary and cross-national panel database of micro data on health, socio-economic

status and social and family networks of more than 123,000 individuals aged 50 and over from many European countries and Israel, are used. The number of participants increased from 15 countries in wave five to 18 countries in wave six.⁵ The sixth wave releases on 2017, contains information on 67,346 individuals, and includes Croatia as a new country (Börsch-Supan [2017f]). As a result, the pooled database contains almost 250,000 observations, and individuals are present on average 2.1 years in the panel. We choose to focus on this survey since it has all the information needed to carry out this research.⁶

The health variable in our study is the binary transformation of self-perceived health status where individuals are asked to classify their health from “poor” to “excellent” (binary variable equals to 1 when individuals report being in good, very good and excellent health, see figure 2.2). Concerning, the variables of control, we use quadratic

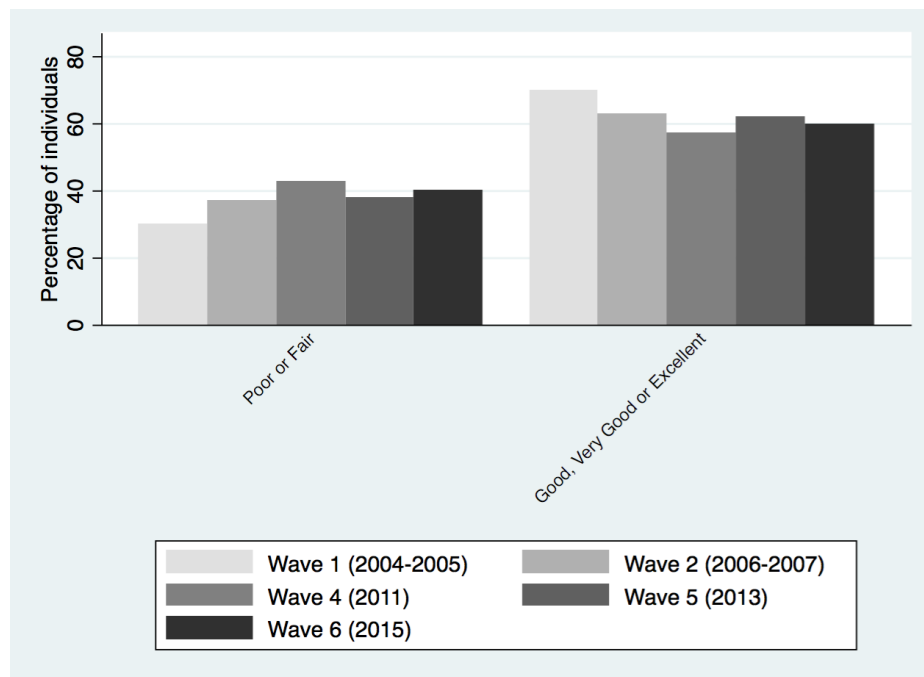


Figure 2.2 – Distribution of self-perceived health status in Europe

Source: SHARE

age, education (quantitative version following the International Standard Classification of Education), marital status, current job situation, and wave and country specific Gini

5. Groups of countries are created following the United Nations’ classification. Northern Europe corresponds to Denmark, Estonia and Sweden. Southern Europe corresponds to Croatia, Spain, Greece, Portugal, Slovenia, Israel and Italy. Western Europe corresponds to France, Germany, the Netherlands, Belgium, Luxembourg, Austria and Switzerland. Eastern Europe corresponds to Hungary, Poland and Czech Republic.

6. See subsection 1.2.2 for further information on this database.

coefficient⁷ to have more variability in this measurement (see table 2.2 in appendix part for further information). We also use dummies for groups of countries to capture regional effects.⁸ Then, technical progress is modeled using life expectancy at 65 years old.⁹ We distinguish women’s life expectancy from that of men in each country, in order to have the most accurate information. Technological progress can also be viewed as a variable which is homogeneous for all individuals for a given year. As a result, we also add time dummy variables to the specification. Since, life expectancy is not completely collinear to time dummy variables, both variables are added into the specification, in order to capture the real trend implied by the technical progress.

In this database, income corresponds to the sum of individual imputed income for all household components. We use the logarithm of income to reduce impacts of outliers. We would also like to know what are the changes in health status following positive income shock. Thanks to data availability, we follow the intuition first introduced by [Meer et al. \[2003\]](#) using information about the amount of unexpected gift or inheritance (worth 5,000€ or more). This information is included as a dummy variable. We are mindful that inheritance might not satisfy all exclusion restrictions such that this is not a very strong income shock (a family member dying might signal something about the individual’s health or other unobserved variables might drive both health and inheritance using the idea of “privileged backgrounds”).

It is important to measure health status in terms of non-fatal health outcomes since these are important for the burden of a disease. Morbidity indicators can be broadly defined by the prevalence or incidence of diseases, but also by the degree of disability and the risky behaviors of individuals, which can cause diseases. Morbidity is strongly correlated with self-perceived health status ([Manor et al. \[2001\]](#); [Latham and Peek \[2013\]](#); [Chan et al. \[2015\]](#)). As a result, it has to be taken into account when one studies self-perceived health status. [Dormont et al. \[2006\]](#) use a French microeconomic dataset (Santé Protection Sociale, conducted by IRDES) in order to construct morbidity indicators. We base our construction of indicators on their method, since they produce these indicators with the help of general practitioners who assure their validity. We focus on information about people having long-standing illnesses or health problems, and about activity limitation and disability, which assess self-perceived long-standing limitations in usual activities due to health problems. Thus, we use a vector of chronic illnesses and disability indicators

7. The Gini coefficient goes from 0 to 1, with 0 representing the situation of perfect equality where incomes in a population are distributed completely equally.

8. Dummies for countries are not included because of quasi-multicollinearity which can arise with the Gini coefficient.

9. Source: OECD website.

for morbidity. Indeed, a variety of lifestyle factors and health-related behaviors, such as alcohol consumption, physical activity and dietary habits, can affect a person's health. An unhealthy lifestyle often results in a higher risk of chronic diseases. SHARE database has the advantage of providing information about many morbidity indicators which can be divided into three main parts.¹⁰ The first part concerns the degree of invalidity of individuals and is represented using the following indicators: Activities of Daily Living (ADLs), Instrumental Activities of Daily Living (IADLs), the Global Activity Limitation Indicator (GALI) and an indicator about mobility limitation. The second indicator is about chronic diseases and gives the number of chronic diseases of an individual. Finally, the third category of morbidity indicators concerns risky behaviors of individuals.¹¹ We choose the alcohol consumption variable which informs on the drinking habits.

2.4 Econometric framework and results

2.4.1 Identification strategy

In order to assess the real impact of (i) income on health, and (ii) health on income, we focus on the concept of Granger causality, which takes into account the temporal dynamic of the relationships. The definition of causality by [Granger \[1969\]](#) distinguishes lag causality from instantaneous one. As a result, we investigate the causal impact of past income (*resp.* health) on current health status (*resp.* income). This approach includes the phenomenon of persistence of health status (*resp.* income) in the relationship. Self-perceived health status is a qualitative variable such that:

$$h_{it} = \begin{cases} 1 & \text{if } h_{it}^* > 0 \\ 0 & \text{otherwise} \end{cases}$$

in which h_{it}^* is a latent variable and implies a latent variable specification of the model. We intend to estimate the following equations simultaneously to highlight permanent causal links:

$$\begin{aligned} \forall i = 1 \dots N \quad \& \quad \forall t = 1 \dots T_i \\ \begin{cases} h_{it}^* = \alpha_0 + \lambda h_{i,t-1} + \delta linc_{i,t-1} + X_{it}\beta + cs_{jt}^1 \Gamma_1 + \epsilon_{it}^1 \\ linc_{it} = \beta_0 + \Lambda h_{i,t-1} + \Omega linc_{i,t-1} + Z_{it}\alpha + cs_{jt}^2 \Gamma_2 + \epsilon_{it}^2 \end{cases} \end{aligned} \quad (2.1)$$

10. See the appendix part in order to have detailed statistics and definitions on these indicators.

11. We do not include information about smoking since this variable contains a lot of missing information such that it would considerably reduce the number of observations. However, we did the entire method with the inclusion of this variable and find similar results. The results are not reported here but available upon request.

where T_i corresponds to the number of observations for an individual i ; h_{it}^* is a latent variable for which h_{it} equals to 1 when individual i reports being in good, very good or excellent health¹² at date t ; $\ln inc_{it}$ denotes the logarithm of income¹³ of individual i at date t ; cs_{jt} represents a set¹⁴ of specific information on a country j at date t ; X_{it} is a set of observed variables representing age, age squared, gender, marital status, a dummy for retired status, schooling and the morbidity indicators. To be sure of correctly assessing the true impact of income on health, we also add an exogenous income shock to the health equation (included in X_{it}). Then, Z_{it} corresponds to age, age squared, gender, education, marital status, dummies for groups of countries and the job statuses.

One limitation of this approach is due to the concept of causality chosen here which is the one of Granger. Indeed, Granger causality corresponds to a weak causality test which allows to solve simultaneity issues but not issues associated to a possible omitted variable bias. For instance, since we focus on Europe, we might think of different social security systems specific in all countries, but such information is difficult to access in the data. Thus, we include groups of countries to try to capture such biases. However, there might still be other missing components causing an omitted variables bias.

The error terms are assumed to be normally distributed and can be decomposed into two terms such that:

$$\begin{cases} \epsilon_{it}^1 = \eta_i^1 + \zeta_{it}^1 \\ \epsilon_{it}^2 = \eta_i^2 + \zeta_{it}^2 \end{cases} \quad (2.2)$$

We have the following variance-covariance matrix for individual effects :

$$\Sigma_\eta = \begin{pmatrix} \sigma_1^2 & \rho\sigma_1\sigma_2 \\ \rho\sigma_1\sigma_2 & \sigma_2^2 \end{pmatrix}$$

with $\rho = \text{corr}(\eta_i^1, \eta_i^2)$. Then, the variance-covariance matrix for idiosyncratic errors is given by:

$$\Sigma_\zeta = \begin{pmatrix} 1 & \rho_1\sigma \\ \rho_1\sigma & \sigma^2 \end{pmatrix}$$

12. This binary variable is derived from self-perceived health status. Individual reporting their health as being excellent, very good, or good are considered as “healthy” such that h_{it} equals 1; in contrast, individuals reporting their health as fair or poor are considered as “unhealthy” where h_{it} equals 0.

13. We use log transformation of income to reduce effects of outliers, as done by [Michaud and Van Soest \[2008\]](#) or [Halliday \[2017\]](#).

14. In the health equation, cs_{jt}^1 includes temporal dummies, life expectancy at 65 years old, a country-specific indicator of income inequalities (Gini coefficient) and dummies for groups of countries. In the income equation, cs_{jt}^2 includes temporal dummies, life expectancy at 65 years old and dummies for groups of countries.

where η and ζ are independent. Whether $\rho\sigma_1\sigma_2$ is statistically significant or not gives us insights on the necessity to conduct either bidirectional simultaneous analyses or univariate ones. A simultaneous equations approach allows to consider unobserved factors which may be common to both equations. For instance, these factors can correspond to the physical maturity (some individuals are “physically stronger” than others due to their genetics) or to intellectual capacity (some individuals have intellectual abilities). As a result, it is important to consider these correlations when considering the health-income relationship. Moreover, since we correctly consider heterogeneity into the fixed effects of both equations using a simultaneous equations model with a Full Information Maximum Likelihood (FIML) estimator, we do not need to specifically instrument health and income.

Concerning the likelihood function, [Roodman \[2011\]](#) specifically discusses conditions for consistency and identification in a simultaneous equations model. We consider a bivariate case with one binary outcome (health) and a linear one (income), such that we can introduce a notation:

$$q_{it}^1 = 2 * h_{it} - 1$$

in which h_{it} corresponds to the binary outcome, i.e., health. Thus we can first write the contribution of individual i to the likelihood function as:

$$\begin{aligned} L_i &= \int_{\mathbb{R}^2} \left\{ \prod_{t=1}^{T_i} f_{\zeta}(\zeta_{it}^1, \zeta_{it}^2 | \eta) \right\} f_{\eta}(\eta_i^1, \eta_i^2) d\eta_i^1 d\eta_i^2 \\ &= \int_{\mathbb{R}^2} \left\{ \prod_{t=1}^{T_i} \ell_{it} \right\} f_{\eta}(\eta_i^1, \eta_i^2) d\eta_i^1 d\eta_i^2 \end{aligned}$$

in which ℓ_{it} is:

$$\ell_{it} = \phi_1(\zeta_{it}^2, 0, \sigma^2) \Phi_1\left(\frac{q_{it}^1 x_{it} - \frac{\rho_1}{\sigma} \zeta_{it}^2}{\sqrt{1 - \rho_1^2}}\right)$$

in which $x_{it} = X_{it}\Gamma + \eta_i^1$ with X_{it} for the explanatory variables in the health equation. In this way, the likelihood function is a multidimensional integral such that we use the adaptive Gauss-Hermite quadrature method as an approximation (as proposed by [Liu and Pierce \[1994\]](#)).¹⁵

Simultaneous equations modeling is feasible only if both equations are identified which is

15. For accuracy of the method and to reduce computing time, we derive the gradient of the log-likelihood and the Hessian of the respective integrand. The estimation method has been implemented using the *d1* method of Stata software (see [Gould et al. \[2010\]](#) and the appendix at the end of the Ph.D. dissertation for further details).

the case here. Indeed, both equations contain two endogenous equations (i.e., $h_{i,t-1}$ and $linc_{i,t-1}$) such that at least two exogenous variables must be specified in each equation and must be different from one equation to another. On one hand, different morbidity indicators are estimated only in the health equation because they can be considered as important determinants of self-perceived health status. On the other hand, different job status are estimated in the income equation which is important when considering a Mincer equation.

In system 2.1, we consider the exogeneity of what we are calling, hereafter, the variables of control (i.e., X_{it} for the health equation, and Z_{it} , for the income equation):

$$E(X'_{it}.\epsilon^1_{it}) = 0 \quad \forall t$$

$$E(Z'_{it}.\epsilon^2_{it}) = 0 \quad \forall t$$

In particular, we consider the exogeneity of the morbidity indicators (in the health equation) and the job statuses (in the income equation) which correspond to our identifying variables for each endogenous ones in both equations. Indeed, changes in self-perceived health status might be due to changes in morbidity as explained in section 2.2. Moreover, job statuses correspond to the identifying variables in the income equation since they are important predictors of income for individuals aged 50 and over.

Then, concerning the other variables of control, schooling is important because a higher level allows an individual to have better access to health systems and jobs, and therefore one's subjective health and income should improve. Education shapes future occupational opportunities and earnings potential. Thus, it also provides knowledge that allows better educated persons to gain more access to information, which in turn promotes health and income. Grossman [1972a] and Mincer [1974] propose, in addition, that variables such as age and education influence the optimal levels of health and income. As a result, if one decides to control for age, then we should also control for education. Then, we are focusing on the health-income gradient such that we need to consider an indicator for income inequalities in a country since these play a role and have an impact on individual current health status (Adeline and Delattre [2017]).

Moreover, Granger causality involves a delayed causality of income on health in a manner that income creates disparities throughout time. Moreover, income affects health and might also affect other unobserved variables (such as lifestyle or food expenditures) which in turn might influence health status. In health economics literature concerning causality, due to endogeneity issues, the difficulty is to distinguish causes and effects. From an early stage in the debate, it was argued that higher income causes better health (Pre-

ston [1975]). Smith [1999] explains that this positive relationship leads to a number of interpretations: causality may go from income to health (high economic resources lead to better health status for many reasons such as: more resources devoted to health or better knowledge about what improves health), from health to income (poor health may restrict a family's capacity to earn income or to accumulate assets by limiting work or by raising medical expenses), or both may be determined by other common factors. For instance, η_i^1 and η_i^2 (system 2.2) might contain common factors to both h_{it}^* and $linc_{it}$, implying:

$$\begin{cases} E(\epsilon_{it}^1 | linc_{it}) \neq 0 \\ E(\epsilon_{it}^2 | h_{it}^*) \neq 0 \end{cases}$$

Similarly, Wooldridge [2010] brings two issues to the forefront which need to be taken into account in solving this endogeneity problem:

1. The issue of reverse-causality is a concern when one studies income-related health relationship: a positive income shock can lead to an improvement in health status through, for example, better access to medical services. However, we can also think of the reverse relationship where people in good health are likely to be more economically productive and thus have higher incomes.
2. Some individual characteristics which are not identified by the researcher may determine both income and self-assessed health status. A biased estimation between income and health results from a failure to control for these effects.

Finally, both equations in system 2.1 are auto-regressive forms, which are due to the data generating process underlying by the Granger causality. These auto-regressive forms imply biased estimates if we have:

$$\begin{aligned} E(h_{i,t-1}^* \cdot \epsilon_{it}^1) &\neq 0 & \forall t \\ E(linc_{i,t-1} \cdot \epsilon_{it}^2) &\neq 0 & \forall t \end{aligned}$$

As a result, these endogeneity issues further justify the use of a simultaneous equations model to correctly consider correlation in the error terms. Even if each identified equation can be estimated by two-stage least squares (2SLS), system estimation methods, such as Full Information Maximum Likelihood (FIML) are more efficient since they take into account the possible correlation of errors of all equations (ϵ_{it}^1 and ϵ_{it}^2) resulting from the simultaneous determination of health and income. Moreover, because we have a non linear equation and a linear one, 2SLS estimator does not adequately consider such specifications.

2.4.2 Results

In order to highlight Granger causal links, we include lagged variables for income and health in both equations. As a result, we lose observations due to these delayed variables, because all individuals are not always interviewed during the five waves of the panel.¹⁶ We thus estimate the health and income equations simultaneously with a Full Information Maximum Likelihood estimator, while correctly considering the panel structure of system 2.1 and correlations between the error terms.

Results in column (1) of table 2.1, corresponding to the health equation, display a strong phenomenon of persistence in health status. More specifically, when turning to the average marginal effect (AME, column (1') of table 2.1), it appears that moving from bad to good health at the previous period ($t - 1$) increases the probability of being in good health at date t by 6.7%. In other words, individuals in good health at the previous date have a higher propensity to be in good health today, compared to individuals who are in bad health at the previous date. Then, past income is positively related to the feelings of individuals concerning their current health. This result is significant and has the intuitive sign according to the literature, where it is said that a higher income is positively associated to health status. Thus, an increase in income in the past has a positive effect on current health status. Especially, looking at the average marginal effect of this variable, we can say that a 1% increase in income at the previous date implies a 0.012 percentage points increase in the probability of being in good health today (at date t), at the average point of the sample. Since the average probability of being in good health is 0.615 in our sample (see table 2.2 in the appendix section 2.A), then, following a 1% increase in income at the previous date, there is a 1.95% increase in the probability of being in good health today, compared to being in bad health, for each individual.¹⁷ Moreover, the latter result is also supported by the positive effect of the income shock (financial gift of 5,000€ or more), meaning that an expected amount of money has a positive effect on health. The associated average marginal effect is also positive and significant and further details that having received a financial gift increases the probability of being in good health by 0.3%. Concerning morbidity indicators, which represent the prevalence or incidence of a disease, results imply that being affected by a disease, or by limitations, is negatively correlated to self-rated health status. Individuals consider these effects when rating their

16. Thus, this analysis (system 2.1) gives access to 90,684 observations corresponding to almost 50,000 individuals. Indeed, in the panel we have 116,388 individuals, including 42,986 individuals who are present only once in the panel, 33,912 present twice, 25,955 present during three waves, 7,384 individuals are interviewed during four waves, and only 6,151 individuals are followed during the five waves.

17. Indeed, in our sample, the average probability of being in good health is 0.615. Thus, following a 1% increase in income at the previous date, the probability of being in good health today for an individual increases by $\frac{0.012}{0.615} = 0.0195$, that is to say, by 1.95%.

health. For technical progress, we include both life expectancy and cohort fixed effects.¹⁸ Individuals feel better when life expectancy increases. We include an indicator of income inequalities in a country (Gini coefficient) which is negatively related to current health status, meaning that when inequalities increase, health status is negatively affected. We also include dummies for groups of countries to capture specific country effects. These dummies are negatively related to health status when compared to individuals who live in Western Europe. Finally, we control for the retirement status in this equation with a dummy, but the latter does not have a significant effect on health. Average marginal effects for this equation are reported in column (1') of table 2.1 , and confirm the results such that there are considered as robust.

On the other hand, results in column (2) correspond to the income equation. Granger causality seems to be at play too since there is a strong phenomenon of persistence in income (a 1% increase in income at the previous date increases current income by 0.31%), and health has a positive and permanent impact on current individual income (switching from being in bad health to being in good health at the previous period implies an increase of 0.215% in income). This supports the idea that health might determine earnings on the labor market or that health might induce costs (such that being healthy means no costs). Technical progress also improves individual income whereas living in Eastern, Northern and Southern Europe decreases income when compared to countries of Western Europe. We control for marital status, and results suggest that never being married, being divorced or being widowed have a negative impact on income. Indeed, in these cases, there are no insurance effects between partners concerning income. Then, we also control for the job status of individuals because this study considers a population aged 50 and older, and results suggest that being employed compared to retired (reference category) has a positive impact on income. Indeed, incomes from employment are generally higher than other sources of income for individuals who do not work or are retired.

All the previous enumerated results are confirmed by the univariate analyses. In appendix 2.B, table 2.6 provides the results when the two equations of system 2.1 are estimated separately. These results are qualitatively similar to the ones provided in table 2.1 (bivariate analyses). Moreover, the average marginal effects concerning the health equation are smaller in the joint analysis than in the univariate one, such that, the use of a FIML estimator allows to have unbiased estimates. Thus, simultaneous analyses are useful when studying the health-income gradient. Concerning the average marginal effects of the income equation, results are quantitatively and qualitatively similar.

18. The first wave of SHARE is not included since the analysis has been performed using lagged variables

Finally, one important result is the correlation between the error terms which is statistically significant, meaning that one should consider these correlations when studying health-income gradient since simultaneity between health and income is at play. Thus, considering a simultaneous equation model is necessary in our case where we study causal links between health and income on elderly Europeans. In other words, there are unobserved factors common to both equations. Thus, thanks to this method, our results ensure the Granger causality of income on health on one hand, and of health on income on the other hand. These two relationships, which highlight permanent causal links and persistence in these relationships, should be considered simultaneously.

Table 2.1 – Results of the simultaneous equations model

Variables	Coefficients		
	(1)	(2)	(1')
Equation:	Health _t	Income _t	AME - Pr(Health _t =1)
<u>Granger causality</u>			
Health _{t-1}	0.814*** (0.015)	0.215*** (0.007)	0.067*** (0.001)
Log of income _{t-1}	0.132*** (0.006)	0.319*** (0.003)	0.012*** (0.0004)
<u>Exogenous income shocks</u>			
Financial gift (5000€ or more)	0.051* (0.029)		0.003* (0.002)
<u>Morbidity Indicators</u>			
ADL	-0.044*** (0.013)		-0.003*** (0.001)
IADL	-0.017* (0.009)		-0.001* (0.001)
GALI	-1.005*** (0.015)		-0.079*** (0.001)
Mobility indicator	-0.186*** (0.004)		-0.014*** (0.0004)
Chronic diseases	-0.235*** (0.006)		-0.016*** (0.0004)
Drinking	-0.048*** (0.016)		-0.003*** (0.001)
<u>Technical progress</u>			
Wave 2	0.061** (0.027)	0.307*** (0.011)	0.001 (0.002)
Wave 4	0.025 (0.021)	0.661*** (0.009)	-0.015*** (0.001)
Wave 5	0.031* (0.018)	0.579*** (0.007)	-0.001 (0.001)
Wave 6			
Life Expectancy	0.046*** (0.009)		0.003*** (0.001)
<u>Co-variables</u>			
Age/10	-0.682*** (0.110)	0.437*** (0.047)	-0.002** (0.001)
Age squared/100	0.046*** (0.008)	-0.028*** (0.003)	
Gender (=1 if women)	-0.058 (0.038)	-0.048*** (0.007)	-0.004* (0.002)
Gini	-1.032*** (0.138)		-0.066*** (0.009)
Education	0.086*** (0.005)	0.069*** (0.002)	0.007*** (0.004)
Married			
Living with partner	0.008 (0.058)	0.157*** (0.024)	0.0004 (0.004)
Living as a single	0.015 (0.032)	0.068*** (0.014)	0.001 (0.002)
Never married	-0.105*** (0.032)	-0.218*** (0.013)	-0.013*** (0.002)
Divorced	0.047* (0.026)	-0.174*** (0.011)	-0.001 (0.002)
Widowed	0.038* (0.021)	-0.211*** (0.009)	-0.003** (0.001)
Retired	0.024 (0.019)	Reference	0.002 (0.001)

Table 2.1 – Results of the simultaneous equations model (continued)

Variables	Coefficients		
	(1)	(2)	(1')
Equation:	Health _t	Income _t	AME - Pr(Health _t =1)
Employed		0.202*** (0.011)	0.001* (0.0004)
Unemployed		-0.285*** (0.022)	-0.009*** (0.001)
Permanently sick		-0.028 (0.018)	-0.0004 (0.0005)
Homemaker		-0.183*** (0.012)	-0.005*** (0.0006)
Other		-0.178*** (0.025)	-0.005*** (0.001)
Western Europe		<i>Reference</i>	
Eastern Europe	-0.118*** (0.038)	-1.034*** (0.011)	-0.075*** (0.002)
Northern Europe	-0.441*** (0.025)	-0.229*** (0.008)	-0.034*** (0.002)
Southern Europe	-0.032 (0.021)	-0.579*** (0.009)	-0.027*** (0.001)
Constant	1.681*** (0.438)	4.428*** (0.173)	
$\rho\sigma_1\sigma_2$: correlation		0.022*** (0.009)	
Numb. of obs.			90,684

***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are into parentheses, below the coefficients.

2.5 Conclusion

A heavily researched topic in health economics is the relationship between income and health and more specifically the direction of causality between the two. This chapter sheds light on the question of causal effects of health on socioeconomic status and vice versa, for elderly individuals in Europe. The main difference with earlier approaches is that our framework allows to control for unobserved heterogeneity and correlations in the error terms of both equations. All waves of the SHARE survey, which follows a statistically representative sample of European people aged 50 and older from 2004 to 2015, are used. While it seems well-known that people with higher incomes enjoy better health, it is far more difficult to establish the direction of the causality. The definition of causality chosen here is that of Granger which includes a persistence phenomenon in relationships, as well as permanent causal links thanks to lagged variables. We implement a simultaneous equations model to highlight bidirectional causal links. This enables to identify components of the health-income relationship and to control for endogeneity by considering a specific error terms' structure. The originality of this chapter is the simultaneous bivariate analysis settled, which, to the best of our knowledge, has not yet

been performed.

