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**On the Socio-Economic Determinants of Frailty:
Findings from Panel and Retrospective Data
from SHARE**

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On the Socio-Economic Determinants of Frailty: Findings from Panel and Retrospective Data from SHARE

Nicolas Sirven^{a, b}

ABSTRACT : Recent studies on the demand for long-term care emphasised the role of frailty as a specific precursor of disability besides chronic diseases. Frailty is defined as vulnerable health status resulting from the reduction of individuals' reserve capacity. This medical concept is brought here in an economic framework in order to investigate the role social policies may play in preventing disability or maintaining life quality of people in a disablement process.

Using four waves of panel data from the *Survey on Health, Ageing, and Retirement in Europe* (SHARE), a frailty index is created as a count measure for five physiologic criteria (Fried model) for respondents aged 50+ in 10 European countries, between 2004 and 2011.

The longitudinal dimension is explored in two ways. First, differences in frailty dynamics over a seven-year-time period are analysed through variables that are relevant for social policy (income maintenance, housing adaptation, and prevention of social isolation) in a panel model for count data with fixed effects. Second, the individual fixed effects are decomposed by means of a random effects model with Mundlak specification. SHARE additional retrospective data on life history (SHARELIFE) are then used to investigate differences in frailty levels.

The results reveal the presence of various sources of social inequalities over the life-course. Social Protection Systems thus appear to play a major role in accompanying, preventing or reducing the frailty process. Several policy implications are suggested.

JEL CODES: I12, J14, C23

KEYWORDS: Demand for health, Long-term care, Income maintenance, Health prevention, Panel models for count data, Mundlak device

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Une analyse des déterminants socio-économiques de la fragilité des personnes âgées à partir des données de panel et rétrospectives de SHARE

Nicolas Sirven^{a, b}

RÉSUMÉ : Les études récentes sur la demande de soins de long-terme ont mis en évidence le rôle de la fragilité en tant que précurseur de la perte d'autonomie, indépendamment des maladies chroniques. La fragilité est définie comme un état de santé vulnérable résultant de la diminution de la réserve physiologique de la personne âgée. Ce concept gériatrique est ici mobilisé en population générale et dans un cadre économique afin d'analyser le rôle des politiques publiques dans la prévention et l'accompagnement des personnes âgées dans un processus de perte d'autonomie.

A partir des données de panel et rétrospectives de l'enquête SHARE entre 2004 et 2011, nous étudions les déterminants socio-économiques de la fragilité en Europe. Dans un modèle à effets fixes, les différences individuelles dans la dynamique de la fragilité sont analysées au regard de trois piliers de l'action sociale : politique de soutien au revenu, lutte contre l'isolement social, promotion de l'aménagement du logement. Les différences persistantes dans les niveaux de fragilité sont explorées en utilisant les données rétrospectives sur l'histoire de vie (SHARELIFE) dans un modèle à effets aléatoires avec une spécification à la Mundlak.

Les résultats principaux indiquent la présence d'inégalités sociales de santé sous différentes formes et à différentes époques de la vie. L'importance des systèmes de protection sociale en Europe est reconnue comme un moyen d'accompagner, voire de retarder l'évolution du processus de perte d'autonomie. Plusieurs considérations de politique publique sont discutées.

CODES JEL : I12, J14, C23

MOTS-CLEFS : Demande en santé, Soins de long-terme, Soutien au revenu, Prévention, Économétrie de panel, Spécification à la Mundlak

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*“Nothing in life is to be feared,
it is only to be understood.”*

Marie Curie

1. Introduction

The substantial increase in the number and the share of older people in Europe in the next decades¹ combined with mixed evidence regarding trends in healthy life expectancy² lead policy-makers to anticipate serious public health and economic issues (WHO, 2011; OECD, 2011, European Commission, 2012). Research though suggests that there is still room for public policies to meet the challenges of ageing populations, in particular because ageing processes are modifiable (Christensen *et al.*, 2009). Improvements in the functional status of elderly people could improve their quality of life, and help mitigate the rise in the demand for, and hence expenditure on, long-term care. These prospects plead for disability prevention and health promotion strategies for older Europeans (Heikkinen, 2003).