Since researchers need a clear understanding of the direction of the causality in this relationship, results presented here contribute to a central point in the analysis of health and income. Our dynamic method and results suggest that, on one hand, income has a permanent effect on subjective health status, and on the other hand, health has a permanent effect on income. Especially, individuals in good health at the previous date have a higher propensity to be in good health today, compared to individuals who are in bad health at the previous date. Moreover, switching from being in bad health to being in good health at the previous date implies an increase of 0.215% in income. Results also suggest that a 1% increase in income at the previous date implies a 1.95% increase in the probability of being in good health today (at date t), and an increase in the current income by 0.319%. More precisely, our results imply that simultaneity between income and health is at play such that it is essential to consider bidirectional analyses. Indeed, one should correctly tackle endogeneity since there might be unobserved components common to both equations. Moreover, our results are further reinforced when we compare them to the univariate analyses of health and income. Specifically, the univariate analysis of health seems to present biased estimates due to bigger average marginal effects than in the joint analysis (bivariate causal relationship).

This analysis is important for policy makers who want to reduce health inequalities in which income is shown to be an important lever. In this way, this study suggests that governments should jointly act on health and income. In order to reduce inequalities, policy makers should promote the access to complementary health insurance and facilitate the access to health care services. Thus, a virtuous circle might be set up since positive impacts of income have positive impacts on health, which, in turn, have positive effects on income. Finally, this is the first study analyzing health-income relationships, with a simultaneous equations model, using the SHARE database and establishing strong and permanent Granger causal links.

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2.A Appendix A: descriptive statistics

2.A.1 Variables of interest and control variables

Table 2.2 – Descriptive statistics of the variables of interests and some covariates

Variables	Mean	Std. dev.	Min.	Max.	Nb. of obs.
Self perceived health	2.833	1.083	0	5	248,966
Binary health =1 (%)	61.49				
Log of income	9.9695	1.328	-6.389	16.122	247,731
<u>Exogenous inc. shock:</u>	%				
Financial gift 5000€ or more	13.83				171,027
Age	66.637	10.108	50	111	248,966
Gini	0.392	0.069	0.273	0.772	248,966
<u>Job situation:</u>	%				246,123
Retired	55.62				
Employed	26.10				
Unemployed	2.80				
Permanently sick	3.47				
Homemaker	9.52				
Other	1.35				
Missing	1.14				
<u>Education:</u>	%				248,736
Without diploma	4.78				
Primary	19.97				
Lower secondary	18.17				
Upper secondary	35.90				
First Stage of tertiary	20.33				
Second stage of tertiary	0.75				
<u>Marital Status:</u>	%				246,510
Married living with spouse	68.18				
Married living single	7.03				
Registered partnership	2.04				
Never married	4.15				
Divorced	6.47				
Widowed	11.15				
Missing	0.98				

2.A.2 Morbidity indicators

As explained earlier, the morbidity indicators have been chosen following the methodology of [Dormont et al. \[2006\]](#). Our morbidity indicators are divided into three main parts corresponding to the indicators of the Minimum European Health Module (MEHM). The first category concerns the degree of invalidity of individuals and contains information on four health aspects. ADLs consist of “basic activities that are necessary to independent living (e.g. walking, bathing, dressing, toileting, brushing teeth and eating)”, according to the World Health Organization (WHO). This concept determines an individual’s ability to perform the activity with or without assistance. IADLs, according to the WHO, are “activities with aspects of cognitive and social functioning, including shopping, cooking, doing housework, managing money and medication, and using the telephone or the computer”. These tasks support an independent lifestyle. GALI belongs to the family of disability indicators, targeting situations in which health disorders and conditions have impacted people’s usual activities (number of limitations with mobility, arm function and fine motor skills). It is a single-item survey instrument where individuals are asked: “For at least the last 6 months, have you been limited because of a health problem in activities people usually do?” and they have to answer: “1) Yes, strongly limited, 2) Yes, limited, or 3) No, not limited”. Moreover, in SHARE, individuals are asked to give the number of their limitations concerning mobility (from 0 to 10). The second category of indicators corresponds to the chronic diseases, and gives the number of chronic diseases an individual suffers from (heart problem, high blood pressure/high blood cholesterol, stroke or cerebral vascular disease, diabetes, cancer...). Finally, we also consider the risky behavior with a drinking variable. The WHO recommendations for a reasonable consumption is a maximum of two glasses of alcohol per day.¹⁹

Table 2.3 – Detailed descriptive statistics on morbidity indicators

Variables	Mean	Std. dev.	Min.	Max.	Nb. of obs.
ADLs	0.257	0.882	0	6	248,966
IADLs	0.407	1.243	0	9	248,966
GALI	0.462	0.499	0	1	248,966
Mobility	1.657	2.371	0	10	248,679
Chronic diseases	1.746	1.572	0	14	248,653
Drinking	0.289	0.453	0	1	248,035

19. However, the WHO also states to abstain from alcohol at least one day in the week, and not to consume more than four drinks on an one-time opportunity.

2.A.3 Technical progress

Table 2.4 – Detailed descriptive statistics on life expectancy at 65 years old for all waves and individuals (females and males) from OECD data

Country	Mean	Std. dev.	Min.	Max.	Nb. of obs.
Austria	20.003	1.689	17.3	21.7	15,344
Germany	18.622	2.683	11.9	21.2	16,954
Sweden	19.868	1.459	17.4	21.5	16,033
Netherlands	19.289	1.776	16.3	21.2	12,306
Spain	20.943	2.158	17.2	23.4	19,880
Italy	20.436	1.879	17.3	22.6	18,365
France	21.374	2.258	17.7	23.8	19,757
Denmark	18.769	1.538	15.9	20.7	14,091
Greece	19.379	1.546	16.9	21.3	10,449
Switzerland	20.745	1.547	18.2	22.6	11,767
Belgium	19.605	1.842	16.5	21.6	23,173
Israel	20.303	1.092	18.7	21.3	6,685
Czech Republic	17.674	1.803	14.3	19.3	18,453
Poland	17.608	2.226	14.5	20.1	5,918
Luxembourg	20.531	1.422	18.9	21.9	3,138
Hungary	16.547	1.985	14.3	18.3	2,974
Portugal	19.973	1.867	17.8	21.7	3,586
Slovenia	19.569	2.009	16.9	21.4	9,723
Estonia	18.217	2.573	14.3	20.7	17,923
Croatia	18.414	2.884	15.2	21	2,447
Total	19.565	2.276	11.9	23.8	248,966

2.A.4 Exogenous Shock

Table 2.5 – Detailed descriptive statistics on the exogenous shock of income per country

Country	Gift 5,000€ or more		
	Yes (%)	No (%)	Nb. of obs.
Austria	11.38	88.62	11,062
Germany	17.51	82.49	11,382
Sweden	22.74	77.26	11,374
Netherlands	17.50	82.50	8,533
Spain	7.72	92.28	13,023
Italy	8.31	91.69	12,158
France	11.72	88.28	13,775
Denmark	21.56	78.44	9,818
Greece	14.68	85.32	7,185
Switzerland	19.73	80.27	8,466
Belgium	21.13	78.87	12,928
Israel	4.83	95.17	3,955
Czech Republic	9.73	90.27	12,560
Poland	8.41	91.57	4,008
Luxembourg	19.73	80.27	2,347
Hungary	15.32	84.68	1,952
Portugal	9.44	90.56	2,256
Slovenia	10.06	89.94	6,931
Estonia	6.12	93.88	12,262
Croatia	13.66	86.34	1,588
Total	13.83	86.17	171,027

2.B Appendix B: univariate analyses

Table 2.6 – Results of univariate models

Variables	Coefficients		
	(1)	(2)	(1')
Equation:	Health _t Probit	Income _t OLS	AME - Pr(Health ₁ =1)
<u>Granger causality</u>			
Health _{t-1}	0.872*** (0.012)	0.217*** (0.007)	0.321*** (0.005)
Log of income _{t-1}	0.112*** (0.005)	0.299*** (0.003)	0.041*** (0.002)
<u>Exogenous income shocks</u>			
Financial gift (5000€ or more)	0.047** (0.023)		0.017** (0.000)
<u>Morbidity Indicators</u>			
ADL	-0.029*** (0.011)		-0.011*** (0.004)
IADL	-0.011 (0.007)		-0.004 (0.003)
GALI	-0.776*** (0.013)		-0.285*** (0.043)
Mobility indicator	-0.135*** (0.004)		-0.049*** (0.001)
Chronic diseases	-0.174*** (0.004)		-0.064*** (0.002)
Drinking	-0.037*** (0.013)		-0.014*** (0.005)
<u>Technical progress</u>			
Wave 2	0.032 (0.021)	0.295*** (0.012)	0.012 (0.008)
Wave 4	0.019 (0.018)	0.655*** (0.009)	0.007 (0.006)
Wave 5	0.023 (0.015)	0.577*** (0.007)	0.008 (0.005)
Wave 6		<i>Reference</i>	
Life Expectancy	0.018** (0.007)		0.007** (0.003)
<u>Co-variables</u>			
Age/10	-0.573*** (0.083)	0.420*** (0.051)	-0.211*** (0.030)
Age squared/100	0.039*** (0.006)	-0.027*** (0.004)	
Gender (=1 if women)	0.019 (0.028)	-0.049*** (0.007)	0.007 (0.010)
Gini	-0.817*** (0.107)		-0.301*** (0.039)
Education	0.066*** (0.004)	0.072*** (0.002)	0.024*** (0.001)
Married		<i>Reference</i>	
Living with partner	0.011 (0.044)	0.161*** (0.027)	0.004 (0.016)
Living as a single	0.016 (0.025)	0.075*** (0.015)	0.006 (0.009)
Never married	-0.023*** (0.066)	-0.228*** (0.015)	-0.024*** (0.008)

Table 2.6 – Results of univariate models (continued)

Variables	Coefficients		
	(1) Health _t Probit	(2) Income _t OLS	(1') AME - Pr(Health ₁ =1)
Equation:			
Divorced	0.051*** (0.019)	−0.185*** (0.012)	0.019*** (0.007)
Widowed	0.042*** (0.016)	−0.219*** (0.009)	0.016*** (0.006)
Retired	0.017 (0.015)	<i>Reference</i>	0.006 (0.005)
Employed		0.203*** (0.011)	
Unemployed		−0.286*** (0.024)	
Permanently sick		−0.034* (0.019)	
Homemaker		−0.183*** (0.013)	
Other		−0.179*** (0.026)	
Western Europe		<i>Reference</i>	
Eastern Europe	−0.102*** (0.028)	−1.052*** (0.012)	−0.037*** (0.010)
Northern Europe	−0.344*** (0.018)	−0.225*** (0.009)	−0.127*** (0.007)
Southern Europe	−0.007 (0.016)	−0.587*** (0.009)	−0.003 (0.006)
Constant	1.514*** (0.328)	4.686*** (0.186)	
Numb. of obs.		90,684	
***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are into parentheses below coefficients.			

Chapter 3

Smoking habits, time preferences
and risk preferences: a comparative
study over European elderly people

Abstract

Higher rates of smoking increase health risks to smokers, and also to non-smokers who are at risk through the effects of passive smoking. This chapter uses two waves of the Survey of Health, Ageing and Retirement in Europe to examine the links between heterogeneity in time and risk preferences, income and cigarette consumption while taking into account endogeneity issues due to simultaneity biases from income and risk preferences, with smoking. Using a Full Information Maximum Likelihood estimator to estimate a simultaneous equations model with three equations (smoking probability, risk preferences and income), results suggest that, for elderly Europeans, (i) income has a negative and significant impact on the probability to smoke, such that the latter might be considered as an inferior good (however, smokers are richer than non-smokers on average); (ii) future-oriented people are less likely to smoke because they consider the harmful effects of smoking on health; and (iii) risk averse individuals, depending on where they live, are either more likely to smoke, which might be the result of an anxiety pathway, or, less likely to smoke, such that they do not want to take the risk to alter their health.

Keywords: Smoking; future-oriented; risk aversion; simultaneity; FIML.

JEL Classification: C33; C59; I12; I14; J14.

3.1 Introduction

According to the World Health Organization, tobacco consumption is the most significant cause of premature death in the European Union (EU), responsible for nearly 700,000 deaths every year.¹ Smoking prevalence is an important component of the difference in death rates between countries. Higher rates of smoking increase health risks to smokers, and also to non-smokers who are at risk through the effects of passive smoking. According to Eurostat, in 2014, 5.9% of Europeans aged 15 and over smoked at least 20 cigarettes per day, and around 12.6% consumed less than 20.² Among this population, the proportion of daily smokers ranged from 8.7% in Sweden to 27.3% in Bulgaria. The proportion of men daily smokers ranged from 7.5% in Sweden to 37.3% in Cyprus, while among women, the proportion ranged from 8.3% in Romania to 22% in Austria. As a result, governments need appropriate public policies to curb individuals from smoking and to inform people concerning health damages which result from smoking.

Smoking is considered as a costly addiction in the sense that it creates a dependence which is a cause and a consequence of consumption. The theory of rational addiction, developed by [Becker and Murphy \[1988\]](#), gives insights about the factors explaining the demand of addictive goods. Many factors can have an impact on the costs and benefits of smoking, and more precisely, individual heterogeneity and income. Knowing individual's time preferences shed light on who should be targeted by anti-tobacco public policies. Indeed, in economics we can identify two types of individuals. Present-oriented people do not take into account detrimental effects of smoking on health. On the other hand, future-oriented individuals integrate future health consequences of smoking when they choose to smoke. The impact of time preferences on smoking prevalence has been study ([Carbone et al. \[2005\]](#); [Arcidiacono et al. \[2007\]](#); [Van Der Pol \[2011\]](#) and [Balía \[2014\]](#)).³ Moreover, an important question is on whether individuals know the risks and on whether they make decision to smoke in light of this information. The fact that individuals are cognizant of the risks is one of the key element of the rationality of choices. As a result, the knowledge on risk preferences for individuals is an essential component of the assessment of the rationality of smoking decisions. Economic research on the relationship between risk and consumption of addictive goods began with [Viscusi \[1990\]](#) on US data. Focusing on risk

1. "Compared to the rest of the world, the European Union (EU) has one of the highest percentages of tobacco-related deaths" (WHO).

2. Article available online at http://ec.europa.eu/eurostat/statistics-explained/index.php/Tobacco_consumption_statistics. Source: European Health Interview Survey (EHIS) database.

3. However, the majority of study only focus on this particular heterogeneity, and rarely consider other aspects of preferences and tastes.

perceptions⁴, he finds that individuals overestimate the risks of lung cancer. Then, most recent works also focus on this subject, mainly on US data or on specific countries (Lundborg and Andersson [2008]; and Jusot and Khlal [2013]). In the literature, it is not clear whether risk preferences are a determinant of smoking (Gerking and Khaddaria [2012])⁵, or whether it is the smoking status which determines risk attitudes (Lundborg and Lindgren [2004]). Thus simultaneity issues between the two might be at play. Furthermore, smoking is a costly and dangerous habit for health, thus income might play a role in the consumption of this good, since it corresponds to the individual budget constraint. There might be an income endogeneity issue due to simultaneity between smoking and the latter. There are many possible pathways through which earnings can impact consumption of cigarettes. Smoking prevalence and the cumulative amount of tobacco differ across occupations and sectors, and thus across income. In the literature, other authors focus on the impact of income on the consumption of addictive goods (Marsh and McKay [1994]; Busch et al. [2004]; Pfeifer [2012] and Balia [2014]) and find varied results. However, we can also think of the reverse pathway in which smoking prevalence determines income. Indeed, economic costs of smoking reflect income reductions associated with smoking on the labor market (Auld [2005] and Lokshin and Beegle [2006]).

This work aims to assess risk and time preferences of smokers underlying the reasons for income differences in smoking behavior since these are not well understood. Previous studies only linked either two of the previous aspects and focus on the US or on particular European countries and find varied results. This chapter tries to address this gap in the literature by focusing on these three aspects. We thus investigate simultaneity biases between smoking and income on one hand, and smoking and risk preferences on the other hand, in Europe, using a simultaneous equations model. We choose to study the smoking decision on individuals aged 50 and older. This population is of particular interest since smoking has been proved to be one of the only modifiable factor to reduce the prevalence of dementia among ageing people, for whom smoking is a cause (WHO [2014]). This is an important issue for policy makers whose aim is to limit health damages due to smoking, and thus reduce health inequalities, maybe by increasing public awareness of the risk of tobacco-caused dementia among Europeans aged 50 and over.

This analysis confronts on an empirical basis the impact of individual heterogeneity in time and risk preferences on smoking prevalence using qualitative variable revealing individual heterogeneity (survey questions on risk and time horizon of planning); and, is the

4. Risk perceptions correspond to a subjective judgment makes by individuals and these are easier to access in databases than risk preferences items which correspond to attitudes in terms of risk aversion.

5. Specifically little is known on the impact of risk preferences on smoking since the majority of empirical studies focus on risk perceptions.

first econometric study on European individual data, to the best of our knowledge. We use two waves of the Survey of Health, Ageing, and Retirement in Europe to account for heterogeneity in preferences. In our simultaneous equations model approach, estimated with a Full Information Maximum Likelihood estimator, income has a negative impact on the probability to smoke, such that tobacco might be considered as an inferior good for individuals aged 50 and over, but among these individuals, the ones who smoke are, on average, richer than the ones who do not. Then, present-oriented individuals do not consider the harmful effects of smoking on health, such that they are more likely to smoke. Finally, concerning the impact of risk aversion on the probability to smoke, results depend on where individuals aged 50 and over live in Europe.

The chapter proceeds as follows. In section 3.2, we bring to the forefront the literature review concerning smoking, by making explicit the consumption of tobacco in Europe, as well as individual heterogeneity in the smoking decision. Section 3.3 describes the database. Then, in section 3.4 we detail our econometric framework. Section 3.5 reports on the results of the empirical analysis. Section 3.6 concludes this research.

3.2 Literature review

3.2.1 Tobacco consumption in Europe

Tobacco regulatory environment

Health inequalities are preventable differences in health outcomes between different population groups. Reducing health inequalities remains a key goal of public policy. Because smoking is so harmful, differences in smoking prevalence across population translate into major differences in death rates and illnesses. Prevention on the risks of smoking began in Europe in 1954 when the British Ministry of Health has publicly circulated information of lung cancer risks from tobacco consumption. Thus, in the early sixties, individuals are informed of the carcinogenic effects of this addictive substance by the World Health Organization and the Surgeon General of the United States.⁶ From that moment on, in developed countries, tobacco control policies are settled. Taking the example of France, the government engages in tobacco control in the seventies with the Veil law (see [Etilé \[2004\]](#)). In 1976, this law was the first French one against smoking in which the major steps were to restrict tobacco advertising, the first smoking bans, the affixing of

6. The Surgeon General of the United States is the operational head of the US Public Health Service Commissioned Corps (PHSCC).

health warnings on packages of tobacco products and tax increases.⁷ Since then, the European Union has adopted a series of measures for tobacco control such as legislation, recommendations and information campaigns. In 2003, the first treaty of the WHO about the detrimental effects of smoking is settled. Indeed, the WHO Framework Convention on Tobacco Control is implemented and developed in response to the globalization of the tobacco epidemic. Its aim is “to protect present and future generations from the devastating health, social, environmental and economic consequences of tobacco consumption and exposure to tobacco smoke”. These measures include: the regulation of tobacco products on the EU market (packaging, labeling, ingredients); advertising restrictions for tobacco products; the creation of smoke-free environments; tax measures and actions against illegal trade; and, anti-smoking campaigns. Smoking consumption is a real issue for public policies since worldwide, tobacco use causes nearly six million deaths per year (see figure 3.1). According to the WHO, in order to reduce demand for cigarettes, the option that works best consists in an increase in tobacco prices by applying consumption taxes. They further explain that following a 10% price increase on a cigarette pack, there should be a 4% drop in demand in high-income countries and 4-8% in low-income countries. More specifically, article 6 of the WHO Framework Convention on Tobacco Control highlights the “financial and tax measures to reduce the demand for tobacco” and, states the importance of this policy to encourage governments to follow this taxation and prices policy. For instance concerning France, the French Observatory for Drugs and Drug Addiction (Observatoire Français des Drogues et Toxicomanies, OFDT in French) in their 2017-report⁸ explains that, in the 2000-2016 period, the price of a cigarette pack almost doubles which reduces the sales of the latter by two (see figure 3.2). Increase in prices mainly comes from an increase in taxes, such that taxation is a tool used to control tobacco consumption, which has a uniform impact regardless the individual characteristics. In the EU, tobacco prices are decomposed between tobacco taxation and the prices paid by retailers and manufacturers (see figure 3.3). Tobacco taxation is composed of a Value Added Tax (VAT), and two other country-specific taxes called consumer rights: (i) a specific or fixed duty applied to a quantity or a weight of tobacco, and (ii) a proportional or ad-valorem excise which represents a certain percentage of the selling price of the product.

7. After the Evin law (1991), governments took the tobacco out of the calculation of the “official” price index in order to make tax increases without preventing consideration of the Maastricht convergence criteria.

8. “Tobacco addiction and stopping smoking”, or Tabagisme et arrêt du tabac en 2016, in French, by Aurélie Lermenier-Jeannet from OFDT.

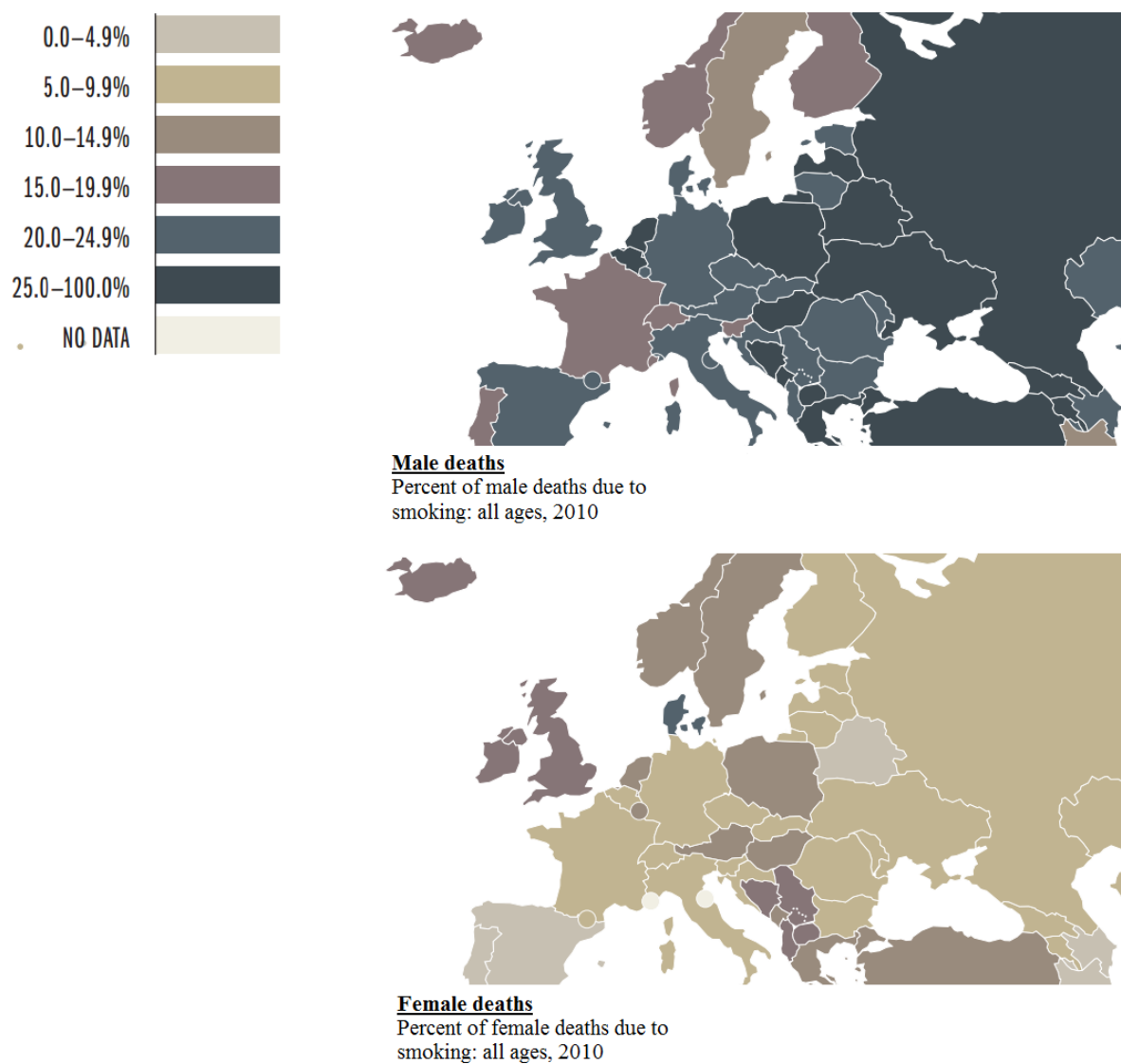


Figure 3.1 – Smoking-related mortality data, 2010

Source: Institute for Health Metrics and Evaluation (IHME) - University of Washington.

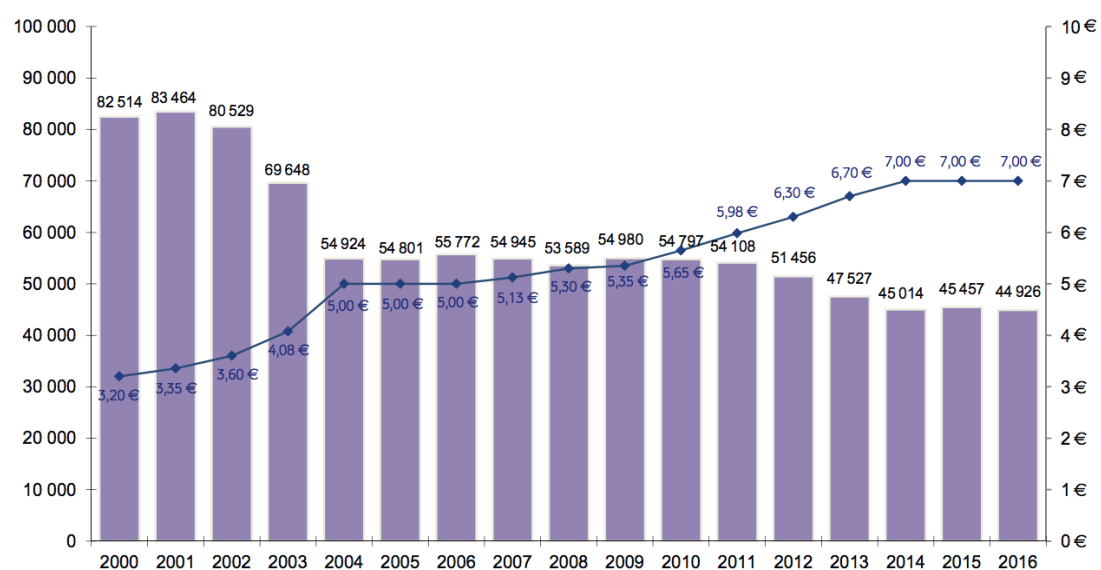


Figure 3.2 – Cigarette sales (millions of units) and average annual price of the best-selling cigarette pack in France

Source: French General Directorate of Customs and Excise (Direction Générale des Douanes et Droits Indirects, DGDDI in French).

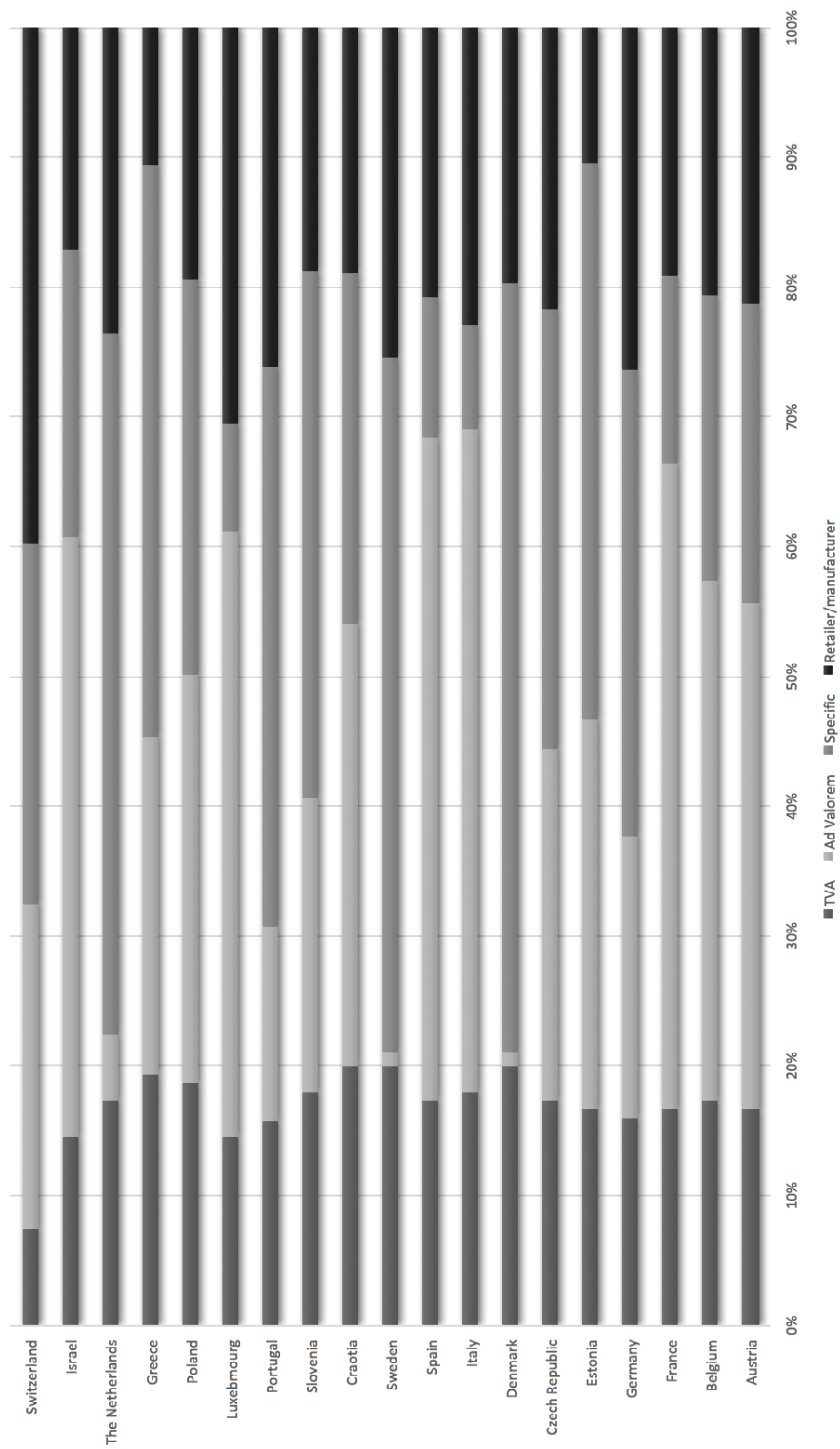


Figure 3.3 – Decomposition of tobacco prices in EU in 2018

Source: European Commission

Tobacco among ageing people

Studying smoking among older people is interesting per se. Indeed, the European Commission has identified the need for scientific knowledge about ageing people in Europe. In fact, people of the European Innovation Partnership on Active and Health Ageing project estimate that in 2050, one in three Europeans will be over 60 years old and one in ten will be over 85 years old; this is why the European Innovation Partnership on Active and Healthy Ageing - A Europe 2020, initiative has been implemented.⁹ The goal is to settle public policies and action across the different governments to: “significantly improve the health and well-being of populations, reduce health inequalities, strengthen public health and ensure people-centered health systems that are universal, equitable, sustainable and of high quality”. Moreover, some evidence suggest that tobacco consumption causes dementia, whose prevalence increases with age and is defined as the leading cause of dependency and disability among older people (Almeida et al. [2002]; Anstey et al. [2007]; WHO [2014]). Dementia is an important issue for individual health but also for its economical costs due to care needed (the average annual global cost is estimated around US \$17,000). According to WHO and ADI [2012], over the next 40 years, the prevalence of this disease will increase due to the ageing global population pattern. Thus, researchers need to focus on modifiable risk factors, like tobacco consumption, in order to reduce the occurrence of the disease and lessen the burden among ageing people, until effective treatments are identified. Finally, heterogeneity among an ageing population is also interesting. For instance, concerning time preferences, Chao et al. [2009] explain that in theory, bad health outcomes, related to impatience, could be prevented, if future is emphasized. Indeed, mortality and morbidity can be reduced through investments in health or through public health interventions, whereas age is not changeable. Due to the prevalence of diseases at older age, individual have to make choices in which time preferences play a crucial role (Huffman et al. [2017]).

3.2.2 Heterogeneity in smoking decision

Becker and Murphy [1988]’s model of rational addiction assumes that smokers are subject to a constraint due to habits formation (addictive effect). Current satisfaction from smoking is reinforced by past consumption but the greater was past consumption, the lower is current utility (such that past smoking implies a current negative marginal utility). In such a framework, smoking depends on the following structural components: past (and future) consumption, current (and future) prices, and income.¹⁰ This model relies on a questionable perfect information hypothesis. As a result, Orphanides and

9. See <http://ec.europa.eu/> for further details.

10. Empirically, this implies to include socio-demographic explanatory variables and an error term.

[Zervos \[1995\]](#) extend the rational addiction theory by considering that an individual is uncertain about negative health consequences of smoking consumption. Such uncertainty justifies the major role of identifying individual heterogeneity. The latter explains part of risky behaviors which contribute to diseases and social inequalities, and constitute a public health issue. When designing this heterogeneity, we focus on time and risk preferences because identifying smokers personal tastes allow policy makers to target population for smoking prevention.¹¹ In the literature, it is difficult to identify these preferences. Indeed, empirically, time and risk preferences are hard to quantify and proxies are rarely available in databases. As a result, there is not a clear understanding about the impact of time and risk preferences, and research papers mainly result in calibration models (where these parameters are computed according to theory) or experimental studies. In this research, we examine smoking consumption as an economic choice which depends on individual preferences (time and risk preferences) and a budget constraint, represented by income. Indeed, when individuals smoke, they favor their satisfaction. However, this choice induces costs due to the harmful effects on health and the expansive characteristics of this addiction. In this way, time preferences are related to whether individuals promote their health in the future, and risk preferences are associated to hazards on future health (health depreciation).

Time preferences

One element of personal tastes is time preference which corresponds to the preference for immediate satisfaction over a delayed one. This concept involves a compromise between present satisfaction and future losses in health capital due to smoking. Health-related behaviors correspond to an inter-temporal decision making, in which individuals allocate their resources to maximize their discounted utility. The literature differentiates two theories of economic models of addiction: rational addiction and myopic addiction ([Chaloupka and Warner \[2000\]](#)). Smokers defined as myopic people care more about present satisfaction than about future one. In this theory, tobacco uptake improves individual's utility directly, such that the negative impacts on future health are not considered. Myopic people corresponds to a property in behavioral economics' models of time discounting named the hyperbolic discounting ([Gruber and Koszegi \[2001\]](#); [Chabris et al. \[2008\]](#)). A smaller discount rate is applied to future choices compared to immediate choices by hyperbolic discounters. The second theory is the rational addiction one in which individuals are forward-looking smokers. [Becker and Murphy \[1988\]](#)'s model¹² is the starting point of the literature review on rational addiction. Forward-looking smokers

11. [Grignon \[2009\]](#) explains that smokers are heterogeneous such that current taxes on cigarettes might be an inefficient instrument of public health.

12. They built their analysis on the model of rational addiction introduced by [Stigler and Becker \[1977\]](#).

take into account the future consequences of smoking on health. In this model, smokers assimilate the detrimental effects of cigarettes in their utility. At any time, the stock of past addictive consumption is a component of the utility. Consequently, according to both theories, myopic smokers are more likely addicts than forward-looking individuals who smoke if the benefits from smoking are larger than the expected costs. In this context, the basis of the analysis is individual decision-making, taking into account the opportunity costs associated to the smoking decision. When they do so, individuals have to choose between an immediate but small reward and a larger delayed reward. Whether the health consequences of smoking are not too large influences the decision to smoke through the discount factor ([Adda and Lechene \[2013\]](#)).¹³ In the literature, some authors focus on the impact of time preferences on smoking prevalence. [Carbone et al. \[2005\]](#) base their analysis on the calibration of an expected lifetime utility and show that individuals are rational concerning their smoking behavior and the effects of smoking on their future health. [Arcidiacono et al. \[2007\]](#) investigate which model (i.e., rational or myopic model) better explains smoking (and drinking) behavior among the elderly using the Health and Retirement Study. They show that the myopic model is just a particular case of the rational model and the latter fits the data better than the former. [Adams \[2009\]](#) studies the impact of time preferences in the smoking cessation of older individuals using the English Longitudinal Survey of Ageing (ELSA). He finds that, future-oriented people are more inclined to quit smoking. [Balía \[2014\]](#), using the Survey of Health, Ageing and Retirement in Europe, identifies the effects of smoking on survival expectations with a finite mixture model. Results highlight two types of individuals. On one hand, some individuals do not assess neither the short nor the long-term effects of smoking. On the other hand, for some individuals, the short-term effects of smoking are considered while the long-term effects are not.

Risk preferences

Smoking is a risky choice and is harmful for health, implying diseases and illnesses which can cause premature death. Risk associated to smoking comes from the depreciation of health when an individual smokes (i.e., health reduction due to illnesses related to smoking). Indeed, health status of a smoker is smaller than the one of a non-smoker. In the economic theory, [Arrow \[1974\]](#) highlights the choice made by an individual between an option with a certain pay-off (risk averse) over another one with an uncertain pay-off but possibly higher (risk lover). Following this intuition, no tobacco health risks should be considered by risk lovers. On the other hand, health risks from smoking should be

13. According to economic theory, a discount factor closed to 1 corresponds to a more patient individual, such that the latter does not engage in smoking.

considered as high enough for risk averse individuals who might consider smoking-related illnesses such as cancers. Risk averse individuals might not want to take the risk of altering their health by smoking. In other words, risk lovers might be more likely to smoke and might consume more cigarettes than risk averse individuals. In the majority of studies, researchers do experimental economics in order to explicit risk preferences, making the representativeness of the sample questionable. Empirically, authors focus on risk perceptions because risk aversion items are difficult to access in databases.¹⁴ Risk perceptions can be defined as the subjective judgment an individual makes when he is asked to characterize and evaluate risky activities. Using risk perceptions proxies, which are easier to access, [Viscusi \[1990\]](#) investigates the latter among smokers using as a risk measure the lung cancer risk perception because of smoking. Results of the logit model show that the probability of lung cancer because of smoking is overestimated by individuals. [Viscusi and Hakes \[2008\]](#) find that higher risk beliefs decrease the probability of starting to smoke. [Gerking and Khaddaria \[2012\]](#) use [Viscusi \[1990\]](#)'s risk measure to investigate the probability of smoking and find that this deters smoking among individuals aged 14-22 years. Concerning risk preferences, only one study investigates their impacts on smoking status. [Pfeifer \[2012\]](#) uses the German Socio-Economic Panel (GSEP) and questions about the willingness to take risks in general and another one concerning risks in health, as measures of risks. Using both a two-part model as well as a tobit model, evidence shows a higher demand for cigarettes for risk lovers who are thus more likely to smoke.

From this literature, it emerges that there is a strong correlation between risk perceptions and smoking. Indeed, in other studies ([Feinberg \[1977\]](#), [Paranda-Contzen \[2017\]](#)), smoking is used as a proxy for risk preferences in cases where direct measures of risk are not available. Moreover, some authors investigate the reverse relationship in which smoking has an impact on risk perceptions. [Viscusi \[1991\]](#) investigates the relationship between smoking and lung cancer risk perception because of smoking. He implements both a smoking equation, as well as, a risk perception equation. He finds that risk perceptions are higher at younger age and are negatively correlated with smoking. Risk perceptions, however, have no effect for younger people concerning their decision to smoke. [Lundborg and Lindgren \[2004\]](#), using cross-sectional survey data of Swedish teenagers, investigate the impact of smoking on risk perceptions and the reverse association, working with the same risk measure as [Viscusi \[1990\]](#). By estimating both a risk perceptions equation and a smoker equation, they find that risk perceptions from lung cancer is overestimated by non-smokers and smokers, the latter being more likely to have higher risk perceptions.

14. When we consider a simple example of an expected utility for a lottery: $EU = pU(x_1) + (1-p)U(x_2)$, then risk aversion is identified through $U(x)$ in which an individual faces an uncertain amount, whereas risk perceptions is identified through p , which is the variation in the probability.

Lastly, [Anderson and Mellor \[2008\]](#) investigate the impact of smoking on risk aversion and find that the former has a negative impact on the latter. Thus, smoking and risk might simultaneously determine each other. In this study, we focus on risk preferences to give some insights about their impacts since there is only a small literature on this subject. Moreover, smoking and either risk preferences or risk perceptions seem to be linked such that both might simultaneously determine each other. An econometric specification for risk preferences is needed when investigating heterogeneity and smoking.

Other studies investigate both risk and time preferences. Recent studies find that when eliciting time discount rates, one should consider risk and time preferences jointly ([Andersen et al. \[2008\]](#); [Tanaka et al. \[2010\]](#)).¹⁵ [Ida and Goto \[2009\]](#) survey Japanese adults and simultaneously measure these preferences, using choices of the respondents between two alternatives composed of rewards, probabilities and time delays. They apply a mixed logit model and find that smokers are more impatient and risk lover than non-smokers. [Harrison et al. \[2010\]](#) elicit measures of individual discount rates using the expected utility theory with constant relative risk aversion to identify both risk and time preferences. On a sample of the Danish population (268 individuals), male smokers have higher discount rates than male non-smokers. [Van Der Pol \[2011\]](#) investigates the impact of time and risk preferences in the education-health gradient. The Dutch DNB Household Survey incorporates stated preference questions (2,300 individuals, and six closed-ended intertemporal choices for time preferences). By assuming the discounted utility model and a linear utility function, risk lover individuals tend to be in poorer health. She also points out to a negative relationship between time preferences and health. [Jusot and Khlal \[2013\]](#)'s paper is about the impacts of heterogeneity in time and risk preferences in the educational gradient in smoking. They use the 2008-wave of the French National Health Care and Insurance Survey ("Enquête sur la Santé et la Protection Sociale" in French) which includes subjective scales to measure time and risk preferences. Two-step logistic regression models are used to test the different associations and they find that smokers are more impatient and risk lover than non-smokers.

Income

Another dimension in smoking is the income since smoking is considered as an economic choice. Different theories have been proposed to explain the correlation between income and smoking because both might simultaneously determine each other (smoking is a costly addiction but smoking might also determine earnings on the labor market). On one hand, socioeconomic factors such as income may affect the behavior of starting and

15. Theoretically, the idea is to apply a parametric form on the time preference utility function to make appear a risk aversion parameter.

continuing smoking because it corresponds to an expensive addiction which crowds-out the consumption of other goods (Busch et al. [2004]; Wang et al. [2006]). The impact of income on smoking might be ambiguous. In fact, as cigarettes are increasingly expensive, one can think that only people with high income can afford to smoke. In her paper, Balia [2014] finds a positive impact of income on the probability of starting smoking. However, Huisman et al. [2005] study the impact of income and education on the prevalence of smoking in the European Union and find that both income and education are negatively related to smoking. Pfeifer [2012] also finds a negative relationship between income and the number of cigarettes consumed per day. Smoking is not only an unhealthy habit but it is also an expensive one, and the level of some vital expenditures decreases with smoking-related expenditures (Wang et al. [2006]). Moreover, smoking prevalence might also be related to the job of individuals, such that smoking prevalence is generally higher among disadvantaged groups (Hiscock et al. [2012]; Syamlal et al. [2015]). In this sense, Marsh and McKay [1994] find that, in Britain, smoking prevalence is higher among individuals who have a smaller income. Similarly, Hersch [2000] explains that people in lower positions (like blue-collar workers) are more inclined into smoking. On the other hand, other studies (Auld [2005]) focus on the impact of smoking on wages. Indeed, the health effects of smoking cause diseases and make smokers less healthy. This can therefore lead to a lower labor productivity. Moreover, there is a considerable cost for employers of hiring a smoker because of healthcare costs. Finally, there might be a self-selection of smokers into occupations which provide a better health coverage with lower wages in exchange (Lokshin and Beegle [2006]). Thus, the effect of income on smoking is contrasted among studies and both factors seem to be linked, leading to a simultaneity bias. Empirically, we estimate both outcomes simultaneously to overcome this issue.

3.3 SHARE survey

In this chapter, we conduct country-specific analyses, using a pooled database from waves five and six of SHARE. Our main dependent variable determines the smoking prevalence. SHARE offers detailed information on smoking, such that we choose a dummy variable identifying people who currently smoke. 10.60% of our sample currently smoke, corresponding to 14,046 individuals aged 50 and over. Among them, a majority is men (52.34%), Czech (9.36%), retired (43.22%), reaches high-school (as a highest degree, 43.02%), says being in good health (35.98%) and does not have a complementary health insurance (61.74%).

The choice of smoking, as it is harmful for health, might be affected by the rate of time preference, where individuals with a low discount rate might invest more in health and

thus might not engage in smoking behaviors. In order to accurately capture time preferences, we use a financial planning horizon item as a proxy. Individuals are asked: “In planning your saving and spending, which of the following time periods is most important to you? Next few months (this option also includes next few days and next few weeks); next year; next few years; next 5-10 years; longer than 10 years”. This measure has been previously used by [Picone et al. \[2004\]](#) or [Adams \[2009\]](#), who find consistent results (see section 3.2.2). [Picone et al. \[2004\]](#) use this measure to capture time preferences of individuals in the demand for medical tests.¹⁶ They choose to create binary variables for short time horizon (corresponding to individuals who answer “less than a year”), for large time horizon (when individuals answer “longer than 10 years”), and for their reference group (which concerns a financial time horizon between a year and 10 years). We follow a similar intuition by creating a dummy variable equal to one when individuals respond “next few months”, and zero otherwise. Figure 3.4 shows the distribution of this variable between

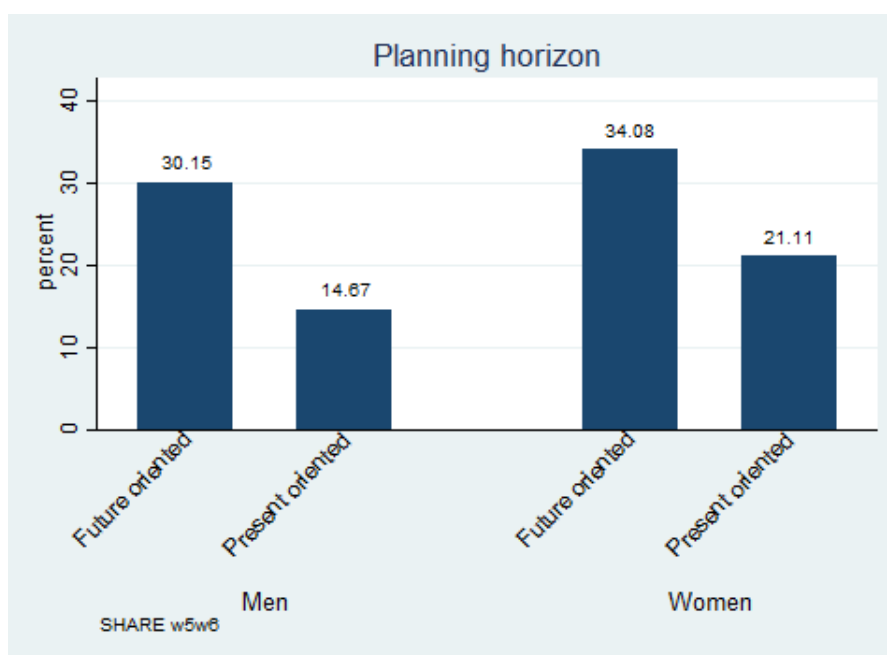


Figure 3.4 – Planning horizon

Source: SHARE

men and women.¹⁷ In our sample, the Danish seem to be the most future-oriented people (90.26% of them).¹⁸

16. They find that individuals who do cancer screening are the ones with lower time preference.

17. Some imputations have been done to overcome a missing information issue. Details are given in the appendix section 3.A.2.