Efficient interventions to prevent, reduce, or accompany the process of loss of autonomy in the elderly population require extensive knowledge of the pathways to disability at old ages. A large body of research has demonstrated the importance of chronic diseases as the primary contributor to disability (e.g. Guccione *et al.*, 1994; Boulton *et al.*, 1996). Recent research on the determinants of disability have emphasised both (i) the role of frailty as a specific medical precursor of disability besides chronic diseases (*cf.* Landrum, Stewart & Cutler, 2009), and (ii) the influence of improved socio-economic outcomes in the reduction of disability levels during the period 1980-2000 (Schoeni, Freedman & Martin, 2009).

These findings bring in some hope, first because frailty is a “new” potential candidate for disability prevention and health promotion. Frailty is defined as vulnerable health status resulting from a multisystem reduction in older people’s reserve capacity (*cf.* Studinger, Marsiske & Baltes, 1995; Spini *et al.*, 2007). Evolution of the frailty process leads to adverse health outcomes (such as dependence, falls, need for long-term care, and death, e.g. Klein *et al.*, 2005; Bergman *et al.*, 2007). Frailty is a progressive condition that begins with a preclinical stage (Ferrucci *et al.*, 1996; Fried *et al.*, 2001) and allows for reversible pathways (Fried *et al.*, 2004), thus offering opportunities for early detection and prevention. Specific tools for frailty have already been developed to operationalize the concept for health care professionals (Romero-Ortuno *et al.*, 2010) and public health policies (Vermeulen *et al.*, 2011).

A second reason to be confident in the potential health gains from prevention lies in the yet unexplored relationship between social policies features and the frailty process – despite the above mentioned relevance of socio-economic situations in disability pa-

¹ According to the United Nations (UN, 2011), about 30% of the European population will be 65 years old or more by 2060. Similarly, Eurostat projected the number of people aged 80 years or over to almost triple from 21.8 million in 2008 to 61.4 million in 2060 (Giannakouris, 2008).

² The literature does not indicate any clear signs of a reduction in disability among older people in Europe: Dolbhammer & Kytir (2001), Nusselder (2003), Mor (2005), Fries (1980, 1989, 2005), Jagger *et al.* (2007), LaFortune & Balestat (2007), and Suhrcke, Fumagalli & Hancock (2010).

thways. Only a few recent studies paid attention to the role of socio-economic factors in the distribution of frailty (Szanton *et al.* 2011; Etman *et al.*, 2012) and they did not specifically consider the role social policies may play in reducing frailty or maintaining life quality of people in a frailty process.

There is thus a need to investigate the influence on frailty of variables that are relevant for social policy. Three main domains of intervention common to most social policies in Europe are under consideration – income maintenance, support for housing adaptation, and actions to prevent social isolation. Although they are believed to help reduce elders' vulnerability by “ensuring people reach later life with reserve, reducing the challenges they face in later life, and providing adequate compensatory supports” (Grundy, 2006), there is not yet empirical evidence on how these three components of social policy are related to frailty.