18. One issue with this variable is its financial concern instead of having a discount for health. However, since our variable of interest is smoking, which is a costly dependence, there is good reason to believe that financial aspect of time preference is important in this case. Moreover, such proxy has already been used, as explained earlier.

Questions about the willingness to take risks in health are difficult to access. In order to have the most accurate measurement of risk attitudes, we should turn to experimental studies which measure risk-taking behavior with real money outcomes. However, with a representative sample it is costly and difficult to perform. SHARE provides a question about risk aversion which we use to investigate risk preferences. Individuals have to answer the following question: “When people invest their savings they can choose between assets that give low return with little risk to lose money, for instance a bank account or a safe bond, or assets with a high return but also a higher risk of losing, for instance stocks and shares. Which of the following statements comes closest to the amount of financial risk that you are willing to take when you save or make investments? Take substantial financial risks expecting to earn substantial returns; take above average financial risks expecting to earn above average returns; take average financial risks expecting to earn average returns; not willing to take any financial risks”. To increase variability in each category, we create a dummy variable equal to 1 when an individual answers “not willing to take any risks” and 0 otherwise. Figure 3.5 shows the distribution of this variable

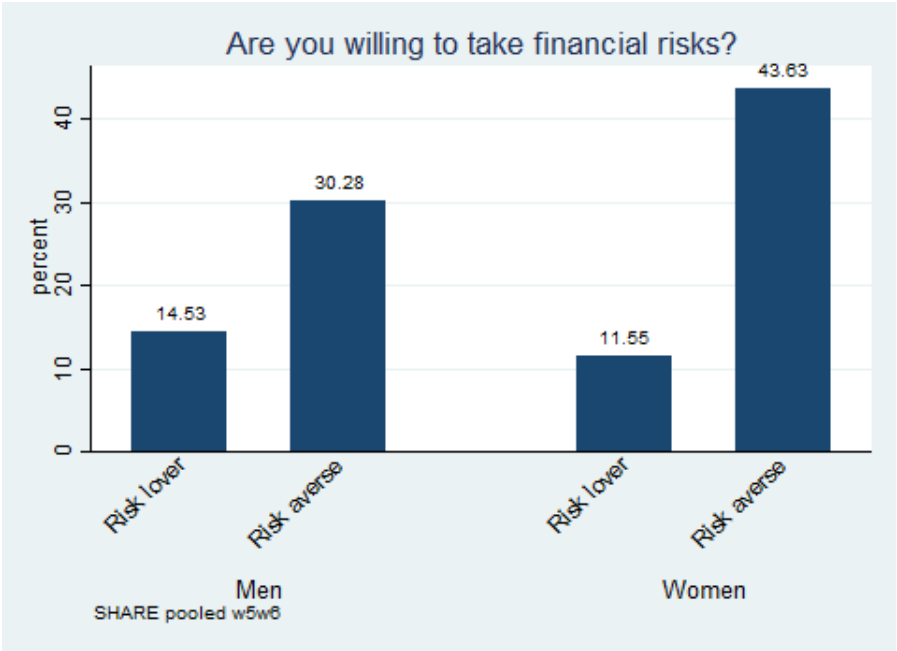


Figure 3.5 – Risk preferences

Source: SHARE

where the majority of individuals seems to be risk averse.¹⁹ This tendency is overriding in Spain (91.26% of them) whereas individuals who are the most risk lover live in Sweden (53.38 % of them). In the survey, risk preference items refer to financial aspect instead of

19. Some imputations have been done to overcome a missing information issue. Details are given in the appendix section 3.A.2.

health items (such as, for instance, the risk of lung cancer from smoking which has been previously used). However, because smoking is a costly addiction, financial aspect might be important as individuals aged 50 years old and over might also face more health issues inducing more spending. Moreover, [Dohmen et al. \[2011\]](#) prove that risk-taking questions, either general or specific in contexts, are strongly correlated to each other and relate to individual choices made over a real-stakes lottery experiment, such that subjective risk-taking item is a reliable predictor (see also [Arrondel et al. \[2004\]](#)).

We also consider the logarithm of individual income, defined as the sum of individual imputed income for all household components in the database.²⁰ Indeed, we consider smoking as an economic choice in which individuals face a budget constraint represented by income. Figure 3.6 shows the deviation from the mean of the logarithm of income, by country, where, individual income is higher in Switzerland than in other countries. SHARE contains a lot of information on individual sociodemographic characteristics. We

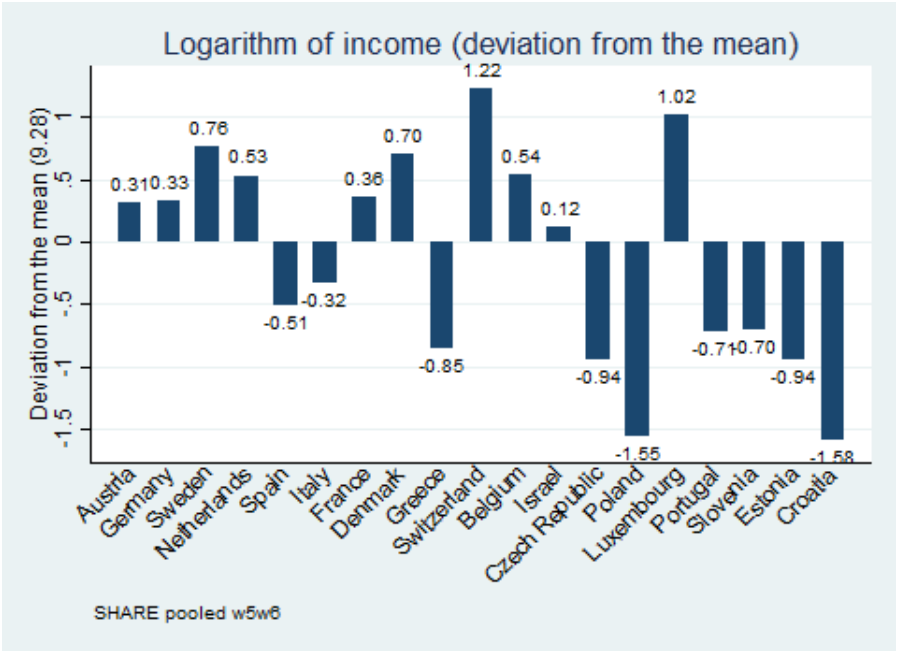


Figure 3.6 – Logarithm of individual income, deviation from the mean

Source: SHARE

thus include control variables in our estimates such as age, education²¹, marital status²² or gender. We also include a dummy which is equal to 1 when individuals report having a complementary health insurance. Indeed, when individuals care about their health by

20. See table 3.3 in the appendix section for further information.
 21. This is a continuous variable (education in years) derived from the International Standard Classification of Education (ISCED).
 22. This is a dummy variable equal to one when the individual is in couple.

subscribing to complementary health insurance, they might be less prone to smoke and less risk lovers. Moreover, we consider a dummy variable derived from self-perceived health status because depending on their health status, individuals might adjust their decision concerning their smoking decisions.²³ Furthermore, health status might be a determinant of income for individuals aged 50 and over since they face more health issues which in turn induce health spending or reduce the ability to work and thus earned income (Michaud and Van Soest [2008]). Then, retirement status is also considered. It is specifically used as a determinant of income since in our sample individuals might be either retired or not. Finally, we include a quantitative information on how often an individual sees a medical doctor as a determinant for risk preferences. Indeed, we suppose that an individual who often sees a medical doctor is someone who does not like risk (see table 3.3 in Appendix 3.A.1 for further details.).

Finally, as explained earlier, in Becker and Murphy [1988]’s rational addiction model, current smoking consumption depends on past consumption of the addictive good. Unfortunately, this variable contains a lot of missing information, considerably reducing our sample. We thus assume that past consumption is not observable, however captured by the errors terms and individual heterogeneity. Concerning future consumption, this might be a determinant of current consumption (according to the rational addiction theory). Indeed, the latter corresponds to information acquired by individuals about health risks from smoking, such that they anticipate addictive and negative risks. Unfortunately, we cannot observe future consumption such that we assume the latter to be directly captured by current consumption and through individual heterogeneity (as Etile [2002]).

3.4 Econometric framework

Smoking participation is modeled as an outcome of utility maximization in which an individual maximizes his utility under an income constraint and becomes a smoker only if the expected benefits from smoking are larger than the expected costs. This econometric model follows the theoretical work of Grossman [1972a] and Becker and Murphy [1988] in which the maximization yields to an optimal demand of the addictive good and labor market productivity (i.e. income), these outcomes being simultaneously affected by each other and thus, simultaneously determined (Auld [2005]). We implement a simultaneous equations model to consider endogeneity issue associated to this simultaneity.

In order to identify determinants of smoking decision, we take into account variables determining the net benefits of smoking. We use a probit model to estimate the probability

23. This variable is equal to one when individuals answer being in good, very good or excellent health.

of smoking. Smoking participation is a qualitative variable such that:

$$S_{it} = \begin{cases} 1 & \text{if } S_{it}^* > 0 \\ 0 & \text{otherwise} \end{cases}$$

in which S_{it}^* is a latent variable underlying the smoking status of individual i at date t and implies the following latent variable specification of the model:

$$S_{it}^* = X_{it}\beta + \epsilon_{it}^1$$

where X_{it} corresponds to explanatory variables defined hereafter, and ϵ_{it}^1 is an error term assumed to be normally distributed. For individual i , this can be stated as follows:

$$\forall i = 1 \dots N \quad \& \quad \forall t = 1 \dots T_i$$

$$S_{it}^* = \beta_0 + \beta_1 \ln Inc_{it} + \beta_2 RP_{it} + \beta_3 TP_{it} + \beta_4 X_{it}^4 + \epsilon_{it}^1 \quad (3.1)$$

in which T_i corresponds to the number of observations for an individual i ; $\ln Inc_{it}$ is a continuous variable representing the logarithm of income of individual i at date t ; RP_{it} corresponds to the risk preferences variable; TP_{it} corresponds to individual's time preferences, and, X_{it}^4 is a vector of control variables (such as age, gender, schooling, health status, and complementary health insurance). There is not a huge literature investigating risk preferences empirically such that it is difficult to make a hypothesis on the sign of β_2 . However, according to [Pfeifer \[2012\]](#), we expect β_2 to be negative because as the costs of smoking increase, a person is less inclined in becoming a smoker.²⁴ As a result, a risk lover person might be more likely to smoke. Concerning parameter β_3 which represents time preferences, it is expected to be negatively correlated to the probability of smoking. Future-oriented individuals have no interest in smoking because this is bad for their health and decreases life expectancy. Individuals with higher discount rates will place higher value on immediate consumption and less value on the risk of early death due to smoking. We thus test the hypothesis that individuals with high risk aversion (following [Pfeifer \[2012\]](#)'s results) and low discount rates (future-oriented people) do not select in smoking. The effect of parameter β_1 might be ambiguous. In fact, as cigarettes are becoming more expensive we can think that only people with high income choose to smoke. However, people aged 50 and over are the ones who have a lot of health spending. As a result, the elderly might choose to reduce smoking since this is expensive and bad for their health, and, might rather promote health expenditure instead of tobacco expenditures.

24. However, we should note that our population of interest, individuals aged 50 and over, differs from the population studied in [Pfeifer \[2012\]](#), in which individuals aged 18 to 65 are studied.

Finally, we cannot estimate only equation 3.1 because of unobserved components which might explain both S_{it} and $\ln Inc_{it}$ on one hand, and S_{it} and RP_{it} on the other hand, thus creating biases (see subsection 3.2.2 for a literature review).

Mincer [1974] models income through a human capital earnings function in which logarithm of earnings is estimated using years of education and years of potential labor market experience. Considering the data availability, we can write out a rearranged Mincer equation, modified to include smoking:

$$\begin{aligned} \forall i = 1 \dots N \quad \& \quad \forall t = 1 \dots T_i \\ \ln Inc_{it} = & \alpha_0 + \alpha_1 S_{it} + \alpha_2 Educ_{it} + \alpha_3 Retired_{it} + \alpha_4 Age_{it} \\ & + \alpha_5 Age_{it}^2 + \alpha_6 Gender_i + \epsilon_{it}^2 \end{aligned} \quad (3.2)$$

in which $Retired_{it}$ corresponds to the retirement status of individual i at date t ; Age_{it} captures the impact of the age on the salary; $Gender_i$ is a binary variable equals to one if the individual is a women and $Educ_{it}$ corresponds to education in years.

Then, because risk preferences and smoking might simultaneously determine each other (see section 3.2.2), we also specify a risk equation. Risk preference's proxy is defined as a qualitative variable such that:

$$RP_{it} = \begin{cases} 1 & \text{if } RP_{it}^* > 0 \\ 0 & \text{otherwise} \end{cases}$$

in which RP_{it}^* is a latent variable and implies the following latent variable specification:

$$\begin{aligned} \forall i = 1 \dots N \quad \& \quad \forall t = 1 \dots T \\ RP_{it}^* = & \Phi_0 + \Phi_1 S_{it} + \Phi_2 X_{it}^2 + \epsilon_{it}^3 \end{aligned} \quad (3.3)$$

in which X_{it}^2 corresponds to a set of variable of controls such as age, gender, marital status, schooling, complementary health insurance and whether the individual often sees/talks to a medical doctor.

On one hand, probability of smoking and income seem to be simultaneously determined (equations 3.1 and 3.2). On the other hand, in equations 3.1 and 3.3, smoking and risk preferences also simultaneously determine each other. A simultaneous equations model is used when three equations with linkages among their error processes are jointly estimated. Moreover, such methods are necessary to take into account simultaneity biases. As a

result, we have the following three-equations system:

$$\left\{ \begin{array}{l} S_{it}^* = \beta_0 + \beta_1 \ln Inc_{it} + \beta_2 RP_{it} + \beta_3 TP_{it} + \beta_4 X_{it}^4 + \epsilon_{it}^1 \\ \ln Inc_{it} = \alpha_0 + \alpha_1 S_{it} + \alpha_2 Educ_{it} + \alpha_3 Retired_{it} + \alpha_4 Age_{it} \\ \quad + \alpha_5 Age_{it}^2 + \alpha_6 Gender_i + \epsilon_{it}^2 \\ RP_{it}^* = \Phi_0 + \Phi_1 S_{it} + \Phi_2 X_{it}^2 + \epsilon_{it}^3 \end{array} \right. \quad (3.4)$$

These three equations constitute a simultaneous equations model because they each have an economic meaning on their own, implying the autonomy requirement of such models (i.e., their meanings are derived from economic theory, [Wooldridge \[2015\]](#)). In order to identify the smoking equation (equation 3.1), which is the equation we are interested in, we need to specify the order condition (necessary but not sufficient) to identify any particular equations in system 3.4 ([Wooldridge \[2010\]](#)) and thus to establish the rank condition ([Wooldridge \[2015\]](#)). The order condition implies that at least one exogenous variable is excluded from this equation (equation 3.1), but included in the other equations of the system (equations 3.2 and 3.3). Moreover, the rank condition states that the smoking equation (equation 3.1) is identified if, and only if, the other equations (the modified Mincer equation, i.e., equation 3.2, and, the risk equation, i.e., equation 3.3) contain at least one exogenous variable which is excluded from the “first” equation (smoking equation, i.e., equation 3.1). In this framework, the smoking equation (equation 3.1) is just identified because we need two exogenous variables for the two endogenous variables, income and risk preference, ($\ln Inc_{it}$ and RP_{it}); and, we have two available variables, which are the retirement status in the income equation (equation 3.2), and, the variable on the number of times an individual sees/talks to a medical doctor in the risk equation (equation 3.3). In this way, identification of the other equations (equations 3.2 and 3.3) is just the mirror image of the statement for the previous equation. Equations 3.2 and 3.3 are over-identified since we need one exogenous variable for the smoking variable and we have at least two available in the other equations (time preference, TP_{it} , in the smoking equation, i.e., equation 3.1; and, (i) “How often seen/talk to medical doctor?” in the risk equation, i.e., equation 3.3, for the endogenous variable in the income equation, i.e., 3.2; or, (ii) the retirement status for the endogenous variable in the risk equation, i.e., equation 3.3).

Even if each identified equation can be estimated by two stage least square (2SLS), system estimation method, such as Full Information Maximum Likelihood (FIML) is more efficient since it takes into account the possible correlation of errors of all equations (ϵ_{it}^1 ; ϵ_{it}^2 and ϵ_{it}^3) resulting from the simultaneous determination of smoking and income on one hand, and, of smoking and risk on the other hand. Moreover, FIML is efficient when we

focus on the reduced form model (Sargan [1964]; Sargan [1970]; Sargan [1988]), and the latter allows the simultaneous estimation of non-linear (smoking equation 3.1 and risk equation 3.3) and linear models (income equation 3.2). As a result, the error terms are specified as follows:

$$\begin{cases} \epsilon_{it}^1 = \eta_i^1 + \zeta_{it}^1 \\ \epsilon_{it}^2 = \eta_i^2 + \zeta_{it}^2 \\ \epsilon_{it}^3 = \eta_i^3 + \zeta_{it}^3 \end{cases} \quad (3.5)$$

With a simultaneous equations method, we have the following variance-covariance matrix for individual effects:

$$\Sigma_\eta = \begin{pmatrix} \sigma_1^2 & \rho_{1,2}\sigma_1\sigma_2 & \rho_{1,3}\sigma_1\sigma_3 \\ \rho_{1,2}\sigma_1\sigma_2 & \sigma_2^2 & \rho_{2,3}\sigma_2\sigma_3 \\ \rho_{1,3}\sigma_1\sigma_3 & \rho_{2,3}\sigma_2\sigma_3 & \sigma_3^2 \end{pmatrix}$$

in which ρ represents the correlation between individual terms η_i .²⁵

3.5 Results

Our objective is to determine whether individual heterogeneity impacts the smoking decision. As a result, we focus our analysis on the coefficients of the smoking equation (i.e., equation 3.1), specifically on the impacts of income (β_1), risk preferences (β_2), and time preferences (β_3). However, due to endogeneity issues of income and risk aversion, we implement two other equations (an income equation, i.e., equation 3.2; and a risk equation, i.e., equation 3.3) in which smoking is a determinant (corresponding to coefficients α_1 and Φ_1 in equations 3.2 and 3.3 respectively). Endogeneity biases are related to simultaneity issues, such that we implement a simultaneous equations system with three equations ((i) smoking, (ii) income and (iii) risk, i.e., equations 3.1, 3.2, and 3.3 respectively) to overcome these issues. Thus, the income equation (i.e., equation 3.2) and the risk equation (i.e., equation 3.3) allow to control for endogeneity biases which may arise in the smoking equation (i.e., equation 3.1). In other words, coefficients associated to smoking (α_1 and Φ_1) in the two additional equations (income and risk equations, i.e., equations 3.2 and 3.3) are estimated in order to strengthen our results; such that income, risk preferences, and time preferences, in the smoking equation (i.e., β_1 , β_2 , and β_3 in equation 3.1), are estimated without biases. Our analysis highlights instantaneous causality such that outcomes are simultaneously determined (there are no lagged variables in our estimates). Thus, we cannot say that whenever a variable x has a positive (or negative) and significant impact

25. You will find the associated variance-covariance matrix for idiosyncratic errors, as well as, the likelihood equation at the end of this Ph.D. dissertation, in Appendix A “**Simultaneous equations model with non-linear and linear dependent variables on panel data**”.

on another variable y , then y also has a positive (or negative) and significant impact on x . Results presented in table 3.1 correspond to a comparative analysis of the determinants of smoking between different European countries, plus Israel.²⁶

First, results concerning the smoking-income relationship correspond to coefficients β_1 in equation 3.1, and α_1 in equation 3.2.

Focusing on the impact of income on smoking (coefficient β_1 in equation 3.1), in the majority of countries, results suggest a negative impact among individuals aged 50 and over.²⁷ Thus, the richest people have a smaller probability to smoke. This result implies that tobacco might be considered as an inferior good for Europeans aged 50 and over. We also control for education in our estimates since education and income are often correlated. For the other countries, results are either not significant (France, Czech Republic and Italy) or positive (Austria).

Turning to the impact of smoking on income (coefficient α_1 in equation 3.2), the positive impact of smoking implies that individuals who smoke today are, on average, richer than individuals who do not smoke. Thus, linking this result ($\alpha_1 > 0$) with the previous one ($\beta_1 < 0$), we can say that Europeans who smoke are richer than non-smoker Europeans on average ($\alpha_1 > 0$), but, when an individual becomes rich, then his probability to smoke decreases ($\beta_1 < 0$).

Then, in order to investigate the smoking-risk aversion relationship, let us turn to the interpretation of coefficients β_2 in equation 3.1, and Φ_1 in equation 3.3.

Concerning the impact of risk preferences on smoking (β_2 in equation 3.1), results suggest two groups of countries. In the first one, composed of France, Germany, Estonia, Czech Republic, Denmark, Italy, Sweden, Switzerland, Belgium and Slovenia, results suggest that risk averse individuals are the ones who have a higher probability to smoke (positive relationship, i.e., $\beta_2 > 0$). An explanation of this result might be the fact that risk aversion characterizes individuals who are more anxious. Indeed, in epidemiological literature it is said that anxious individuals are more risk averse (Charpentier et al. [2017]). Such anxiety is also proved to increase smoking consumption (Moylan et al. [2013]). Then, the second group of countries, corresponding to Israel, Spain, Austria, and Luxembourg, is characterized by a negative impact of risk aversion on smoking ($\beta_2 < 0$). In other words, risk averse individuals have a smaller probability to smoke, the latter results being found in Pfeifer [2012] for instance, such that this result is expected according to the literature. Concerning the impact of smoking on risk aversion (i.e., coefficient Φ_1 in equation 3.3), results tell the same story as the results of the smoking equation (i.e., equation 3.1) for all

26. Results are available for 14 countries.

27. This result holds for the following ten countries: Germany, Estonia, Israel, Denmark, Spain, Sweden, Luxembourg, Switzerland, Belgium, and Slovenia.

countries except Denmark and Spain, for which signs do not coincide; and Switzerland, for which Φ_1 is not statistically significant. As a result, in the majority of the cases, the interpretation of the sign of the smoking coefficient in the risk equation (i.e., Φ_1 in equation 3.3) is consistent with the interpretation of the sign associated to risk preference in the smoking equation (i.e., β_2 in equation 3.1). More specifically, when smoking has a positive effect on being risk averse, the latter is justified with an anxiety pathway. This positive association might reflect the fact also explained by Moylan et al. [2013], where they show that nicotine impacts neurodevelopment of individuals, such that they become more anxious with age and thus more risk averse.

Finally, concerning the impact of time preferences on tobacco consumption (coefficient β_3 in equation 3.1), we can distinguish two groups of countries. The first one, composed of France, Germany, Estonia, Czech Republic, Denmark, Italy and Sweden, suggests that present-oriented people have a higher probability to smoke. The fact that being present-oriented increases the probability to be a smoker (the strongest result) is in line with the literature (Carbone et al. [2005]; Adams [2009]). In the second group of countries time preferences do not impact smoking (Israel, Spain, Austria, Luxembourg, Switzerland, Belgium, and Slovenia). For these people aged 50 and older, their smoking decision is not impacted by their preferences for either present or future.

Now, we can turn to correlations between the unobserved individual factors of the different equations (see table 3.2). Whenever correlation coefficients are statistically significant, then unobserved factors common to each equation might be at play, such that these equations should be considered simultaneously. We do not focus on correlation coefficients between unobserved factors of the income and risk equations ($\rho_{2,3}$) because we are interested on the determinants of tobacco consumption. Thus, we focus our analysis on the correlation coefficients of unobserved factors between smoking and income on one hand, and smoking and risk on the other hand, in order to have insights on the existence of unobserved factors in the smoking equation (equation 3.1).

First of all, correlation coefficients between unobserved components of the smoking and income equations (coefficient $\rho_{1,2}$) are positive and statistically significant for every country but Israel and Austria, so that unobserved factors of the two equations are positively correlated. As a result, there are simultaneity issues between these two outcomes which legitimates our estimation strategy. An unobserved personal trait which might explain this positive sign is the religious status of individuals.²⁸ Indeed, Auld [2005] explains that

28. The religious assumption has also been made by Kaestner [1991] and Hamilton and Hamilton [1997]. Unfortunately, we cannot test this assumption because the religious status of individuals is not given in the database.

Catholic religious respondents tend to smoke more. Moreover, [Ewing \[2000\]](#) finds that persons raised in the Catholic religion earn more than their non-Catholic counterpart.²⁹

Finally, concerning the correlation between unobserved components of the smoking and risk equations (coefficient $\rho_{1,3}$), coefficients are statistically significant for all countries except Belgium and Slovenia, such that there are simultaneity issues between smoking and risk aversion, or in other words, there might exist unobserved components common to both outcomes. These coefficients can be grouped into two groups of countries. In the first group, composed of France, Germany, Estonia, Czech Republic, Israel, Italy, and Spain, coefficients are negative. [Jusot and Khlat \[2013\]](#) explain that an unobserved factor might be past health status. Indeed, an individual who had a very bad health status in the past should be less inclined to smoke today. On the other hand, having experienced a very bad health status in the past may lead to a greater risk aversion today, in the case where the individual would like to take less risk to preserve his current health.³⁰ The second group of countries, composed of Sweden, Austria, Luxembourg, and Switzerland, suggests a positive correlation between smoking and risk. An unobserved individual factor which might explain this positive correlation is anxiety. [Charpentier et al. \[2017\]](#) find that individuals who are the most anxious are also the most risk averse; and, such anxiety is also proved to increase smoking consumption ([Moylan et al. \[2013\]](#)).

Table 3.1 – Results of the simultaneous equations model

Equation:	Smoking			Income	Risk
	Income β_1	Risk aversion β_2	Present-oriented β_3	Smoking α_1	Φ_1
France $N=7,301$	0.001 (0.003)	1.618*** (0.028)	0.012** (0.006)	-0.022 (0.023)	1.585*** (0.028)
Germany $N=9,572$	-0.017*** (0.004)	1.624*** (0.025)	0.027*** (0.008)	-0.102*** (0.017)	1.618*** (0.025)
Estonia $N=9,878$	-0.031*** (0.007)	1.582*** (0.035)	0.029*** (0.007)	-0.123*** (0.018)	1.612*** (0.032)
Czech Rep. $N=9,081$	-0.008 (0.005)	1.554*** (0.033)	0.018*** (0.006)	-0.038** (0.019)	1.540*** (0.024)
Israel $N=2,994$	-0.167*** (0.039)	-0.140* (0.072)	0.061 (0.054)	0.454*** (0.048)	-0.157* (0.033)
Denmark $N=7,206$	-0.013* (0.007)	1.766*** (0.024)	0.048*** (0.012)	-0.051*** (0.014)	-1.745*** (0.025)
Italy $N=8,177$	0.002 (0.001)	1.546*** (0.027)	0.008** (0.004)	-0.069* (0.035)	1.538*** (0.028)
Spain $N=9,924$	-0.018*** (0.003)	-1.578*** (0.036)	0.008 (0.006)	0.155*** (0.030)	1.600*** (0.036)

29. He further explains that “the Catholic religion may add to a person’s stock of human capital and/or it may act as a signal of desirable labor market characteristics such as discipline, honesty, trustworthiness, and high motivation”.

30. However, we cannot control for past health status in this study because it will considerably reduce our sample size due to the short time horizon considered.

Table 3.1 – Results of the simultaneous equations model (continued)

Equation:	Smoking			Income	Risk
	Income	Risk aversion	Present-oriented	Smoking	
	β_1	β_2	β_3	α_1	Φ_1
Sweden $N=7,522$	-0.516^{***} (0.034)	0.192^{***} (0.046)	0.083^{**} (0.035)	0.209^{***} (0.019)	0.198^{***} (0.055)
Austria $N=6,943$	0.144^{***} (0.048)	-2.754^{***} (0.069)	0.091 (0.059)	0.046^{**} (0.020)	-3.118^{***} (0.062)
Luxemb. $N=2,897$	-0.337^{***} (0.027)	-0.795^{***} (0.073)	0.065 (0.045)	0.915^{***} (0.059)	-0.439^{***} (0.126)
Switzer. $N=5,370$	-0.189^{***} (0.029)	1.049^{***} (0.037)	0.002 (0.029)	0.478^{***} (0.032)	0.088 (0.105)
Belgium $N=9,687$	-0.848^{***} (0.010)	0.054^{***} (0.016)	-0.006 (0.014)	0.136^{***} (0.029)	0.161^{***} (0.043)
Slovenia $N=6,218$	-0.079^{***} (0.009)	1.032^{***} (0.025)	0.009 (0.012)	0.619^{***} (0.030)	1.016^{***} (0.032)
N=102,770					

***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are into parentheses below coefficients.

Note: results do not converge for the following countries for which observations are available only one year: Croatia; Portugal; Poland; Greece and The Netherlands.

Table 3.2 – Correlations between individual unobserved effects

Correlation	Smoking/Income	Smoking/Risk	Risk/Income
	$\rho_{1,2}$	$\rho_{1,3}$	$\rho_{2,3}$
France $N=7,301$	0.043^{***} (0.015)	-0.481^{***} (0.012)	0.014 (0.015)
Germany $N=9,572$	0.029^{**} (0.013)	-0.452^{***} (0.011)	0.047^{***} (0.013)
Estonia $N=9,878$	-0.722^{***} (0.006)	0.061^{***} (0.013)	-0.051^{***} (0.013)
Czech Rep. $N=9,081$	0.121^{***} (0.014)	-0.333^{***} (0.012)	-0.029^{**} (0.014)
Israel $N=2,994$	-0.002 (0.023)	-0.348^{***} (0.020)	0.116^{***} (0.023)
Denmark $N=7,206$	0.068^{***} (0.016)	0.041^{***} (0.016)	0.011 (0.016)
Italy $N=8,177$	0.026^* (0.014)	-0.598^{***} (0.009)	-0.014 (0.013)
Spain $N=9,924$	0.031^{**} (0.013)	-0.858^{***} (0.003)	-0.016 (0.013)
Sweden $N=7,522$	0.052^{***} (0.015)	0.112^{***} (0.015)	0.018 (0.015)
Austria $N=6,943$	-0.114^{***} (0.019)	0.995^{***} (0.0003)	-0.108^{***} (0.019)
Luxemb. $N=2,897$	0.721^{***} (0.011)	0.997^{***} (0.0002)	0.773^{***} (0.009)
Switzer. $N=5,370$	0.694^{***} (0.010)	0.262^{***} (0.018)	0.875^{***} (0.005)
Belgium $N=9,687$	0.995^{***} (0.0001)	-0.047 (0.029)	-0.051^* (0.029)
Slovenia $N=6,218$	0.639^{***} (0.009)	-0.509 (0.012)	-0.625^{***} (0.009)

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

Standard deviations are into parentheses below coefficients.

3.6 Conclusion

This analysis sheds light on the determinants of smoking in Europe, while considering simultaneous relationships between both smoking and income on one hand, and, smoking and risk preferences on the other hand. The fifth and the sixth waves of the SHARE survey, which follows Europeans aged 50 and older, are used. We are able to identify proxies for both time and risk preferences for a sample of almost 102,700 individuals, whereas a number of studies investigates the impact of either time preferences or risk preferences among smoking, or use experimental approaches to consider both aspects, but sample size is then questionable. The originality of this article stands from the fact that we implement a simultaneous equations model to correctly capture the impact of preferences and income while controlling for endogeneity issues. Moreover, thanks to this method, we offer a comparative analysis among 14 countries.

Focusing on risk preferences, two relationships with smoking emerge, depending on where individuals live. On one hand, results suggest that individuals who are risk averse have a higher probability to smoke. Risk aversion might relates to anxious people which are more prone to smoke. Thus, governments might develop social policies to prevent and treat anxiety. On the other hand, results also suggest that individuals who are risk averse have a smaller probability to smoke, which is in line with economic theory. In these countries, individuals do not want to take the risk of altering their health by smoking. Concerning smoking and income, we can say that individuals who smoke are, on average, richer than non-smokers. However, Europeans aged 50 years and over also consider cigarettes as an inferior good. Finally, concerning time preferences, individuals who are future-oriented consider negative impacts of smoking consumption on health, so that their probability to smoke is smaller. In the end, a universal result for all countries is that differences in time preferences lead to differences in smoking consumption. Improving individuals foresight into how their preferences will change through time can improve the quality of their decisions and thus their level of investments in health. Public health interventions are needed to favor health investments.

Finally, all our results are confirmed by the presence of unobserved individual factors between smoking and both income and risk preferences. As a result, smoking, income, and risk preferences should be considered simultaneously in order to avoid simultaneity biases.

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3.A Appendix A: descriptive statistics

3.A.1 Variables of interest and covariates

Table 3.3 – Descriptive statistics of the variables - Pooled database

Variables	Mean	Std. dev.	Min.	Max.	# obs.
Income	16,790.45	38,369.54	0	8,809,227	132,554
Log of income	9.22	1.14	-1.07	15.99	130,629
<u>Current smoker</u>	% (44.07% of the sample)				58,420
Yes	24.04 (10.60)				14,046
No	75.96 (33.48)				44,374
<u>Risk pref. (financial risks)</u>	% (55.86% of the sample)				74,050
Take substantial	1.09 (0.61)				807
Take above average	2.94 (1.65)				2,175
Take average	20.90 (11.67)				15,473
Not willing to take any	75.08 (41.94)	<i>Reference</i>			55,595
<u>Time pref.</u>	% (53.69% of the sample)				71,173
Next few days/weeks	36.27 (19.48)	<i>Reference</i>			25,817
Next year	20.70 (11.11)				14,731
Next few years	23.67 (12.71)				16,845
Next 5-10 years	12.66 (6.79)				9,011
Longer than 10 years	6.70 (3.59)				4,769
Age	67.57	10.07	50	106	132,554
Retired (%)	57.54				132,554
<u>Gender</u>	%				
Women	55.74				73,879
Men	44.26	<i>Reference</i>			58,675
<u>Education</u>	%				132,554
No diploma	5.65				7,483
Primary education	16.89				22,394
Secondary education	17.86				23,674
High School	37	<i>Reference</i>			49,033
University degree	21.74				28,822
Ph.D.	0.87				1,148
<u>Health status</u>	%				132,554
Poor	11.15				14,779
Fair	27.81				36,862
Good	36.08	<i>Reference</i>			47,821
Very Good	17.52				23,224
Excellent	7.44				9,868
<u>Complementary health ins.</u>	% (99.32% of the sample)				131,658
Yes	38.22 (37.96)				50,320
No	61.78 (61.36)				81,338

3.A.2 Details of imputations

1. Concerning the planning horizon variable, we first create a dummy variable equal to one when individuals answer they focus on next few months when they plan their saving and spending. However, this leads us with 57.13% of missing information. We are able to recover some missing information by making assumption on the consistency and stability of the answers between the two available waves of data. As a result, among these missing information, we suppose that individuals who do not answer in wave five (*resp.* wave six) but who report being future-oriented (*resp.* present-oriented) in wave six (*resp.* wave five), were future-oriented (*resp.* present-oriented) in wave five (*resp.* wave six). In this way, we recover 49.07% of information among the missing answers, such that our new binary variable contains only 28.21% of missing information.

2. Concerning variable for risk aversion, we first create a dummy variable equal to one when individuals respond they are not willing to take any risks to the question “are you willing to take financial risks?”. However, this variable contains 55.40% of missing information. We are able to reconstruct information for the missing information thanks to responses of either wave five or wave six. As a result, among missing information, we suppose that individuals who do not answer in wave five (*resp.* wave six) but who report being risk lover (*resp.* risk averse) in wave six (*resp.* wave five), were risk lover (*resp.* risk averse) in wave five (*resp.* wave six). In this way, we recover 44.91% of information among the missing answers, such that our new binary variable contains only 30.90% of missing information.

General conclusion

Context

Health equity corresponds to a judgment based on the concept of social justice. It focuses on the injustice related to inequalities observed either in the distribution of a resource dedicated to health or in health outcomes ([Potvin et al. \[2010\]](#)). According to the World Health Organization (WHO), equity is defined when “there are no avoidable or remediable differences among groups of people”, in which groups are defined socially, economically, demographically or geographically. In this way, we can also define health inequities which correspond to differences in health status of different population groups, and which need to be prevented by governments. These inequities are socially and economically costly for individuals and societies. [Sen \[2002\]](#) speaks about “Why health equity?” during the third conference of the International Health Economics Association in 2001. In his speech, first of all, he points to the fact that equity in health cannot just be seen with health only because when speaking about equity, other factors might play an important role (economic allocations, social arrangements or justice, for instance). By considering inequity, one needs to disentangle between individuals who cannot have the opportunity to achieve good health from individuals who do not want to attain this goal, according to personal decisions. Since the early eighties, equity appears to be a key goal in health policy decisions in OECD countries and other developing countries. [Culyer and Wagstaff \[1993\]](#) explore different definitions of equity in health care and conclude that “the equity of distributions of health care is equality of health”, such that looking for equity underlines looking for equality in the health sector. [Aristotle \[1972\]](#) distinguishes between horizontal equity and vertical equity. On one hand, horizontal equity requires the same treatment for similar individuals. On the other hand, vertical equity implies a different treatment for dissimilar individuals by considering differences between them.

Health is not just the outcome of genetic or biological processes but is also influenced by social and economic conditions. These influences are known as the social determinants of health. Inequalities in social conditions rise the appearance of unequal health outcomes

for different social groups. The existence of social inequalities in health³¹, demonstrated since the mid-nineteenth century, has been widely investigated in developed countries (Villermé [1830]; Van Doorslaer and Jones [2003]). Given the existence and the increase of health inequalities in countries with effective health systems, research on health inequalities is oriented in three directions: the extent of social inequalities in health, the investigation of the determinants of health inequalities and finally the study of public policies to reduce these inequalities.

As a result, it is important to focus on the concept of equality since health public policies sometimes favor the most well-off in a first place. Indeed, socioeconomic groups of individuals with better cultural capital or financial resources are more inclined to understand and carry out health prevention, such that it results in more social inequalities in health. These inequalities are linked to individual living conditions, housing, education, employment or income. Public policies need to consider that individual health results from living conditions and employment which interact with individual socioeconomic characteristics. Governments need to consider this interaction because health must not be seen as a sole medical concept but as a medical concept with social and environmental factors.

In this perspective, one should favor cohesion between economists and governments to establish appropriate researches and give precise answers. At the European level, policy makers establish a new common framework: Health 2020. The goal is to settle public policies and action across the different governments to: significantly improve the health and well-being of populations, reduce health inequalities, strengthen public health and ensure people-centered health systems that are universal, equitable, sustainable and of high quality”. Indeed, in order to promote economy, being in good health is a key determinant since it plays a major role in economic and social development. Health 2020 is constituted of seven main components: reach the highest value of health with the adoption of a common policy framework to improve health and reduce inequalities; health development to achieve other goals in life; problem-solving to new challenges; engagement of different partners to broaden opportunities; a focus on equity by identifying health gaps to reduce them; learning from previous experiences; and, strategic thinking across governments. All together, the goals of these key determinants are to improve health, reduce health inequalities, and, improve leadership and governance for health.³²

Social inequalities in health are part of a broad research area in which this Ph.D. dissertation is part of. Indeed, in an ageing population context with increasing inequalities,

31. This term refers to the study of differences in health status observed between social groups.

32. See [WHO](#) website.

governments and policy makers need to understand relationships between health and income among individuals aged 50 and over in order to improve their health while decreasing inequalities. Income seems to impact health in a causal way, however, the reverse relationship might be at play. If so, public policies affecting both income and health are essential to reduce inequalities in the health-income gradient.

Objectives

The aim of this thesis is thus to investigate the health-income gradient, among Europeans aged 50 and over, in order to give some insights for policy makers whose aim is to reduce income-related health inequalities. More precisely, the whole work of this doctoral dissertation aims at trying to give answers to the following issues, using the Survey of Health, Ageing and Retirement in Europe (SHARE): how does socioeconomic status, and more specifically, income, relate to health? What are the pathways through which income inequalities imply health inequalities? Is it income which impact health or is it the reverse relationship? Do health and income simultaneously determine each other? In what extent do individual preferences play a role in the smoking consumption in different European countries?

In this way, the first chapter, entitled **“In-depth overview on the relationship between health and income: microeconomic evidence and pathways”**, investigates different relationships linking health, income and income inequalities, in a first part. It has been widely said that income and income inequalities are associated to health status; thus, any public policy which influences income and/or income inequalities might influence health. Thus, studying the relationship between income, income inequalities and health is interesting per se. Part of this chapter is dedicated to the empirical analysis of three hypotheses. The first one, called the Absolute Income Hypothesis, was initially introduced by [Preston \[1975\]](#) and highlights a positive and concave relationship between income and health. The second one is the strong version of the Income Inequality Hypothesis and brings to the forefront the impact of income inequalities in a country on health status. The last one, a weak version of the Income Inequality Hypothesis, states that income inequalities are a threat to individuals placed at the lower end of the income distribution, such that income inequalities do not impact low income people and high income people in the same magnitude. Then, the second part of this chapter investigates pathways through which income inequalities might result in health inequalities. Understanding these mechanisms is an important pattern for policy makers or governments whose aim is to improve general health and curb health inequalities. Socioeconomic characteristics of individuals, comparison with their relatives, or other mechanisms at the state level might

alter the level of income inequalities and thus, may also have an impact on health. In order to be considered within the context of economic decision, it is important to document such health effects. Three mechanisms, first introduced by [Kawachi and Kennedy \[1999\]](#), are investigated. The first pathway studied is the disinvestment in human capital or in social goods. Negative health outcomes might depend on the availability of public and private resources, which in turn results in inequalities. A second mechanism investigated is the erosion of the “features of social organization that facilitate cooperation for mutual benefit”, the latter being also defined as the social capital. A final channel studied is that income inequalities might contribute to unhealthiness through stressful social comparisons.

Then, the second chapter of this thesis, entitled “**Health and income: testing for causality on European elderly people**”, investigates bidirectional causal links between health and income. In order to shed light on persistent causal links, the Granger causality is highlighted, such that persistence in the relationships is bringing to the forefront. Moreover, when studying the health-income gradient, unobserved individual effects related to both income and health might be at play, biasing the estimates. Indeed, there are many possible pathways through which earnings can impact health. However, we can also think of the reverse association, because the ability to work might be reduced due to a poor health status. The difficulty in disentangling cause and effect is due to endogeneity, more specifically whenever health and income mutually determine one another, there are simultaneity issues. This lack of a clear understanding of the direction of causality is problematic. Policy makers and governments need to understand the true direction of the causality in the health-income gradient, in order to implement adequate public policies to reduce inequalities. Thus, there is a necessity to investigate causal relationships in the health-income gradient in a simultaneous way (causal link from income to health, and from health to income).

Finally, the last objective of this thesis is to focus on a public health issue. Thus, the third chapter, entitled “**Smoking habits, time preferences and risk preferences: a comparative study over European elderly people**”, focuses on the impact of individual preferences on the smoking consumption in different European countries. According to the World Health Organization (WHO), tobacco consumption is the most significant cause of premature death in the European Union (EU), responsible for nearly 700,000 deaths every year. Two aspects of individual heterogeneity are investigated in the economic decision of smoking. On one hand, we bring to the forefront time preferences since future-oriented individuals correspond to people who value their health in the future. On the other hand, risk preferences are also studied as a determinant of smoking because risk averse individ-

uals do not want to take the risk to alter their health by smoking. Moreover, because tobacco consumption is considered as an economic choice, we also investigate the budget constraint associated, represented by income. One must be careful when investigating the impacts of individual preferences and income on smoking because of endogeneity issues. The idea is to simultaneously consider smoking and risk preferences on one hand, and, smoking and income, on the other hand. This subject is of importance for policy makers due to the impact of smoking in the prevalence of dementia. Smoking is one of the only risk modifiable factor in this disease, which is very costly and harmful. Moreover, investigating individual heterogeneity will help governments to identify individuals who are more prone to smoke.

Methods and results

We intend to use different econometric methods in order to respond to the previous explained objectives. In order to study the latter, we focus on self-perceived health status, a subjective measurement of health status³³, which is considered as a strong predictor of an individual's health ([Benitez-Silva et al. \[2004\]](#)). Moreover, we also focus on current consumption of tobacco as another health outcome.

In the first chapter, the two objectives are to highlight, first, how income, or more generally, socioeconomic measures are related to health on one hand; and, then, pathways through which income inequalities result in health inequalities, on the other hand. Concerning the first objective, we bring to the forefront the three hypotheses enumerated above, by implementing an ordered probit model to correctly consider the qualitative aspect of self-perceived health status. Doing so enables to emphasize correlations between socioeconomic measures and health. We also compute marginal effects at mean to see whether results are robust. Results suggest a positive and concave relationship between income and self-perceived health status (Absolute Income Hypothesis). The strong version of the Income Inequality Hypothesis is also supported by the results thanks to the negative coefficients associated to the Theil index.³⁴ However, results do not support the weak version of Income Inequality Hypothesis. Indeed, when introducing individual ranks in the income hierarchy and interaction terms between the measurement of income inequalities and dummies variables based on quintiles of income, no consistent associations are found. Then, we deeply consider the subjective nature of self-perceived health status

33. In SHARE, individuals are asked to classify their health using ordered qualitative labels from “poor” to “excellent”.

34. Analyses have been implemented using the Gini coefficient, another income inequality index which is country-specific. Results are very similar and robust. Concerning these two indicators, zero represents an egalitarian state, thus the negative relationship is in line with health being better if the index is low.

by implementing a generalized ordered probit model, in which the thresholds themselves are linear function of the explanatory variables, to solve the problem of interpersonal comparisons between people aged 50 and over.³⁵ Results, which consider heterogeneity across individuals, are very similar. Thus, results are robust across specifications, and give strong support to the strong version of the Income Inequality Hypothesis. Concerning the second objective of this chapter, we investigate pathways through which income inequalities result in health inequalities in the strong version of the Income Inequality Hypothesis, which constitutes our strongest results.³⁶ In order to correctly identify the mechanisms explained above, we focus on SHARELIFE, a specific SHARE wave, which collects information on the history of the respondents, such that childhood information may be used. Thus, we consider the whole life-cycle effects by studying factors at younger age and their impacts later in life. We implement an ordered probit model while considering the panel dimension of the database, and compute the average marginal effects associated to give an idea about the magnitude of the effects. Overall, results support the three mechanisms. In other words, investments in human capital have positive and significant effects on self-perceived health status. Indeed, these investments are important to narrow inequalities in a society, and more specifically income inequalities. Reducing income inequalities, will in turn, reduce health disparities among individuals in a society. Governments should continue to promote education, access to culture and increase their investments in health. Moreover, focusing on social capital results suggest that there is solidarity between individuals. It appears that people in bad health receive help from others, whereas people in good health offer their help when someone is in need. Then, social comparisons are also important for health status. Effects of school difficulties during childhood accumulate over time, such that they have negative impacts on health after 50 years old. In addition, not feeling in accordance with the common standard way of life (being discriminated or stressed) also negatively impacts health. Tolerance and mutual aid are therefore two elements to put forward in order to improve health of the population.

Furthermore, in order to investigate bivariate causal effects between health and income, in the second chapter, we implement an original method which has not been used before. Indeed, while highlighting Granger causality, we implement a Full Information Maximum Likelihood (FIML) estimator, which considers unobserved individual factors common to both outcomes. This method enables to correct for endogeneity issues due to the reverse causality which might be at play, by considering correlation among error terms of both income and health. We derive a consistent and identifiable likelihood function which takes

35. Indeed, individuals might compare themselves: “Is the way I consider “good health” the same as you consider this health commodity?”.