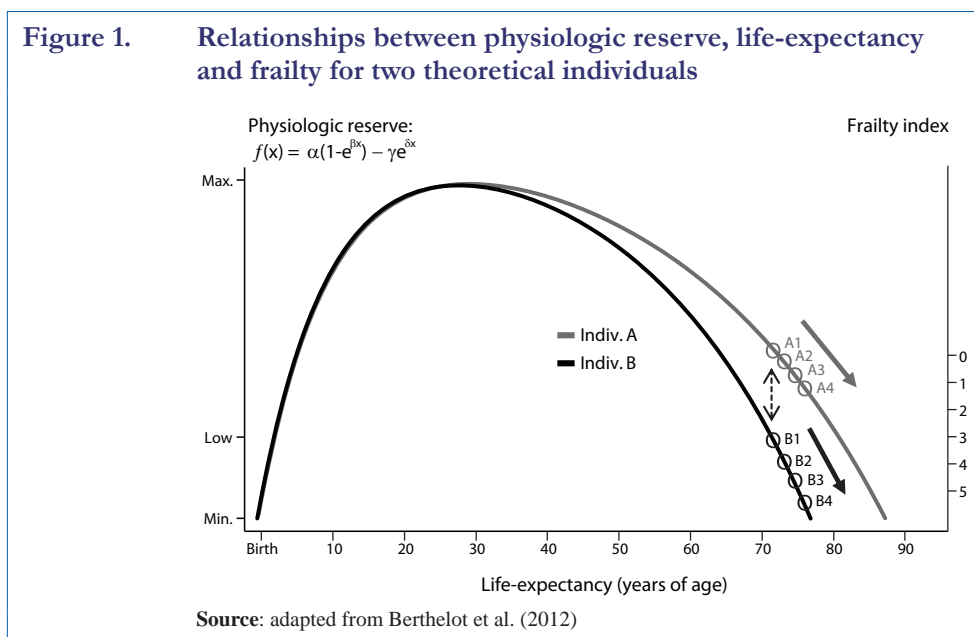
The present work contributes to the general knowledge of individual situations vis-à-vis the frailty process in the general population, and provides useful guidelines for social policy in Europe. Its ambition is to develop an economic analysis of frailty to provide pathways between the medical literature and the social sciences. More specifically, the following questions are addressed: What are the socio-economic determinants of the frailty process for older adults? What population should be targeted for frailty prevention, and to what extent Social Protection Systems provide adequate support for older adults to face challenges at early stages of the disablement process? This study makes use of individual panel data between 2004 and 2011 from the *Survey of Health, Ageing, and Retirement in Europe* (SHARE) for respondents aged 50 or more in 10 European countries. The longitudinal dimension is explored in two ways. First, differences in frailty dynamics over a seven-year-time period are analysed through variables that are relevant for social policy in a fixed effects model. Second, the individual fixed effects are decomposed by means of a random effects model with Mundlak specification. SHARE additional retrospective data on life history (SHARELIFE) are then used to investigate differences in frailty levels.

2. Conceptual framework

The medical approaches to frailty provide theoretical connections with the human capital model in standard economics. A general framework is proposed where older people's physiologic reserve is considered as health capital individuals bring to later life, and frailty is defined as a proxy for both concepts. The human capital model thus provides theoretical pathways between the frailty status and individual's socio-economic determinants.

2.1. Medical models of frailty

Separate models of frailty by Rockwood (Rockwood *et al.*, 1994) and Fried (Fried *et al.*, 2001) prevail in the health literature. Although they share certain similarities, they also have specific properties. The Rockwood model defines frailty as an accumulation of deficits resulting from multisystem physiologic or cognitive changes (Mitnitski, Mogilner & Rockwood, 2001; Rockwood & Mitnitski, 2007). Unlike the Fried criteria displayed below, the Rockwood model can incorporate the patient's mental health or psychosocial status so that the risk of adverse outcomes can be defined more precisely (Rockwood, Song & Mitnitski, 2011; Mitnitski, Fallah, Rockwood & Rockwood, 2011).



The Rockwood model is however typical of the “*black box epidemiology*” perspective since it makes use of a large set of criteria without a theoretical backdrop. This model does not distinguish between the concepts of disability and comorbidities. It is also difficult to implement due to the large number of variables (about 70 variables covering various dimensions of health, diseases, limitations, attitudes, behavioural risks, etc.), and requires additional clinical translation due to its complexity (Rockwood, Andrew & Mitnitski, 2007). The Fried approach to frailty is often preferred in the literature because (i) it is distinct from comorbidity and disability – providing new ways for research and intervention, (ii) it offers ready clinical operationalization, (iii) it is more parsimonious, quite straightforward and inexpensive to apply in general population surveys, and easily comparable across different settings.

The specificity of the Fried model lies in its strict focus on physiologic reserve (Fried *et al.*, 1994; Williamson & Fried, 1996). Leaving aside the cognitive dimension³, the trade-off between a comprehensive and coherent framework leans here in favour of the latter. The five only dimension of the Fried frailty index (shrinking, weakness, poor endurance and energy, slowness, low physical activity level) are derived from a set of logical pathways starting with senescent musculoskeletal changes leading to sarcopenia, and then to decreased strength and power, lower resting metabolic rate, reduction in total energy expenditure and thus chronic malnutrition, eventually reinforcing sarcopenia, and so on (Walston & Fried, 1999). Fried *et al.* (2001) bring into play “[t]his circle of frailty, representing an adverse, potentially downward spiral of energetics” to illustrate the process of reduction in individuals’ physiologic reserve (Walston, 2006).