36. Indeed, this hypothesis considers both the concavity assumption of income and the negative impact of income inequalities on health.

into account a binary outcome (health) and a linear one (income). However, Granger causality corresponds to a weak causality test, allowing to solve simultaneity issues but not an issue associated to a possible omitted variable bias, which might be considered as a drawback. For instance, since we focus on Europe, we might think of different social security systems specific in all countries, but the latter is difficult to access in the data. Thus, we include groups of countries to try to capture such biases. However, there might still be other missing components causing omitted variables biases. Results suggest that moving from bad to good health at the previous period increases the probability of being in good health today by 6.7%. Moreover, a 1% increase in income at the previous date implies a 1.2% increase in the probability of being in good health today. Furthermore, Granger causality seems also to be at play for income since there is a strong phenomenon of persistence in income (a 1% increase in income at the previous date increases current income by 0.31%), and health has a positive and permanent impact on current individual income (switching from being in bad health to being in good health at the previous period implies an increase of 0.215% in income). This supports the idea that health might determine earnings on the labor market or that health might induce costs. Finally, one important result is the correlation between the error terms which is statistically significant, meaning that simultaneity between health and income is at play. In other words, there are individual unobserved factors common to both equation, such as physical maturity or innate ability. As a result, health and income should be considered simultaneously when investigated. This analysis contributes to a better understanding of the health-income relationship and of the direction of the causality between the two among Europeans aged 50 and over. Finally, this is the first study analyzing the health-income relationship, with a simultaneous equations model, using the SHARE database and establishing strong and permanent Granger causal links.

Finally, we choose to focus on smoking habits as a health outcome. In this way, the third chapter of this Ph.D. dissertation examines the links between heterogeneity in time and risk preferences, income and cigarette consumption while taking into account endogeneity issues due to simultaneity biases from income and risk preferences with smoking. In order to investigate these simultaneity issues, we implement a three-equations model (smoking probability, risk preferences and income) using a FIML estimator. As in the second chapter, we derive a consistent and identifiable likelihood function which takes into account two binary outcomes (smoking probability and risk aversion), and a linear one (income). However, in the literature, it is difficult to identify these preferences because empirically time and risk preferences are difficult to quantify, and proxies are rarely available in databases. This chapter contributes to a better understanding of the determinants of the current smoking consumption because using SHARE, we find proxies for both time

and risk preferences. Concerning the smoking-income relationship, results suggest that Europeans who smoke are, on average, richer than non-smoker Europeans, but, when an individual becomes rich, then he decreases his tobacco consumption. Secondly, concerning the relationship between smoking and risk preferences, two opposite results appear, depending on where individuals aged 50 and over live in Europe. On one hand, in France, Germany, Estonia, Czech Republic, Denmark, Italy, Sweden, Switzerland, Belgium and Slovenia, anxious people are the risk averse individuals and are also the ones who smoke more. On the other hand, in Israel, Spain, Austria, and Luxembourg, risk averse individuals smoke less. In other words, risk lover individuals take the risk of altering their health in the future. Moreover, concerning the impact of time preferences on tobacco consumption, results suggest that present-oriented people smoke more. These individuals do not promote their health in the future. Finally, an important result is that, when studying the determinants of the smoking decision, there are unobserved individual effects common to smoking and both income and risk preferences. These unobserved individual factors might be the religious status between smoking and income, or anxiety and past health between smoking and risk preferences. These results imply that a simultaneous equations strategy is needed when the smoking determinants are investigated among Europeans aged 50 and over. Finally, this is the first study analyzing smoking with income, and both time and risk preferences, highlighting strong simultaneous effects between these outcomes. Until now, taxation among tobacco products is an efficient tool to reduce consumption among smokers, however this tool is uniform regardless individual characteristics, which are also important. Preferences should not be considered in a uniform way between Europeans countries among individuals aged 50 and over. Thus, this chapter gives insights about the importance of individual heterogeneity on smoking choices.

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Appendix A

Appendix: Simultaneous equations model with non-linear and linear dependent variables on panel data

Abstract

Multi-equations systems are at the heart of economic modeling. Researchers who want to establish causal links between two outcomes, often need to consider simultaneity between the latter, to overcome endogeneity issues (for instance when considering supply and demand equations). Difficulties arise when considering linear and non-linear outcomes at the same time and this is why [Roodman \[2011\]](#) implements the Stata module `cmp` for multidimensional models. In this research, we further develop this technique to allow the implementation of a simultaneous equations model in a panel dimension setting. Implemented under Stata, our method, `xtcmp`, is a Full Information Maximum Likelihood estimator. This research explains the associated theory (derivation of the log-likelihood function, the associated gradient and the Hessian matrices of the log-integrand function) and offers an application of `xtcmp`, while making comparisons with `cmp`.

Keywords: Causality; Full Information Maximum Likelihood; panel data; Gauss-Hermite quadrature; gradient; Hessian.

JEL Classification: C50; C51; C61; C63.

A.1 Introduction

In empirical economics, a common approach is to consider a linear data-generating process. However, non-linear data are often present and important in research questions. This is due to the structure of the database where interviewers transcribe yes-no answers into binary outcomes. However, when researchers point out a project, they often have to take into account different variables, linear and non-linear, at the same time, while considering simultaneous equations framework in a dynamic setting, in which each dependent variable is endogenous in one (or more) equations of the model. The advantage of simultaneous equations model is to consider the correlation between the error terms of each equation. More specifically, in a dynamic setup, such models allow researchers to consider different individual effects (which are part of error terms, the latter being decomposed into an individual effect fixed across time and an effect which depends on time) across equations. This is of importance since these terms are unobserved, specific to each outcome, and might imply endogeneity issues. For instance, in health economics, when investigating causal relationship between health and income, this can run from income to health and from health to income such that both are endogenous to each other ([Adeline and Delattre \[2018\]](#)). In this way, considering a dynamic simultaneous equation model allows to consider unobserved individual effects such as physical maturity (thanks to genetics for health) or intellectual abilities (for income).

There is almost no automated estimation method in Stata software to estimate parameters in a multi-equations model. An exception is the `cmp` command which is the first general Stata tool for this class of models, and is written as a Seemingly Unrelated Regressions (SUR) estimator ([Roodman \[2011\]](#)). However, this command does not explicitly consider the panel dimension of the data which might be an issue due to the effervescence of databases with a temporal dimension. Moreover, simple relationships among variables at a point in time do not capture adequately the dynamic interaction of changing humans in changing environments. Thus, there is a need to develop a command for simultaneous equations model for panel database.

As a result, we offer an extension of the `cmp` framework, in a case where there is either two equations (one linear and one binary outcomes) or three equations (either two linear dependent variables with one binary, or one linear and two binary dependent variables), while explicitly considering the panel dimension of the data. In this way, our command `xtcmp` is a FIML estimator, taking into account time dimension of the data, as well as, linear and non-linear outcomes (which is not feasible with three-stage least squares, because the latter only takes into account linear dependent variables).

As a result, the likelihood function is a multidimensional integral, such that we use the adaptive Gauss-Hermite quadrature method as an approximation (as proposed by [Liu and Pierce \[1994\]](#)). For the accuracy of the method and to reduce computing time, we derive the gradient of the log-likelihood, and the Hessian of the respective integrand. The estimation method of `xtcmp` has been implemented using the `d1` method of Stata software (see [Gould et al. \[2010\]](#) for further details).

Section [A.2](#) derives the likelihood for a FIML estimator in a general setting, as well as, for three specific cases. Section [A.3](#) discusses the estimation requirements needed, and details the Hessian matrices, as well as the gradient vector according to the parameters. In section [A.4](#), we give some examples on the use of `xtcmp`, while making comparisons with [Roodman \[2011\]](#)'s `cmp` results. Section [A.5](#) concludes this appendix.

A.2 Likelihood for a FIML estimator

Let Y denotes a d -dimensional vector of endogenous variables in a simultaneous equations model. [Roodman \[2011\]](#) specifically discusses conditions for consistency and identification of such model. Let Y^k denotes the k^{th} component of Y such that the value of Y^k for individual i at period t is given by y_{it}^k . We assume that the first d_1 components of Y , where $d_1 < d$, are binary outcomes, and the others, $d_2 = d - d_1$, are continuous. Let ϵ denotes the vector of associated error terms. By assuming a panel random effects model, ϵ can be decomposed into two terms such that $\epsilon = \mu + \nu$, where μ is time-invariant. In this way, the error term for individual i at period t in the k^{th} equation is given by $\epsilon_{it}^k = \mu_i^k + \nu_{it}^k$. The full model can be written as follows:

$$\tilde{Y} = X\beta + \epsilon$$

where \tilde{Y} contains the related latent variables for the first d_1 equation and the original continuous variables for the others. The explanatory matrix X is given by $X = \text{diag}(Z^k)$ where Z^k , corresponds to the explanatory variables for the k^{th} equation (with $k = 1, \dots, d$). Similarly, the parameter vector is $\beta = (\beta_1, \dots, \beta_d)'$ where β^k is the parameter vector of the k^{th} equation. We suppose that the classical hypotheses on independence between (i) the error components, and (ii) the error components and explanatory variables, are satisfied. Furthermore, let us assume that the error components are independent and identically distributed with zero means and covariance matrices Σ_μ and Σ_ν , the latter being defined as follows:

$$\Sigma_\nu = \begin{pmatrix} \Sigma_1 & \Sigma_3' \\ \Sigma_3 & \Sigma_2 \end{pmatrix}$$

in which Σ_1 is a d_1 dimension matrix with 1 over the diagonal (which corresponds to the covariance matrix structure for simultaneous equations with only binary outcomes), Σ_2 is a d_2 dimension matrix, and Σ_3 is a $d_2 * d_1$ dimension matrix. Thus, the overall individual likelihood is given by:

$$L_i = \int_{\mathbb{R}^d} \left\{ \prod_{t=1}^{T_i} f_{\nu}(\nu_{it}^1, \dots, \nu_{it}^d | \mu_i^1, \dots, \mu_i^d) \right\} f_{\mu}(\mu_i^1, \dots, \mu_i^d) d\mu_i^1, \dots, d\mu_i^d \quad (\text{A.1})$$

where $f_{\mu}(\mu_i^1, \dots, \mu_i^d) = \frac{1}{(2\pi)^{d/2} \sqrt{\det(\Sigma_{\mu})}} \exp\left(\frac{-1}{2} \mu' \Sigma_{\mu}^{-1} \mu\right)$. The d_1 first equations being related to binary outcomes, if we define $q_{it}^k = 2 * y_{it}^k - 1$, then, the density function $f_{\nu}(\nu_{it}^1, \dots, \nu_{it}^d | \mu_i^1, \dots, \mu_i^d)$ is given by¹:

$$\ell_{it} = f_{\nu}(\nu_{it}^1, \dots, \nu_{it}^d | \mu_i^1, \dots, \mu_i^d) = \int_{-\infty}^{q_{it}^1 z_{it}^1} \dots \int_{-\infty}^{q_{it}^{d_1} z_{it}^{d_1}} \phi_d(\nu_{it}^1, \dots, \nu_{it}^d) d\nu_i^1 \dots d\nu_i^{d_1}$$

where $z_{it}^k = Z_{it}^k \beta^k + \mu_i^k$, with $k = 1, \dots, d$, and $\nu_{it}^k = \tilde{y}_{it}^k - z_{it}^k$, with $k = d_1 + 1, \dots, d$. The idiosyncratic error is $\nu = (\nu_1, \dots, \nu_{d_1}, \nu_{d_1+1}, \dots, \nu_d) \sim \mathcal{N}(0, \Sigma_{\nu})$, such that $(\nu_{d_1+1}, \dots, \nu_d) \sim \mathcal{N}(0, \Sigma_2)$ and $(\nu_1, \dots, \nu_{d_1}) | (\nu_{d_1+1}, \dots, \nu_d) \sim \mathcal{N}(m_{(\nu_1, \dots, \nu_{d_1}) | (\nu_{d_1+1}, \dots, \nu_d)}, \Sigma_{(\nu_1, \dots, \nu_{d_1}) | (\nu_{d_1+1}, \dots, \nu_d)})$ with:

$$m_{d_1|d_2} = m_{(\nu_1, \dots, \nu_{d_1}) | (\nu_{d_1+1}, \dots, \nu_d)} = \Sigma_3' (\Sigma_2)^{-1} (\nu_{d_1+1}, \dots, \nu_d)'$$

and

$$\Sigma_{d_1|d_2} = \Sigma_{(\nu_1, \dots, \nu_{d_1}) | (\nu_{d_1+1}, \dots, \nu_d)} = \Sigma_1 - \Sigma_3' (\Sigma_2)^{-1} \Sigma_3$$

Thus, we have:

$$\ell_{it} = \phi_{d_2}(\nu_{it}^{d_1+1}, \dots, \nu_{it}^d) \Phi_{d_1}\left((q_{it}^1 z_{it}^1, \dots, q_{it}^{d_1} z_{it}^{d_1}), m_{d_1|d_2}, \Sigma_{d_1|d_2}\right)$$

in which $\Phi_{d_1}\left((q_{it}^1 z_{it}^1, \dots, q_{it}^{d_1} z_{it}^{d_1}), m_{d_1|d_2}, \Sigma_{d_1|d_2}\right)$ denotes the cumulative distribution function of a multivariate normal function with mean $m_{d_1|d_2}$ and a covariance matrix $\Sigma_{d_1|d_2}$.

We now focus on two cases. The first one is related to a simultaneous equations model with two outcomes, one binary and the other one continuous. Then, we focus on a case with three outcomes, composed of either one binary and two continuous variables, or two binary and one continuous variables.

1. The density function is denoted by ℓ_{it} for simplification.

A.2.1 Case with two outcomes: one binary and one continuous

Let us consider the two following equations:

$$\tilde{y}_{it}^1 = Z_{it}^1 \beta^1 + \epsilon_{it}^1 \quad (\text{A.2})$$

$$\tilde{y}_{it}^2 = Z_{it}^2 \beta^2 + \epsilon_{it}^2 \quad (\text{A.3})$$

in which y_{it}^1 is a binary variable equal to 1 if $\tilde{y}_{it}^1 > 0$ (considering chapter 2, this might refer to health), and $\tilde{y}_{it}^2 = y_{it}^2$ is a linear outcome (this might represent individual income).

The associated variance/covariance matrices of error components are:

$$\Sigma_\nu = \begin{pmatrix} 1 & \rho_1 \sigma \\ \rho_1 \sigma & \sigma^2 \end{pmatrix}$$

$$\Sigma_\mu = \begin{pmatrix} \sigma_1^2 & \rho_{1,2} \sigma_1 \sigma_2 \\ \rho_{1,2} \sigma_1 \sigma_2 & \sigma_2^2 \end{pmatrix}$$

By identification, we have $\Sigma_1 = 1$, $\Sigma_2 = \sigma^2$, and $\Sigma_3 = \rho_1 \sigma$. Thus, $m_{\nu_1|\nu_2} = \frac{\rho_1}{\sigma} \nu_2$ and $\Sigma_{\nu_1|\nu_2} = 1 - \rho_1^2$. In this way, the likelihood has the following form:

$$L_i = \int_{\mathbb{R}^2} \left\{ \prod_{t=1}^{T_i} f_\nu(\nu_{it}^1, \nu_{it}^2 | \mu) \right\} f_\mu(\mu_i^1, \mu_i^2) d\mu_i^1 d\mu_i^2$$

$$= \int_{\mathbb{R}^2} \left\{ \prod_t \ell_{it} \right\} f_\mu(\mu_i^1, \mu_i^2) d\mu_i^1 d\mu_i^2$$

in which ℓ_{it} is the individual likelihood:

$$\ell_{it} = \phi_1(\nu_{it}^2, 0, \sigma^2) \Phi_1\left(\frac{q_{it}^1 z_{it}^1 - \frac{\rho_1}{\sigma} \nu_{it}^2}{\sqrt{1 - \rho_1^2}}\right) \quad (\text{A.4})$$

A.2.2 Case with three outcomes: one binary and two continuous

Let us consider the two previous equations (equations A.2 and A.3), and a new one:

$$\tilde{y}_{it}^3 = Z_{it}^3 \beta^3 + \epsilon_{it}^3 \quad (\text{A.5})$$

also corresponding to a linear equation with $\tilde{y}_{it}^3 = y_{it}^3$. The associated variance/covariance matrices of error components are:

$$\Sigma_\nu = \begin{pmatrix} 1 & \rho_1\sigma_a & \rho_2\sigma_b \\ \rho_1\sigma_a & \sigma_a^2 & \rho_3\sigma_a\sigma_b \\ \rho_2\sigma_b & \rho_3\sigma_a\sigma_b & \sigma_b^2 \end{pmatrix}$$

$$\Sigma_\mu = \begin{pmatrix} \sigma_1^2 & \rho_{1,2}\sigma_1\sigma_2 & \rho_{1,3}\sigma_1\sigma_3 \\ \rho_{1,2}\sigma_1\sigma_2 & \sigma_2^2 & \rho_{2,3}\sigma_2\sigma_3 \\ \rho_{1,3}\sigma_1\sigma_3 & \rho_{2,3}\sigma_2\sigma_3 & \sigma_3^2 \end{pmatrix}$$

By identification, we have $\Sigma_1 = 1$, $\Sigma_2 = \begin{pmatrix} \sigma_a^2 & \rho_3\sigma_a\sigma_b \\ \rho_3\sigma_a\sigma_b & \sigma_b^2 \end{pmatrix}$, and $\Sigma_3 = (\rho_1\sigma_a, \rho_2\sigma_b)'$. Thus, $m_{\nu_1|(\nu_2, \nu_3)} = \frac{(\rho_1 - \rho_2\rho_3)\frac{\nu_2}{\sigma_a} + (\rho_2 - \rho_1\rho_3)\frac{\nu_3}{\sigma_b}}{1 - \rho_3^2}$ and $\Sigma_{\nu_1|(\nu_2, \nu_3)} = \frac{\rho_1^2 + \rho_2^2 - 2\rho_1\rho_2\rho_3}{1 - \rho_3^2}$. As a result, we can write the following likelihood:

$$L_i = \int_{\mathbb{R}^3} \left\{ \prod_{t=1}^{T_i} f_\nu(\nu_{it}^1, \nu_{it}^2, \nu_{it}^3 | \mu) \right\} f_\mu(\mu_i^1, \mu_i^2, \mu_i^3) d\mu_i^1 d\mu_i^2 d\mu_i^3$$

$$= \int_{\mathbb{R}^3} \left\{ \prod_t \ell_{it} \right\} f_\mu(\mu_i^1, \mu_i^2, \mu_i^3) d\mu_i^1 d\mu_i^2 d\mu_i^3$$

in which ℓ_{it} is the individual likelihood, defined as:

$$\ell_{it} = \phi_2((\nu_{it}^2, \nu_{it}^3), 0, \Sigma_2) \Phi_1\left(\frac{q_{it}^1 z_{it}^1 - m_{\nu_1|(\nu_2, \nu_3)}}{\sqrt{\Sigma_{\nu_1|(\nu_2, \nu_3)}}}\right) \quad (\text{A.6})$$

A.2.3 Case with three outcomes: two binary and one continuous

In order to derive the likelihood function for a case with two binary and one continuous outcomes, let us consider equation A.2 for the first binary outcome and equation A.5 for the linear variable (considering chapter 3, these might represent the smoking status and the income respectively). To consider another binary outcome, let us redefine the following:

$$\tilde{y}_{it}^2 = Z_{it}^2 \beta^2 + \epsilon_{it}^2 \quad (\text{A.7})$$

where y_{it}^2 is a binary variable, equal to 1 if $\tilde{y}_{it}^2 > 0$ (in chapter 3, this is equal to 1 when an individual is risk averse). The associated variance/covariance matrices of the error components are:

$$\Sigma_\nu = \begin{pmatrix} 1 & \rho_1 & \rho_2\sigma \\ \rho_1 & 1 & \rho_3\sigma \\ \rho_2\sigma & \rho_3\sigma & \sigma^2 \end{pmatrix}$$

$$\Sigma_\mu = \begin{pmatrix} \sigma_1^2 & \rho_{1,2}\sigma_1\sigma_2 & \rho_{1,3}\sigma_1\sigma_3 \\ \rho_{1,2}\sigma_1\sigma_2 & \sigma_2^2 & \rho_{2,3}\sigma_2\sigma_3 \\ \rho_{1,3}\sigma_1\sigma_3 & \rho_{2,3}\sigma_2\sigma_3 & \sigma_3^2 \end{pmatrix}$$

By identification, we have $\Sigma_1 = \begin{pmatrix} 1 & \rho_1 \\ \rho_1 & 1 \end{pmatrix}$, $\Sigma_2 = \sigma^2$, and $\Sigma_3 = (\rho_2\sigma, \rho_3\sigma)'$. Thus, $m_{(\nu_1, \nu_2)|\nu_3} = (\frac{\rho_2}{\sigma}\nu_3, \frac{\rho_3}{\sigma}\nu_3)'$ and $\Sigma_{(\nu_1, \nu_2)|\nu_3} = \begin{pmatrix} 1 - \rho_2^2 & \rho_1 - \rho_2\rho_3 \\ \rho_1 - \rho_2\rho_3 & 1 - \rho_3^2 \end{pmatrix}$. The likelihood has the following form:

$$L_i = \int_{\mathbb{R}^3} \left\{ \prod_{t=1}^{T_i} f_\nu(\nu_{it}^1, \nu_{it}^2, \nu_{it}^3 | \mu) \right\} f_\mu(\mu_i^1, \mu_i^2, \mu_i^3) d\mu_i^1 d\mu_i^2 d\mu_i^3$$

$$= \int_{\mathbb{R}^3} \left\{ \prod_t \ell_{it} \right\} f_\mu(\mu_i^1, \mu_i^2, \mu_i^3) d\mu_i^1 d\mu_i^2 d\mu_i^3$$

in which ℓ_{it} is the individual likelihood:

$$\ell_{it} = \phi_1(\nu_{it}^3, 0, \sigma^2) \Phi_2\left(\frac{q_{it}^1 z_{it}^1 - \frac{\rho_2}{\sigma} \nu_{it}^3}{\sqrt{1 - \rho_2^2}}, \frac{q_{it}^2 z_{it}^2 - \frac{\rho_3}{\sigma} \nu_{it}^3}{\sqrt{1 - \rho_3^2}}; q_{it}^1 q_{it}^2 \rho\right) \quad (\text{A.8})$$

with $\rho = \frac{\rho_1 - \rho_2\rho_3}{\sqrt{(1 - \rho_2^2)(1 - \rho_3^2)}}$.

A.3 Estimation requirement

The likelihood function being a d -dimensional integral function, we use the Gauss-Hermite quadrature method (see [Moussa and Delattre \[2018\]](#)). Implementing this method requires to (i) compute the mode $\hat{\mu}$ of the log-integrand

$\log(f) = \log\left(\left\{\prod_{t=1}^{T_i} \ell_{it}\right\} f_\mu(\mu_i^1, \dots, \mu_i^d)\right)$ in $\mu = (\mu_i^1, \dots, \mu_i^d)$ and derive the Hessian matrix H at $\hat{\mu}$ with respect to μ ; and, (ii) derive the gradient of the overall likelihood function with respect to the parameters.

Let Q denotes the selected number of quadrature points, x denotes the Q dimension vector of quadrature nodes, and w denotes the Q dimension vector of quadrature weight. By applying the adaptive Gauss-Hermite quadrature ([Liu and Pierce \[1994\]](#)), the likelihood

function in equation A.1 can be rewritten as:

$$L_i = \sum_{k_1=1}^Q, \dots, \sum_{k_d=1}^Q w_{k_1}^* \dots w_{k_d}^* \left\{ \prod_{t=1}^{T_i} \ell_{it} \right\} f_{\mu}(\mu_i^1, \dots, \mu_i^d) \Big|_{\mu=x^*} \quad (\text{A.9})$$

in which $x^* = \hat{\mu} + \sqrt{2}H^{-1/2}x$ and $w^* = (w_1^* \dots w_d^*)' = 2^{d/2} \det(H^{-1/2}). \text{diag}(w'. \exp(x^2))$.

A.3.1 Hessian matrix at $\hat{\mu}$

Based on the expressions of ℓ_{it} for each case described in section A.2 (equations A.4, A.6, and A.8), we first need to write the associated log-integrand $\log(f)$ corresponding to each three cases. Then, we focus on the computation of the Hessian matrices, where we derive $\frac{\partial^2}{\partial \eta_i^k \partial \eta_i^j} \log(f)$, with $k, j = 1, \dots, d$.