³ Mitnitski, Fallah, Rockwood & Rockwood (2011) compare three measures of frailty as predictor to cognitive impairments: a frailty Index based on the Comprehensive Geriatric Assessment evaluated from 47 potential deficits, a Clinical Frailty Score and the Fried frailty phenotype. They found that Frail elderly people have an increased risk of cognitive decline. All frailty measures allowed quantification of individual vulnerability and predict both cognitive changes and mortality.

2.2. From frailty to the economic model of health capital

Figure 1 presents the theoretical relationships between unobserved physiologic reserve and life-expectancy for two theoretical individuals. A reversed scale displays Fried's index which counts the number of frailty criteria associated with individual's physiologic status at old ages. This index thus goes from 0 (non-frail or robust) to 5 (very frail). It is considered in the literature as a good proxy of the individual's physiologic reserve at old ages (Fried *et al.*, 2001, 2004). The general trend of the physiologic reserve follows a biphasic development with two antagonistic processes of increase before decline (Berthelot *et al.*, 2012). In the example, the two individuals, A and B, were born the same day and experienced comparable increases in their physiologic reserves until they reached a peak. Then the decline process started, being much stronger for B – for some reason – and creating a gap between the two physiologic reserves. Eventually B died before A. During the last period of their life, the frailty index appeared higher for B than for A. It has been argued elsewhere that the frailty index (whether Fried's or Rockwood's) is a good proxy for biological age (Mitnitski *et al.*, 2004; Schuurmans *et al.*, 2004).

From a health economics perspective, the physiologic reserve can be associated with the concept of health capital (Grossman, 1972, 2000; Bolin, 2011); the analogy is especially fitting when it comes to the decreasing process of the physiologic reserve. The standard economic theory considers that each individual is born with a certain amount of health capital, which depreciates with age, and is assumed to produce investments in health in order to align the realised amount of health with the demanded amount. Increasing resources have to be deployed in order to keep the stock of health at a certain level because the rate of depreciation increases over time.⁴ Age thus affects the demand for health by making the possession of a certain level of health capital more expensive. As a consequence, the model predicts that health decreases with age, and individuals with more resources have a higher ability to maintain their health stock. In our case, reference to the health capital theory helps bring into play individuals' socio-economic characteristics to explain the differences in the levels (the 'gaps' symbolised by the double arrow in dots in Fig. 1) and the dynamics (the 'slopes' symbolised by the two vectors in Fig. 1) of the physiologic reserves. In this context, Frailty is a proxy for both physiologic reserve and health capital at older ages. The frailty index is measured hereafter using panel data from SHARE.

3. Data

Empirical analyses are usually more relevant for prevention policies when they rely on general population settings. The options to define the working sample here meet this requirement and minimise the potential selection biases. Distribution of the frailty index in the working sample concurs with previous findings in the general population.

3.1. Sources and sample

The *Survey on Health, Ageing, and Retirement in Europe* (SHARE) is a multidisciplinary and cross-national cohort of individual data on health, socio-economic status and social and family relationships of more than 80,000 respondents aged 50 or over (*cf.* Börsch-Supan & Jürges, 2005). Eleven countries contributed to the 2004 SHARE baseline study

⁴ As indicated by the estimations of the theoretical physiologic reserve by Berthelot *et al.* (2012).

Table 1. Selected panel sample from SHARE

Country	Wave 1	Wave 2	Wave 3	Wave 4	Total
Austria	633	584	842	442	2,501
Germany	1,145	1,487	1,861	1,166	5,659
Sweden	1,478	1,415	1,945	1,181	6,019
Netherlands	1,427	1,629	2,202	1,433	6,691
Spain	1,088	1,238	2,094	1,075	5,495
Italy	1,398	1,955	2,496	1,642	7,491
France	1,443	1,696	2,459	1,426	7,024
Denmark	928	1,771	2,098	1,464	6,261
Switzerland	534	1,067	1,256	923	3,780
Belgium	2,274	2,232	2,803	1,866	9,175
Total	12,348	15,074	20,056	12,618	60,096