Focusing on the first case with two equations, we have the following log-integrand:

$$\log(f) = \log(f_{\mu}(\mu_i^1, \mu_i^2)) + \sum_{t=1}^{T_i} \log(\phi_1(\nu_{it}^2, 0, \sigma^2)) + \sum_{t=1}^{T_i} \log\left(\Phi_1\left(\frac{q_{it}^1 z_{it}^1 - \frac{\rho_1}{\sigma} \nu_{it}^2}{\sqrt{1 - \rho_1^2}}\right)\right)$$

With the notation $b_{it} = \frac{q_{it}^1 z_{it}^1 - \frac{\rho_1}{\sigma} \nu_{it}^2}{\sqrt{1 - \rho_1^2}}$, we find:

$$\begin{aligned} \frac{\partial^2}{\partial (\eta_i^1)^2} \log(f) &= -\frac{1}{\sigma_1^2(1 - \rho_{1,2}^2)} + \frac{1}{(1 - \rho_1^2)} \sum_{t=1}^{T_i} \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) - \left(\phi_1(b_{it})\right)^2}{\left(\Phi_1(b_{it})\right)^2} \\ \frac{\partial^2}{\partial (\eta_i^2)^2} \log(f) &= -\frac{1}{\sigma_2^2(1 - \rho_{1,2}^2)} - \frac{T_i}{\sigma^2} + \frac{\rho_1^2}{\sigma^2(1 - \rho_1^2)} \sum_{t=1}^{T_i} \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) - \left(\phi_1(b_{it})\right)^2}{\left(\Phi_1(b_{it})\right)^2} \\ \frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) &= \frac{\rho_{1,2}}{\sigma_1 \sigma_2 (1 - \rho_{1,2}^2)} + \frac{\rho_1}{\sigma(1 - \rho_1^2)} \sum_{t=1}^{T_i} q_{it}^1 \frac{-b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it})\right)^2}{\left(\Phi_1(b_{it})\right)^2} \end{aligned}$$

Thus, the Hessian matrix is given by:

$$H = \begin{pmatrix} -\frac{\partial^2}{\partial (\eta_i^1)^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) \\ -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) & -\frac{\partial^2}{\partial (\eta_i^2)^2} \log(f) \end{pmatrix} \quad (\text{A.10})$$

Then, focusing on the case of three equations with one binary outcome, the log-integrand is given by:

$$\log(f) = \log(f_\mu(\mu_i^1, \mu_i^2, \mu_i^3)) + \sum_{t=1}^{T_i} \log(\phi_2((\nu_{it}^2, \nu_{it}^3), 0, \Sigma_2)) + \sum_{t=1}^{T_i} \log\left(\Phi_1\left(\frac{q_{it}^1 z_{it}^1 - m_{\nu_1|(\nu_2, \nu_3)}}{\sqrt{\Sigma_{\nu_1|(\nu_2, \nu_3)}}}\right)\right)$$

The associated derivatives, assuming $b_{it} = \frac{q_{it}^1 z_{it}^1 - m_{\nu_1|(\nu_2, \nu_3)}}{\sqrt{\Sigma_{\nu_1|(\nu_2, \nu_3)}}$, $a_1 = \sqrt{1 - \rho_{1,3}^2}$, $a_2 = \sqrt{1 - \rho_{2,3}^2}$, and $ra = \frac{\rho_{1,2} - \rho_{1,3}\rho_{2,3}}{a_1 a_2}$, are:

$$\begin{aligned} \frac{\partial^2}{\partial(\eta_i^1)^2} \log(f) &= -\frac{1}{\left(\sigma_1 a_1 \sqrt{(1 - ra^2)}\right)^2} - \frac{1}{(1 - \rho^2)(1 - \rho_2^2)} \sum_{t=1}^{T_i} \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it})\right)^2}{\left(\Phi_1(b_{it})\right)^2} \\ \frac{\partial^2}{\partial(\eta_i^2)^2} \log(f) &= -\frac{1}{\left(\sigma_2 a_2 \sqrt{(1 - ra^2)}\right)^2} - \frac{T_i}{(1 - \rho_3^2) \sigma_a^2} - \\ &\quad \frac{(\rho_1 - \rho_2 \rho_3)^2}{(1 - \rho^2)(1 - \rho_2^2) (\sigma_a (1 - \rho_3^2))^2} \sum_{t=1}^{T_i} \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it})\right)^2}{\left(\Phi_1(b_{it})\right)^2} \\ \frac{\partial^2}{\partial(\eta_i^3)^2} \log(f) &= -\frac{1 - \rho_{1,2}^2}{\left(\sigma_3 a_1 a_2 \sqrt{(1 - ra^2)}\right)^2} - \frac{T_i}{(1 - \rho_3^2) \sigma_b^2} - \\ &\quad \frac{(\rho_2 - \rho_1 \rho_3)^2}{(1 - \rho^2)(1 - \rho_2^2) (\sigma_b (1 - \rho_3^2))^2} \sum_{t=1}^{T_i} \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it})\right)^2}{\left(\Phi_1(b_{it})\right)^2} \end{aligned}$$

$$\begin{aligned}
\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) &= \frac{\rho_{1,2} - \rho_{1,3} \rho_{2,3}}{\sigma_1 \sigma_2 \left(a_1 a_2 \sqrt{(1 - r a^2)} \right)^2} - \\
&\quad \frac{\rho_1 - \rho_2 \rho_3}{\sigma_a (1 - \rho^2) (1 - \rho_2^2) (1 - \rho_3^2)} \sum_{t=1}^{T_i} q_{it}^1 \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it}) \right)^2}{\left(\Phi_1(b_{it}) \right)^2} \\
\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^3} \log(f) &= \frac{\rho_{1,3} - \rho_{1,2} \rho_{2,3}}{\sigma_1 \sigma_3 \left(a_1 a_2 \sqrt{(1 - r a^2)} \right)^2} - \\
&\quad \frac{\rho_2 - \rho_1 \rho_3}{\sigma_b (1 - \rho^2) (1 - \rho_2^2) (1 - \rho_3^2)} \sum_{t=1}^{T_i} q_{it}^1 \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it}) \right)^2}{\left(\Phi_1(b_{it}) \right)^2} \\
\frac{\partial^2}{\partial \eta_i^2 \partial \eta_i^3} \log(f) &= \frac{\rho_{2,3} - \rho_{1,2} \rho_{1,3}}{\sigma_2 \sigma_3 \left(a_1 a_2 \sqrt{(1 - r a^2)} \right)^2} + \frac{\rho_3 T_i}{(1 - \rho_3^2) \sigma_a \sigma_b} - \\
&\quad \frac{(\rho_2 - \rho_3 \rho_3)(\rho_1 - \rho_2 \rho_3)}{\sigma_a \sigma_b (1 - \rho^2) (1 - \rho_2^2) (1 - \rho_3^2)^2} \sum_{t=1}^{T_i} \frac{b_{it} \phi_1(b_{it}) \Phi_1(b_{it}) + \left(\phi_1(b_{it}) \right)^2}{\left(\Phi_1(b_{it}) \right)^2}
\end{aligned}$$

Thus, the Hessian matrix is given by:

$$H = \begin{pmatrix} -\frac{\partial^2}{\partial (\eta_i^1)^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^3} \log(f) \\ -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) & -\frac{\partial^2}{\partial (\eta_i^2)^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^2 \partial \eta_i^3} \log(f) \\ -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^3} \log(f) & -\frac{\partial^2}{\partial \eta_i^2 \partial \eta_i^3} \log(f) & -\frac{\partial^2}{\partial (\eta_i^3)^2} \log(f) \end{pmatrix} \quad (\text{A.11})$$

Finally, for the three equations with two binary outcomes case, the log-integrand can be written as:

$$\begin{aligned}
\log(f) &= \log(f_\mu(\mu_i^1, \mu_i^2, \mu_i^3)) + \sum_{t=1}^{T_i} \log(\phi_1(\nu_{it}^3, 0, \sigma^2)) + \\
&\quad \sum_{t=1}^{T_i} \log \left(\Phi_2 \left(\frac{q_{it}^1 z_{it}^1 - \frac{\rho_2}{\sigma} \nu_{it}^3}{\sqrt{1 - \rho_2^2}}, \frac{q_{it}^2 z_{it}^2 - \frac{\rho_3}{\sigma} \nu_{it}^3}{\sqrt{1 - \rho_3^2}}, q_{it}^1 q_{it}^2 \rho \right) \right)
\end{aligned}$$

Then, considering a_1 , a_2 and ra previously defined and the following notations:

$$\begin{aligned}
b_{it}^1 &= \frac{q_{it}^1 z_{it}^1 - \frac{\rho_2}{\sigma} \nu_{it}^3}{\sqrt{1 - \rho_2^2}} \\
b_{it}^2 &= \frac{q_{it}^2 z_{it}^2 - \frac{\rho_3}{\sigma} \nu_{it}^3}{\sqrt{1 - \rho_3^2}} \\
r_n &= \sqrt{(1 - \rho_2^2)} \\
r_m &= \sqrt{(1 - \rho_3^2)} \\
\rho &= \frac{\rho_1 - \rho_2 \rho_3}{r_n \cdot r_m} \\
p_{it}^1 &= q_{it}^1 \phi(b_{it}^1) \Phi\left(\frac{b_{it}^2 - q_{it}^1 q_{it}^2 \rho b_{it}^1}{\sqrt{1 - \rho^2}}\right) \\
p_{it}^2 &= q_{it}^2 \phi(b_{it}^2) \Phi\left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1 - \rho^2}}\right) \\
p_{it}^3 &= \frac{\rho_3}{\sigma \sqrt{(1 - \rho^2)}} \phi(b_{it}^2) \Phi\left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1 - \rho^2}}\right) + \frac{\rho_2}{\sigma} \phi(b_{it}^1) \Phi\left(\frac{b_{it}^2 - q_{it}^1 q_{it}^2 \rho b_{it}^1}{\sqrt{1 - \rho^2}}\right)
\end{aligned}$$

We find the following:

$$\begin{aligned}
\frac{\partial^2}{\partial(\eta_i^1)^2} \log(f) &= -\frac{a_2^2}{\left(\sigma_1 a_1 a_2 \sqrt{(1 - r_a^2)}\right)^2} + \\
&\quad \sum_{t=1}^{T_i} \frac{\frac{q_{it}^1}{r_n} \left(-b_{it}^1 p_{it}^1 - q_{it}^2 \rho \phi(b_{it}^1) \phi\left(\frac{b_{it}^2 - q_{it}^1 q_{it}^2 \rho b_{it}^1}{\sqrt{1 - \rho^2}}\right) \right) \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho) - (p_{it}^1)^2}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2} \\
\frac{\partial^2}{\partial(\eta_i^2)^2} \log(f) &= -\frac{a_1^2}{\left(\sigma_2 a_1 a_2 \sqrt{(1 - r_a^2)}\right)^2} + \\
&\quad \sum_{t=1}^{T_i} \frac{\frac{q_{it}^2}{r_m} \left(-b_{it}^2 p_{it}^2 - q_{it}^1 \rho \phi(b_{it}^2) \phi\left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1 - \rho^2}}\right) \right) \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho) - (p_{it}^2)^2}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2}
\end{aligned}$$

$$\begin{aligned}
\frac{\partial^2}{\partial(\eta_i^3)^2} \log(f) &= -\frac{1 - \rho_1^2}{\left(\sigma_3 a_1 a_2 \sqrt{(1 - r_a^2)}\right)^2} - \frac{T_i}{\sigma^2} - \sum_{t=1}^{T_i} \frac{(p_{it}^3)^2}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2} + \\
&\sum_{t=1}^{T_i} \frac{\frac{\rho_3}{\sigma a_2 \sqrt{(1 - \rho^2)}} \left(\left(\frac{\rho_2}{\sigma a_1} - q_{it}^1 q_{it}^2 \rho \frac{\rho_3}{\sigma a_2} \right) \phi(b_{it}^2) \phi\left(\frac{b_{it}^2 - q_{it}^1 q_{it}^2 \rho b_{it}^1}{\sqrt{1 - \rho^2}}\right) - \frac{\rho_3}{\sigma} b_{it}^2 p_{it}^2 \right) \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2} + \\
&\sum_{t=1}^{T_i} \frac{\frac{\rho_2}{\sigma a_1 \sqrt{(1 - \rho^2)}} \left(\left(\frac{\rho_3}{\sigma a_2} - q_{it}^1 q_{it}^2 \rho \frac{\rho_2}{\sigma a_1} \right) \phi(b_{it}^1) \phi\left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1 - \rho^2}}\right) - \frac{\rho_2}{\sigma} b_{it}^1 p_{it}^1 \right) \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2}
\end{aligned}$$

$$\begin{aligned}
\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) &= \frac{\rho_1 - \rho_2 \rho_3}{\sigma_1 \sigma_2 \left(a_1 a_2 \sqrt{(1 - r_a^2)}\right)^2} + \\
&\sum_{t=1}^{T_i} \frac{\frac{q_{it}^1 q_{it}^2}{r_n r_m \sqrt{(1 - \rho^2)}} \left(\phi(b_{it}^1) \phi\left(\frac{b_{it}^2 - q_{it}^1 q_{it}^2 \rho b_{it}^1}{\sqrt{1 - \rho^2}}\right) \right) \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho) - p_{it}^1 p_{it}^2}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2} \\
\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^3} \log(f) &= \frac{\rho_2 - \rho_1 \rho_3}{\sigma_1 \sigma_3 \left(a_1 a_2 \sqrt{(1 - r_a^2)}\right)^2} + \\
&\sum_{t=1}^{T_i} \frac{\frac{q_{it}^1}{r_n} \left(\frac{\rho_3}{\sigma r_m} - q_{it}^1 q_{it}^2 \rho \frac{\rho_2}{\sigma r_n} \right) \phi(b_{it}^1) \phi\left(\frac{b_{it}^2 - q_{it}^1 q_{it}^2 \rho b_{it}^1}{\sqrt{1 - \rho^2}}\right) - \frac{\rho_2}{\sigma} b_{it}^1 p_{it}^1}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2} \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho) - p_{it}^1 p_{it}^3 \\
\frac{\partial^2}{\partial \eta_i^2 \partial \eta_i^3} \log(f) &= \frac{\rho_3 - \rho_1 \rho_2}{\sigma_2 \sigma_3 \left(a_1 a_2 \sqrt{(1 - r_a^2)}\right)^2} + \\
&\sum_{t=1}^{T_i} \frac{\frac{q_{it}^2}{r_m} \left(\frac{\rho_2}{\sigma r_n} - q_{it}^1 q_{it}^2 \rho \frac{\rho_3}{\sigma r_m} \right) \phi(b_{it}^2) \phi\left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1 - \rho^2}}\right) - \frac{\rho_3}{\sigma} b_{it}^2 p_{it}^2}{\left(\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)\right)^2} \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho) - p_{it}^2 p_{it}^3
\end{aligned}$$

Thus, the Hessian matrix has the same form as the previous one (matrix [A.11](#)), in other words it is given by:

$$H = \begin{pmatrix} -\frac{\partial^2}{\partial(\eta_i^1)^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^3} \log(f) \\ -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^2} \log(f) & -\frac{\partial^2}{\partial(\eta_i^2)^2} \log(f) & -\frac{\partial^2}{\partial \eta_i^2 \partial \eta_i^3} \log(f) \\ -\frac{\partial^2}{\partial \eta_i^1 \partial \eta_i^3} \log(f) & -\frac{\partial^2}{\partial \eta_i^2 \partial \eta_i^3} \log(f) & -\frac{\partial^2}{\partial(\eta_i^3)^2} \log(f) \end{pmatrix} \quad (\text{A.12})$$

A.3.2 Gradient vector with respect to the parameters

Based on the likelihood function given by equation [A.9](#), parameters to estimate are β^k , with $k = 1, \dots, d$, and the associated covariance matrices Σ_μ and Σ_ν . Thus, the gradient

has to be calculated with respect to these parameters. The first order derivative of the log-likelihood function with respect to a parameter α , in the set of parameters, is given by:

$$\frac{\partial \log(L_i)}{\partial \alpha} = \sum_{k_1=1}^Q, \dots, \sum_{k_d=1}^Q \frac{\partial f / \partial \alpha}{L_i} \quad (\text{A.13})$$

Focusing on the three cases, we apply this formula to compute derivatives with respect to each parameter.

First, considering the two outcomes case, we need to consider the six following parameters: $\beta^1, \beta^2, \sigma, \sigma_1, \sigma_2, \rho_1$. As in subsection A.3.1, we consider the previously defined b_{it} , which is specific to the case with two outcomes, such that we have:

$$\begin{aligned} \frac{\partial f}{\partial \beta^1} &= f * \sum_{t=1}^{T_i} \frac{q_{it}^1 \phi_1(b_{it})}{\sqrt{(1 - \rho_1^2)} \Phi_1(b_{it})} \\ \frac{\partial f}{\partial \beta^2} &= f * \sum_{t=1}^{T_i} \left(\frac{\nu_{it}^2}{\sigma^2} + \frac{\rho_1 \phi_1(b_{it})}{\sigma \sqrt{(1 - \rho_1^2)} \Phi_1(b_{it})} \right) \\ \frac{\partial f}{\partial \log(\sigma)} &= f * \sum_{t=1}^{T_i} \left(-1 + \left(\frac{\nu_{it}^2}{\sigma} \right)^2 + \frac{\rho_1 \nu_{it}^2 \phi_1(b_{it})}{\sigma \sqrt{(1 - \rho_1^2)} \Phi_1(b_{it})} \right) \\ \\ \frac{\partial f}{\partial \log(\sigma_1)} &= f * \left(-1 + \frac{\left(\frac{\mu_i^1}{\sigma_1} \right)^2 - \rho_{1,2} \frac{\mu_i^1 \mu_i^2}{\sigma_1 \sigma_2}}{1 - \rho_{1,2}^2} \right) \\ \frac{\partial f}{\partial \log(\sigma_2)} &= f * \left(-1 + \frac{\left(\frac{\mu_i^2}{\sigma_2} \right)^2 - \rho_{1,2} \frac{\mu_i^1 \mu_i^2}{\sigma_1 \sigma_2}}{1 - \rho_{1,2}^2} \right) \\ \frac{\partial f}{\partial \log \left(\frac{1 + \rho_1}{1 - \rho_1} \right)^{1/2}} &= f \sum_{t=1}^{T_i} \frac{\left(q_{it}^1 \rho_1 z_{it}^1 - \frac{\nu_{it}^2}{\sigma} \right) \phi_1(b_{it})}{\sqrt{(1 - \rho_1^2)} \Phi_1(b_{it})} \\ &= f * \left(\rho_{1,2} - \frac{\rho_{1,2} \left(\frac{\mu_i^1}{\sigma_1} \right)^2 + \rho_{1,2} \left(\frac{\mu_i^2}{\sigma_2} \right)^2 - (1 + \rho_{1,2}^2) \frac{\mu_i^1 \mu_i^2}{\sigma_1 \sigma_2}}{1 - \rho_{1,2}^2} \right) \end{aligned}$$

Now, focusing on the case of three equations with one binary outcome, we consider notations associated to this case in subsection A.3.1 (b_{it} , a_2 , and ra). We compute derivatives

with respect to $\beta^1, \beta^2, \beta^3, \sigma_a, \sigma_b, \rho_1, \rho_2, \rho_3, \sigma_1, \sigma_2, \sigma_3, \rho_{1,2}, \rho_{1,3}$ and $\rho_{2,3}$, such that:

$$\begin{aligned}\frac{\partial f}{\partial \beta^1} &= f * \sum_{t=1}^{T_i} \frac{q_{it}^1 \phi_1(b_{it})}{\sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \\ \frac{\partial f}{\partial \beta^2} &= f * \sum_{t=1}^{T_i} \left(\frac{\frac{\nu_{it}^2}{\sigma_a^2} - \rho_3 \frac{\nu_{it}^3}{\sigma_a \sigma_b}}{1-\rho_3^2} + \frac{(\rho_1 - \rho_2 \rho_3) \phi_1(b_{it})}{\sigma_a \sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \right) \\ \frac{\partial f}{\partial \beta^3} &= f * \sum_{t=1}^{T_i} \left(\frac{\frac{\nu_{it}^3}{\sigma_b^2} - \rho_3 \frac{\nu_{it}^2}{\sigma_a \sigma_b}}{1-\rho_3^2} + \frac{(\rho_2 - \rho_1 \rho_3) \phi_1(b_{it})}{\sigma_b \sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \right)\end{aligned}$$

$$\begin{aligned}\frac{\partial f}{\partial \log(\sigma_a)} &= f * \sum_{t=1}^{T_i} \left(-1 + \frac{\left(\frac{\nu_{it}^2}{\sigma_a}\right)^2 - \rho_3 \frac{\nu_{it}^2 \nu_{it}^3}{\sigma_a \sigma_b}}{1-\rho_3^2} + \frac{(\rho_1 - \rho_2 \rho_3) \nu_{it}^2 \phi_1(b_{it})}{\sigma_a (1-\rho_3^2) \sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \right) \\ \frac{\partial f}{\partial \log(\sigma_b)} &= f * \sum_{t=1}^{T_i} \left(-1 + \frac{\left(\frac{\nu_{it}^3}{\sigma_b}\right)^2 - \rho_3 \frac{\nu_{it}^2 \nu_{it}^3}{\sigma_a \sigma_b}}{1-\rho_3^2} + \frac{(\rho_2 - \rho_1 \rho_3) \nu_{it}^3 \phi_1(b_{it})}{\sigma_b (1-\rho_3^2) \sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \right)\end{aligned}$$

$$\begin{aligned}\frac{\partial f}{\partial \log\left(\frac{1+\rho_1}{1-\rho_1}\right)^{1/2}} &= f * \sum_{t=1}^{T_i} \left((1-\rho_1^2) \frac{\left(\frac{\rho_3 \frac{\nu_{it}^3}{\sigma_b} - \frac{\nu_{it}^2}{\sigma_a}}{1-\rho_3^2} + b_{it} \sqrt{(1-\rho^2)(1-\rho_2^2)} \frac{\rho^2}{(1-\rho^2)(\rho_1-\rho_2\rho_3)} \right) \phi_1(b_{it})}{\sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \right) \\ \frac{\partial f}{\partial \log\left(\frac{1+\rho_2}{1-\rho_2}\right)^{1/2}} &= f * \sum_{t=1}^{T_i} \left(\frac{\left(\frac{1-\rho_2^2}{1-\rho_3^2} \left(\rho_3 \frac{\nu_{it}^2}{\sigma_a} - \frac{\nu_{it}^3}{\sigma_b} \right) + b_{it} \sqrt{(1-\rho^2)(1-\rho_2^2)} \left(\rho_2 + \frac{(\rho_1 \rho_2 - \rho_3) \rho^2}{(1-\rho^2)(\rho_1-\rho_2\rho_3)} \right) \right) \phi_1(b_{it})}{\sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})} \right) \\ \frac{\partial f}{\partial \log\left(\frac{1+\rho_3}{1-\rho_3}\right)^{1/2}} &= f * \sum_{t=1}^{T_i} \left(\rho_3 \left(1 - \frac{\left(\frac{\nu_{it}^2}{\sigma_a}\right)^2 + \left(\frac{\nu_{it}^3}{\sigma_b}\right)^2}{1-\rho_3^2} \right) + (1+\rho_3^2) \frac{\frac{\nu_{it}^2 \nu_{it}^3}{\sigma_a \sigma_b}}{1-\rho_3^2} \right) + \\ &\quad f * \sum_{t=1}^{T_i} \frac{\left(\frac{\rho_2 \nu_{it}^2}{\sigma_a} + \frac{\rho_1 \nu_{it}^3}{\sigma_b} - 2\rho_3 \frac{(\rho_1-\rho_2\rho_3) \frac{\nu_{it}^2}{\sigma_a} + (\rho_2-\rho_1\rho_3) \frac{\nu_{it}^3}{\sigma_b}}{1-\rho_3^2} + \frac{b_{it} \sqrt{(1-\rho^2)(1-\rho_2^2)} (\rho_1 \rho_3 - \rho_2) \rho^2}{(1-\rho^2)(\rho_1-\rho_2\rho_3)} \right) \phi_1(b_{it})}{\sqrt{(1-\rho^2)(1-\rho_2^2)} \Phi_1(b_{it})}\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log(\sigma_1)} &= f * \left(-1 + \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1}\right)^2 - (\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} - (\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3}}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right) \\
\frac{\partial f}{\partial \log(\sigma_2)} &= f * \left(-1 + \frac{(1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2}\right)^2 - (\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} - (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right) \\
\frac{\partial f}{\partial \log(\sigma_3)} &= f * \left(-1 + \frac{(1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3}\right)^2 - (\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} - (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right)
\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log \left(\frac{1+\rho_{1,2}}{1-\rho_{1,2}} \right)^{1/2}} &= f * \left(\frac{1 - \rho_{1,2}^2}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \left(\rho_{1,2} \left(\frac{\mu_3^i}{\sigma_3}\right)^2 + \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} - \rho_{2,3} \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} - \rho_{1,3} \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3} \right) + \right. \\
&\quad f \frac{(1 - \rho_{1,2}^2)(\rho_{1,2} - \rho_{1,3}\rho_{2,3})}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \left(1 - \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1}\right)^2 + (1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2}\right)^2}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right) + \\
&\quad f \frac{(1 - \rho_{1,2}^2)(\rho_{1,2} - \rho_{1,3}\rho_{2,3})}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \left(\frac{-(1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3}\right)^2 + 2(\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2}}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right) + \\
&\quad f \frac{(1 - \rho_{1,2}^2)(\rho_{1,2} - \rho_{1,3}\rho_{2,3})}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \left(2 \frac{(\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} + (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right) \\
\frac{\partial f}{\partial \log \left(\frac{1+\rho_{1,3}}{1-\rho_{1,3}} \right)^{1/2}} &= f * \left(\frac{(1 - \rho_{1,3}^2)(\rho_{1,3} - \rho_{1,2}\rho_{2,3}) + \left(\rho_{1,3} \left(\frac{\mu_2^i}{\sigma_2}\right)^2 - \rho_{2,3} \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} - \rho_{1,2} \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3} \right) a_1^2}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} \right) - \\
&\quad f \left(\rho_{1,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{1,3} - \rho_{2,3}) \right) * \\
&\quad \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1}\right)^2 + (1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2}\right)^2 + (1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3}\right)^2}{(a_1 a_2 \sqrt{(1 - ra^2)})^2} + \\
&\quad 2f \left(\rho_{1,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{1,3} - \rho_{2,3}) \right) * \\
&\quad \frac{(\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + (\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} + (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{(1 - ra^2)})^2}
\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log \left(\frac{1+\rho_{2,3}}{1-\rho_{2,3}} \right)^{1/2}} &= f * \left(\frac{(1 - \rho_{2,3}^2)(\rho_{2,3} - \rho_{1,2}\rho_{1,3}) + \left(\rho_{2,3} \left(\frac{\mu_1^i}{\sigma_1} \right)^2 - \rho_{1,3} \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3} - \rho_{1,2} \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} \right) a_2^2}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} \right) - \\
&f \left(\rho_{2,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{2,3} - \rho_{1,3}) \right) * \\
&\frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1} \right)^2 + (1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2} \right)^2 + (1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3} \right)^2}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} + \\
&2f \left(\rho_{2,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{2,3} - \rho_{1,3}) \right) * \\
&\frac{(\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + (\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} + (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2}
\end{aligned}$$