Table 2. Unbalanced sample features in regular panel waves

Country	Repeated observations in three regular panel waves			
	Once only	Twice	Three times	Total
Austria	111	522	1,026	1,659
Germany	269	1,456	2,073	3,798
Sweden	223	1,298	2,553	4,074
Netherlands	263	1,616	2,610	4,489
Spain	363	1,340	1,698	3,401
Italy	358	1,772	2,865	4,995
France	377	1,662	2,526	4,565
Denmark	297	1,880	1,986	4,163
Switzerland	134	1,220	1,170	2,524
Belgium	256	1,556	4,560	6,372
Total	2,651	14,322	23,067	40,040

(Israel took also part in SHARE wave 1 only). They are a balanced representation of the various regions in Europe, ranging from Scandinavia (Denmark and Sweden) through Central Europe (Austria, France, Germany, Switzerland, Belgium, and the Netherlands) to the Mediterranean (Spain, Italy and Greece). Further data were collected in 2006-07 during the second wave of SHARE in these countries and The Czech Republic, Poland, and Ireland. SHARELIFE, the third wave of the project, was conducted in 2008-09 over the same population (apart from Ireland). This time, the respondents were interviewed about their life history. Different fields such as childhood health, education, job career, family life, housing, etc. were surveyed and provide useful information on initial conditions and life course. In 2010, Estonia, Slovenia, Hungary, and Portugal joined SHARE wave 4, which is the third regular panel wave of the survey following the SHARELIFE life history questionnaire.

The sample retained here consists of 17,501 individuals corresponding to 60,096 observations, of which 40,040 belong to the regular panel waves 1, 2, or 4, and 20,056 come within SHARELIFE (Table 1). This sample is restricted to 10 countries in northern (Denmark, Sweden, the Netherlands) continental (Austria, Germany, France, Belgium,

Switzerland) and southern (Italy, Spain) regions of Europe. Excluded countries are those which (i) did not take part in SHARELIFE, making impossible to investigate frailty differences through the lenses of life-history; or (ii) did not carry out three waves of regular panel – the aim here is to avoid a systematic bias due to missing observations for some countries. Among these 10 countries, individuals retained in the sample were those interviewed in SHARELIFE (wave 3) and at least once in a regular panel wave (wave 1, 2, or 4). Finally, only full-rank data matrices are kept at each wave so that observations with missing data are deleted. These two latter rules contribute to consider an unbalanced panel in the analysis (Table 2). Notice that 93.4% of the sample is observed twice (N=14,322) or three times (N=23,067) in the regular panel waves. Only 6.6% of the sample (N=2,651) do not provide any information on the dynamics of the frailty process since they are observed only once besides SHARELIFE.

3.2. The frailty index

Previous studies using SHARE data derive a frailty index based on the five criteria from the Fried model (Santos-Eggiman *et al.*, 2009; Romero-Ortuno *et al.*, 2010; Etman *et al.*, 2012). Operationalization of these criteria required adaptation to the SHARE survey contents for which the definition by Santos-Eggimann *et al.* (2009) was used:

- *Exhaustion* was identified as a positive response to the question, “In the last month, have you had too little energy to do things you wanted to do? (yes/no).”
- *Shrinking* was fulfilled by reporting a “diminution in desire for food” in response to the question, “What has your appetite been like” or, in the case of an uncodable response to this question, by responding “less” to the following question: “So have you been eating more or less than usual?”
- *Weakness* was derived from the highest of four consecutive dynamometer measurements of handgrip strength (two from each hand), applying gender and body mass index cut-offs by quintiles of the distribution.
- *Slowness* was defined using mobility questions: “Because of a health problem, do you have difficulty [expected to last more than 3 months] walking 100 meters” or “... climbing one flight of stairs without resting”.
- *Low physical activity* was fulfilled in participants responding “one to three times a month” or “hardly ever or never” to the question, “How often do you engage in activities that require a low or moderate level of energy such as gardening, cleaning the car, or going for a walk?”

Following previous studies, one point was allocated for each fulfilled criterion. In that case, the frailty index is a score ranging from 0 to 5 where each criterion contributes to the score in the same way.⁵ It is standard practice in the literature to set cut-off points of this above frailty score: 0 non-frail or robust, 1-2 pre-frail, and 3-5 frail. Although this is especially useful for health care professionals, such an arbitrary dichotomy is not required here. In addition, using the variable without specified thresholds appears judicious to investigate frailty as a progressive condition.