Finally, we compute derivatives with respect to β^1 , β^2 , β^3 , σ , ρ_1 , ρ_2 , ρ_3 , σ_1 , σ_2 , σ_3 , $\rho_{1,2}$, $\rho_{1,3}$ and $\rho_{2,3}$ for the three equations with two binary outcomes case. To do so, we consider notations defined for this case in subsection A.3.1 concerning b_{it}^1 , b_{it}^2 , a_1 , a_2 , ra , r_n , r_m , ρ , p_{it}^1 , and p_{it}^2 , such that:

$$\begin{aligned}
\frac{\partial f}{\partial \beta^1} &= f * \sum_{t=1}^{T_i} \frac{p_{it}^1}{\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \\
\frac{\partial f}{\partial \beta^2} &= f * \sum_{t=1}^{T_i} \frac{p_{it}^2}{\Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \\
\frac{\partial f}{\partial \beta^3} &= f * \sum_{t=1}^{T_i} \left(\frac{\nu_{it}^3}{\sigma^2} + \frac{\rho_3 p_{it}^2}{\sigma q_{it}^2 \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} + \frac{\rho_2 p_{it}^1}{\sigma q_{it}^1 \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \right) \\
\frac{\partial f}{\partial \log(\sigma)} &= f * \sum_{t=1}^{T_i} \left(-1 + \left(\frac{\nu_{it}^3}{\sigma} \right)^2 + \frac{\rho_3 \nu_{it}^3 p_{it}^2}{\sigma q_{it}^2 \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} + \frac{\rho_2 \nu_{it}^3 p_{it}^1}{\sigma q_{it}^1 \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \right)
\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log \left(\frac{1+\rho_1}{1-\rho_1} \right)^{1/2}} &= f * \sum_{t=1}^{T_i} \frac{q_{it}^1 q_{it}^2 (1 - \rho_1^2) \phi(b_{it}^2) \phi \left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1-\rho^2}} \right)}{r_n r_m \sqrt{1 - \rho^2} \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \\
\frac{\partial f}{\partial \log \left(\frac{1+\rho_2}{1-\rho_2} \right)^{1/2}} &= f * \sum_{t=1}^{T_i} \left(\frac{q_{it}^1 q_{it}^2 (\rho_1 \rho_2 - \rho_3) \phi(b_{it}^2) \phi \left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1-\rho^2}} \right)}{r_n r_m \sqrt{1 - \rho^2} \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} + \frac{(\rho_2 b_{it}^1 - \frac{r_n \nu_{it}^3}{\sigma}) p_{it}^1}{q_{it}^1 \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \right) \\
\frac{\partial f}{\partial \log \left(\frac{1+\rho_3}{1-\rho_3} \right)^{1/2}} &= f * \sum_{t=1}^{T_i} \left(\frac{q_{it}^1 q_{it}^2 (\rho_1 \rho_3 - \rho_2) \phi(b_{it}^2) \phi \left(\frac{b_{it}^1 - q_{it}^1 q_{it}^2 \rho b_{it}^2}{\sqrt{1-\rho^2}} \right)}{r_n r_m \sqrt{1 - \rho^2} \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} + \frac{(\rho_3 b_{it}^2 - \frac{r_m \nu_{it}^3}{\sigma}) p_{it}^2}{q_{it}^2 \Phi_2(b_{it}^1, b_{it}^2; q_{it}^1 q_{it}^2 \rho)} \right)
\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log(\sigma_1)} &= f * \left(-1 + \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_i^1}{\sigma_1} \right)^2 - (\rho_{1,2} - \rho_{1,3} \rho_{2,3}) \frac{\mu_i^1 \mu_i^2}{\sigma_1 \sigma_2} - (\rho_{1,3} - \rho_{1,2} \rho_{2,3}) \frac{\mu_i^1 \mu_i^3}{\sigma_1 \sigma_3}}{(a_1 a_2 \sqrt{1 - r a^2})^2} \right) \\
\frac{\partial f}{\partial \log(\sigma_2)} &= f * \left(-1 + \frac{(1 - \rho_{1,3}^2) \left(\frac{\mu_i^2}{\sigma_2} \right)^2 - (\rho_{1,2} - \rho_{1,3} \rho_{2,3}) \frac{\mu_i^1 \mu_i^2}{\sigma_1 \sigma_2} - (\rho_{2,3} - \rho_{1,2} \rho_{1,3}) \frac{\mu_i^2 \mu_i^3}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{1 - r a^2})^2} \right) \\
\frac{\partial f}{\partial \log(\sigma_3)} &= f * \left(-1 + \frac{(1 - \rho_{1,2}^2) \left(\frac{\mu_i^3}{\sigma_3} \right)^2 - (\rho_{1,3} - \rho_{1,2} \rho_{2,3}) \frac{\mu_i^1 \mu_i^3}{\sigma_1 \sigma_3} - (\rho_{2,3} - \rho_{1,2} \rho_{1,3}) \frac{\mu_i^2 \mu_i^3}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{1 - r a^2})^2} \right)
\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log \left(\frac{1+\rho_{1,2}}{1-\rho_{1,2}} \right)^{1/2}} &= f * \left(\frac{1 - \rho_{1,2}^2}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \left(\rho_{1,2} \left(\frac{\mu_3^i}{\sigma_3} \right)^2 + \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} - \rho_{2,3} \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} - \rho_{1,3} \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3} \right) \right) + \\
&\quad f \frac{(1 - \rho_{1,2}^2)(\rho_{1,2} - \rho_{1,3} \rho_{2,3})}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \left(1 - \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1} \right)^2 + (1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2} \right)^2}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \right) + \\
&\quad f \frac{(1 - \rho_{1,2}^2)(\rho_{1,2} - \rho_{1,3} \rho_{2,3})}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \left(\frac{-(1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3} \right)^2 + 2(\rho_{1,2} - \rho_{1,3} \rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2}}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \right) + \\
&\quad f \frac{(1 - \rho_{1,2}^2)(\rho_{1,2} - \rho_{1,3} \rho_{2,3})}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \left(2 \frac{(\rho_{1,3} - \rho_{1,2} \rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} + (\rho_{2,3} - \rho_{1,2} \rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{(a_1 a_2 \sqrt{(1 - r a^2)})^2} \right)
\end{aligned}$$

$$\begin{aligned}
\frac{\partial f}{\partial \log \left(\frac{1+\rho_{1,3}}{1-\rho_{1,3}} \right)^{1/2}} &= f * \left(\frac{(1 - \rho_{1,3}^2)(\rho_{1,3} - \rho_{1,2}\rho_{2,3}) + \left(\rho_{1,3} \left(\frac{\mu_2^i}{\sigma_2} \right)^2 - \rho_{2,3} \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} - \rho_{1,2} \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3} \right) a_1^2}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} \right) - \\
&\quad f \left(\rho_{1,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{1,3} - \rho_{2,3}) \right) * \\
&\quad \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1} \right)^2 + (1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2} \right)^2 + (1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3} \right)^2}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} + \\
&\quad 2f \left(\rho_{1,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{1,3} - \rho_{2,3}) \right) * \\
&\quad \frac{(\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + (\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} + (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} \\
\\
\frac{\partial f}{\partial \log \left(\frac{1+\rho_{2,3}}{1-\rho_{2,3}} \right)^{1/2}} &= f * \left(\frac{(1 - \rho_{2,3}^2)(\rho_{2,3} - \rho_{1,2}\rho_{1,3}) + \left(\rho_{2,3} \left(\frac{\mu_1^i}{\sigma_1} \right)^2 - \rho_{1,3} \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3} - \rho_{1,2} \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} \right) a_2^2}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} \right) - \\
&\quad f \left(\rho_{2,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{2,3} - \rho_{1,3}) \right) * \\
&\quad \frac{(1 - \rho_{2,3}^2) \left(\frac{\mu_1^i}{\sigma_1} \right)^2 + (1 - \rho_{1,3}^2) \left(\frac{\mu_2^i}{\sigma_2} \right)^2 + (1 - \rho_{1,2}^2) \left(\frac{\mu_3^i}{\sigma_3} \right)^2}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2} + \\
&\quad 2f \left(\rho_{2,3} \left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2 + (\rho_{1,2} - \rho_{1,3}\rho_{2,3})(\rho_{1,2}\rho_{2,3} - \rho_{1,3}) \right) * \\
&\quad \frac{(\rho_{1,2} - \rho_{1,3}\rho_{2,3}) \frac{\mu_1^i \mu_2^i}{\sigma_1 \sigma_2} + (\rho_{1,3} - \rho_{1,2}\rho_{2,3}) \frac{\mu_1^i \mu_3^i}{\sigma_1 \sigma_3} + (\rho_{2,3} - \rho_{1,2}\rho_{1,3}) \frac{\mu_2^i \mu_3^i}{\sigma_2 \sigma_3}}{\left(a_1 a_2 \sqrt{(1 - ra^2)} \right)^2}
\end{aligned}$$

A.4 Examples and comparisons with Roodman's command

In order to shed light on advantages and consistency of our method (`xtcmp`), we decide to implement examples using a dataset, previously used for `xtsur`², in Stata software. This database is an unbalanced panel database of 1,672 observations, corresponding to 142 individuals followed between 1990 and 2003. All explanatory variables used are quantitative and do not contain any missing values.

2. See [Nguyen \[2008\]](#) for more detailed information.

We implement two cases: (i) two equations system with one linear and one binary dependent variables; (ii) three equations system with two binary outcomes and a linear one. Indeed, let us consider the three following equations:

$$\tilde{y}_{it}^1 = \beta_0 + x1_{it}\beta_1 + x2_{it}\beta_2 + x3_{it}\beta_3 + x4_{it}\beta_4 + \mu_i^1 + \nu_{it}^1 \quad (\text{A.14})$$

$$\tilde{y}_{it}^2 = \gamma_0 + x4_{it}\gamma_1 + x6_{it}\gamma_2 + x7_{it}\gamma_3 + \mu_i^2 + \nu_{it}^2 \quad (\text{A.15})$$

$$\tilde{y}_{it}^3 = \alpha_0 + x7_{it}\alpha_1 + x9_{it}\alpha_2 + \mu_i^3 + \nu_{it}^3 \quad (\text{A.16})$$

where y_{it}^1 and y_{it}^2 are binary variables equal to 1 if $\tilde{y}_{it}^1 > 0$ and $\tilde{y}_{it}^2 > 0$; and where $\tilde{y}_{it}^3 = y_{it}^3$ is a linear outcome.

When considering the first case (two equations system) with one linear and one non-linear outcomes, we focus on the simultaneous estimation of equations [A.14](#) and [A.16](#). In this case, the associated variance/covariance matrices of the error components are:

$$\Sigma_\nu = \begin{pmatrix} 1 & \rho\sigma \\ \rho\sigma & \sigma^2 \end{pmatrix} \quad \text{and} \quad \Sigma_\mu = \begin{pmatrix} \sigma_1^2 & \rho_1\sigma_1\sigma_2 \\ \rho_1\sigma_1\sigma_2 & \sigma_2^2 \end{pmatrix}$$

We consider four estimation techniques: (i) each equation is estimated separately as a single panel equation; (ii) the two equations are estimated with **cmp**, while considering a pooled equation; (iii) the two equations are estimated with **cmp**, with a posterior estimate of random effects; and, (iv) the two equations are estimated with our method (**xtcmp**) presented in section [A.2](#). Results are presented in table [A.1](#).

Results suggest that, first, estimating equations separately is misleading since the covariance between idiosyncratic errors and individual random effects is not considered; and, the significance of the coefficients appears to be false. On the other hand, considering equations simultaneously aims at obtaining consistent estimates among the three tests performed (coefficients are closer to each others in the last three columns). However, one should notice that, when using **cmp** with random effects, estimation of the covariance matrices for both individual random effects and idiosyncratic errors is done after the estimation of the coefficients (post-estimation). Comparing with our results (**xtcmp**, last column), we can see that the variance of the individual effects seems to be overestimated in **cmp**'s case.

Then, we offer an example for the second case, a three equations system with two binary outcomes and one linear dependent variable, such that we consider equations [A.14](#), [A.15](#) and [A.16](#). In this case, the associated variance/covariance matrices of the error

components are:

$$\Sigma_\nu = \begin{pmatrix} 1 & \rho_1 & \rho_2\sigma \\ \rho_1 & 1 & \rho_3\sigma \\ \rho_2\sigma & \rho_3\sigma & \sigma^2 \end{pmatrix} \quad \text{and} \quad \Sigma_\mu = \begin{pmatrix} \sigma_1^2 & \rho_{1,2}\sigma_1\sigma_2 & \rho_{1,3}\sigma_1\sigma_3 \\ \rho_{1,2}\sigma_1\sigma_2 & \sigma_2^2 & \rho_{2,3}\sigma_2\sigma_3 \\ \rho_{1,3}\sigma_1\sigma_3 & \rho_{2,3}\sigma_2\sigma_3 & \sigma_3^2 \end{pmatrix}$$

For this example, we cannot provide the third estimation technique where we used `cmp` command with a posterior estimate of the random effects, because this test does not converge. However, we provide the other estimation techniques: (i) each equation is estimated separately as a single panel equation; (ii) the three equations are estimated with `cmp`, considering a pooled database; and, (iii) the three equations are estimated with our method (`xtcmp`). Results are provided in table A.2.

Results suggest that, as before, estimating equations separately leads to errors in the significance of coefficients, especially for the first outcome. Moreover, such method does not consider the covariance between idiosyncratic errors and individual random effects. On the other hand, considering equations simultaneously allows to obtain more consistent estimates (coefficient estimates seem closer in the last two columns, and significance of the latter is persistent along the two last columns).

Table A.1 – Two equations system with one linear and one non-linear outcomes

Variables	Separate equations	Cmp		Our method
		Pooled data	Random effects	xtcmp
<u>Binary outcome \tilde{y}_{it}^1</u>				
$x1$	0.024 (0.017)	−0.006 (0.006)	0.002 (0.022)	0.012 (0.008)
$x2$	0.257*** (0.024)	0.029*** (0.003)	0.048*** (0.005)	0.020*** (0.004)
$x3$	0.317** (0.123)	0.005 (0.006)	0.035 (0.022)	0.025 (0.027)
$x4$	−0.575*** (0.218)	−0.008 (0.011)	−0.058 (0.039)	−0.046 (0.047)
<i>Intercept</i>	−6.438*** (0.375)	−0.708*** (0.038)	1.107*** (0.134)	−2.715*** (0.054)
<u>Continuous outcome \tilde{y}_{it}^3</u>				
$x7$	−0.153*** (0.021)	−0.032* (0.017)	−0.104*** (0.015)	−0.042*** (0.016)
$x9$	0.038*** (0.004)	0.104*** (0.005)	0.036*** (0.004)	0.096*** (0.004)
<i>Intercept</i>	15.996*** (0.734)	10.057*** (0.509)	17.024*** (0.448)	7.431*** (0.478)
<u>Covariance matrix: individual effects</u>				
σ_1	3.864*** (0.214)		2.843*** (0.214)	2.070*** (0.126)
σ_2	6.024		5.706*** (0.179)	2.248*** (0.142)
ρ_1			0.514*** (0.025)	0.819*** (0.042)
<u>Covariance matrix: idiosyncratic errors</u>				

Table A.1 – Two equations system with one linear and one non-linear outcomes (continued)

Variables	Separate equations	Cmp		Our method xtcmp
		Pooled data	Random effects	
σ	2.659	6.772*** (0.117)	2.673*** (0.048)	6.472*** (0.118)
ρ		0.153*** (0.035)	0.034 (0.053)	−0.509*** (0.042)

***: 1% significant; **: 5% significant; *: 10% significant. Standard deviations are into parentheses below the coefficients.

Table A.2 – Three equations system with two binary outcomes

Variables	Separate equations	Cmp Pooled data	Our method xtcmp
<u>Binary outcome 1 \tilde{y}_{it}^1</u>			
$x1$	0.024 (0.017)	−0.007 (0.006)	0.039*** (0.015)
$x2$	0.257*** (0.024)	0.033*** (0.003)	0.231*** (0.004)
$x3$	0.317** (0.123)	0.007 (0.007)	0.045 (0.038)
$x4$	−0.575*** (0.218)	−0.011 (0.013)	−0.083 (0.067)
<i>Intercept</i>	−6.438*** (0.375)	−0.718*** (0.038)	−2.721*** (0.125)
<u>Binary outcome 2 \tilde{y}_{it}^2</u>			
$x4$	0.168*** (0.048)	0.007 (0.008)	0.135** (0.058)
$x6$	−0.107*** (0.014)	−0.049*** (0.003)	−0.020*** (0.004)
$x7$	−0.081*** (0.019)	−0.042*** (0.004)	−0.023*** (0.007)
<i>Intercept</i>	8.867*** (1.098)	4.292*** (0.263)	1.838*** (0.389)
<u>Continuous outcome \tilde{y}_{it}^3</u>			
$x7$	−0.153*** (0.021)	−0.034* (0.017)	−0.064*** (0.012)
$x9$	0.038*** (0.004)	0.101*** (0.005)	0.061*** (0.003)
<i>Intercept</i>	15.996*** (0.734)	10.262*** (0.517)	12.429*** (0.377)
<u>Covariance matrix: individual effects</u>			
σ_1	3.864*** (0.214)		24.123*** (1.518)
σ_2	3.029*** (0.342)		6.540*** (0.485)
σ_3	6.024		3.714*** (0.235)
$\rho_{1,2}$			0.209** (0.085)
$\rho_{1,3}$			0.238*** (0.085)
$\rho_{2,3}$			0.635*** (0.057)
<u>Covariance matrix: idiosyncratic errors</u>			

Table A.2 – Three equations system with two binary outcomes (continued)

Variables	Separate equations	Cmp Pooled data	Our method <code>xtcmp</code>
σ	2.659	6.776*** (0.118)	4.606*** (0.087)
ρ_1		0.247*** (0.046)	0.872*** (0.068)
ρ_2		0.113*** (0.040)	−0.863*** (0.027)
ρ_3		0.150*** (0.035)	−0.591*** (0.057)

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

Standard deviations are into parentheses below the coefficients.

A.5 Conclusion

`xtcmp` is a command implemented under Stata software. We focus on three main cases: (i) a simultaneous equations model with two equations (including one linear and one binary outcomes); (ii) a case with three equations composed of two linear and one binary outcomes; and, (iii) a three equations case with one linear and two binary dependent variables. This command further develops [Roodman \[2011\]](#)’s command `cmp` which does not explicitly consider the panel dimension of the data, nor simultaneous equations model since it is written as a SUR estimator. This technical note gives detailed description of the computations, namely likelihood functions, log-integrand associated, Hessian matrices and gradient vectors with respect to each parameter, specific to the three cases described above.

`xtcmp`’s estimation framework could be further developed in order to consider a broader range of non-linear outcomes (such as *ordered probit*, *multinomial probit* or *truncated framework*, for instance) or to consider much more equations simultaneously, in a dynamic setup.

Still, as it stands, `xtcmp` represents a significant development in Stata’s commands. Indeed, it allows researchers to resolve endogeneity issues in a panel dimension context by analyzing correlation in the error terms of the equations and thus specific individual effects depending on the outcomes.

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Résumé

Les politiques de réduction des inégalités de santé basées sur une politique de redistribution en matière de revenu ou de réduction des coûts d'accès aux soins pour les plus démunis sont communes à de nombreux pays. Ces politiques sont basées sur le fait qu'il existe une relation entre revenu individuel et état de santé. Cette thèse analyse cette relation à partir de l'enquête « Survey of Health Ageing and Retirement in Europe » qui concerne des Européens âgés de 50 ans et plus. Nous montrons que le revenu individuel est associé de manière positive et concave à la santé (hypothèse de revenu absolu), mais aussi que les inégalités de revenu au sein d'un pays affectent tous les individus de ce pays (version forte de l'hypothèse de l'inégalité des revenus). Les mécanismes sous-jacents de cette hypothèse montrent que pour réduire les inégalités de santé liées aux inégalités de revenu, les gouvernements doivent promouvoir les investissements en capital humain et social. Aussi, les individus sont sensibles au mode de vie suivi par la majorité des personnes. Par la suite, nous implémentons une analyse simultanée de la santé et du revenu à l'aide d'un estimateur du maximum de vraisemblance à information complète. La causalité bidirectionnelle du revenu et de la santé est mise en avant, ainsi que la présence de caractéristiques individuelles inobservables communes à ces derniers. Enfin, sur un exemple concret, celui des politiques de lutte contre le tabagisme, cette thèse analyse simultanément la consommation de tabac, le revenu des individus, et l'aversion au risque. Les résultats mettent en avant l'importance des préférences individuelles dans la décision de fumer. En effet, les fumeurs Européens âgés de 50 ans et plus sont orientés vers le présent, de telles sorts qu'ils ne considèrent pas les effets néfastes du tabac sur la santé ; et sont, soit averse au risque du fait de l'anxiété, ou, aiment le risque puisqu'ils acceptent d'altérer leur santé.

Mots-clés: Santé; revenu; inégalités; causalité; hétérogénéité individuelle; Europe.

Summary

In many countries, governments set public policies to reduce health inequalities, based on income redistribution or on reducing the costs associated to the access to care for the poorest individuals. These policies are based on the fact that there is a relationship between individual income and health status. This doctoral dissertation analyses the latter, using the survey "Survey of Health, Ageing, and Retirement in Europe", which considers Europeans aged 50 and over. We first show that individual income is positively related with health in a concave way (Absolute Income Hypothesis). Results also suggest that income inequalities in a country affect all members in this society (strong version of the Income Inequality Hypothesis). Then, the underlying mechanisms of the latter hypothesis show that to reduce income-related health inequalities, governments should promote investments in human and social capital. Moreover, individuals are sensitive in following the common cultural model of the standard of living. Thereafter, we implement a simultaneous analysis of health and income using a Full Information Maximum Likelihood estimator. This allows to highlight two-way causality of income and health, as well as, the presence of unobserved individual characteristics common to both outcomes. Finally, on a concrete example, the one of public policies against smoking, this doctoral dissertation simultaneously investigates smoking, income, and risk aversion. Results highlight the importance of individual preferences in the smoking decision. Indeed, European smokers aged 50 and over are present-oriented, such that they do not consider the harmful effects of smoking on health; and are, either risk averse because of anxiety, or, risk lover when they agree to take the risk of altering their health.

Keywords: Health; income; inequalities; causality; individual heterogeneity; Europe.