The frailty index is available for the three regular panel waves in SHARE – since SHARELIFE did not gather sufficient measures to compute the index. The distribution

⁵ This particular assumption requires to be thoroughly investigated elsewhere. Using SHARE data, King-Kallimanis, Savva & Kenny (2012) found that while a single latent variable model for the Fried frailty phenotype is tenable, the factor loadings and thresholds are not invariant across all countries, suggesting that direct comparisons of the prevalence of frailty across countries may not be appropriate.

Table 3. Distribution of the frailty index between waves and gender
(Percentages displayed below headcounts)

Frailty Index	Wave 1			Wave 2			Wave 4			Total		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
0	3,653 64.8	2,649 39.5	6,302 51.0	4,433 63.0	3,213 39.9	7,646 50.7	3,391 58.3	2,343 34.5	5,734 45.4	11,477 62.1	8,205 38.1	19,682 49.2
1	1,414 25.1	2,192 32.6	3,606 29.2	1,810 25.7	2,630 32.7	4,440 29.5	1,561 26.8	2,206 32.4	3,767 29.9	4,785 25.9	7,028 32.6	11,813 29.5
2	400 7.1	1,104 16.4	1,504 12.2	554 7.9	1,295 16.1	1,849 12.3	526 9.0	1,212 17.8	1,738 13.8	1,480 8.0	3,611 16.8	5,091 12.7
3	130 2.3	492 7.3	622 5.0	178 2.5	567 7.0	745 4.9	229 3.9	625 9.2	854 6.8	537 2.9	1,684 7.8	2,221 5.5
4	34 0.6	228 3.4	262 2.1	50 0.7	273 3.4	323 2.1	93 1.6	330 4.9	423 3.4	177 1.0	831 3.9	1,008 2.5
5	3 0.1	49 0.7	52 0.4	6 0.1	65 0.8	71 0.5	19 0.3	83 1.2	102 0.8	28 0.2	197 0.9	225 0.6
Total	5,634 100	6,714 100	12,348 100	7,031 100	8,043 100	15,074 100	5,819 100	6,799 100	12,618 100	18,484 100	21,556 100	40,040 100

Table 4. Prevalence of 2 or more Fried criteria by gender and age class
(Percentages)

Country	Men					Women					Total				
	50-59	60-69	70-79	80+	Total	50-59	60-69	70-79	80+	Total	50-59	60-69	70-79	80+	Total
Austria	14.5	9.9	12.0	26.9	12.8	16.1	17.8	34.8	61.2	25.9	15.4	14.2	25.8	48.2	20.3
Germany	5.4	7.3	12.3	30.9	9.2	9.4	15.4	30.3	59.2	19.2	7.7	11.3	20.8	47.3	14.4
Sweden	3.8	4.6	9.0	24.6	7.5	13.7	14.5	28.9	58.8	21.7	9.3	10.0	19.5	42.4	15.1
Netherlands	6.4	7.0	11.0	21.6	8.8	12.9	16.8	30.3	49.7	20.4	10.1	12.2	20.9	36.3	15.0
Spain	9.4	15.0	26.5	47.4	19.7	30.6	45.9	64.1	81.2	49.9	21.3	31.9	46.0	67.3	36.3
Italy	9.1	12.3	21.3	39.9	15.9	26.8	34.4	57.2	76.9	39.7	19.6	24.1	38.3	58.8	28.6
France	8.0	9.2	22.0	41.8	14.5	20.2	29.0	44.0	70.0	34.2	14.5	20.2	34.2	59.3	25.4
Denmark	6.8	7.8	14.8	23.6	10.1	15.2	16.8	34.8	60.0	24.8	11.3	12.4	25.6	46.5	17.9
Switzerland	6.5	4.1	7.2	20.4	6.9	10.2	13.4	31.0	53.2	20.8	8.6	9.1	19.3	41.0	14.5
Belgium	7.6	9.0	18.4	33.8	13.1	17.4	25.2	43.1	62.3	31.0	12.7	17.6	31.5	49.6	22.6
Total	7.4	8.6	16.5	31.9	12.0	17.5	23.4	41.4	63.7	29.3	13.0	16.5	29.4	50.1	21.3

of the frailty index is hereafter broken by several main variables; time, age, gender, and country of residence (Tables 3-4). In the details, one may notice:

- *A small prevalence of extreme frailty.* Less than 1% of the sample is credited with the extreme value (5) of the frailty index at each wave. According to the thresholds given by the Fried model, only 8.6% of the population is “frail” over the whole period (frailty score ≥ 3). This is certainly due to the death-proximity of individuals with extreme frailty values.
- *However, more than 20% of the population aged 50 or more is potentially involved in a frailty process.* 21.3% of the sample is credited with at least two criteria of the Fried model over the three waves. Values of the frailty index increase (i) with time spent between waves (e.g. 19.7% of the sample have at least two Fried criteria in wave 1 while this figure rises up to 24.8% in wave 4) and (ii) between age cohorts (16.5% of the overall sample is credited with at least two Fried criteria between 60-69 compared to 31.5% for those 70-79 and up to 49.6% for those 80 or more).
- *Women have higher values of the frailty index than men.* 29.4% of women in the sample have a frailty score ≥ 2 compared to only 12.1% of men over the period. These results remain true when decomposed by countries; the general north-south gradient (Northern countries have lower values of the frailty index) is also more favourable for men when it comes to the frailty index.

These descriptive statistics are coherent with general findings from previous studies on frailty (e.g. Santos-Eggiman *et al.*, 2009). The *ceteris paribus* analysis of the socio-economic determinants of frailty aims at providing new evidence on the distribution and the evolution of the frailty process.

4. Methods

Econometric analysis of the frailty index depends on how the response variable is to be considered: it could be defined as (i) a count measure with regard to the number of fulfilled criteria, (ii) a fractional measure if one consider the rate of “successes” or “failures” out of the five binomial “trials” (the 5 Fried criteria), or even (iii) an ordered category response. The three options are reviewed, with special focus on the first one, because of the nice statistical properties of the fixed effects Poisson estimator. The different models are presented below, and their specification follows from the health capital theory and focuses on socio-economic variables that are relevant for social policy.

4.1. Econometric options

Econometric models for a count dependant variable are well-known in health economics when the data are cross-sectional. In comparison, panel data models for count variables are much less widespread in the literature. Just like in the linear case, fixed effects and random effects are competing alternatives, although rejoinder models using *Chamberlain’s device* (as suggested by Mundlak, 1978; and generalised by Chamberlain, 1982) can be extremely useful here.

4.1.1. Estimation strategy

Let y_{it} denote the discrete count frailty index of individual i , $i = 1, \dots, N$, at time t , $t = 1, \dots, T$; and let X_{it} denote the full-rank data matrix of explanatory variables. The

commonly used exponential model for panel count data assumes multiplicative unobserved heterogeneity:

$$E(y_{it} | X_{it}, c_i) = c_i \exp(X_{it}\beta) \quad (1)$$

where c_i is a permanent scaling factor for the individual specific term. If we suppose

$$c_i = \exp(e_i) \quad (2)$$

then, this implicitly defines a regression model

$$y_{it} = \exp(X_{it}\beta + e_i) + u_{it} \quad (3)$$

Several possible estimators of β are given in the literature depending on various sets of hypotheses (Wooldridge 2010: Chap 18). The econometric options retained below focus on two standard models to be estimated by Maximum Likelihood (ML), the Fixed Effects (FE) and the Random Effect (RE) Poisson models. The latter is a specific case of the former where structural hypotheses are added. Just like in the linear case, the RE model is built upon the FE model. The following assumptions are common to FE and RE Poisson models. First and foremost, the strict exogeneity assumption⁶ of the X_{it} conditional on c_i

$$E(y_{it} | X_{it}, c_i) = E(y_{it} | X_{i1}, \dots, X_{iT}, c_i). \quad (4)$$

Second, following pioneer work by Hausman, Hall & Grilliches (1984), two ancillary assumptions common to FE and RE are usually made, although they are not necessary in the case of FE

$$y_{it} | X_{it}, c_i \sim \text{Poisson}[c_i \exp(X_{it}\beta)] \quad (5)$$

$$y_{it}, y_{ir} \text{ are independent conditional on } x_{it}, c_i, \text{ with } t \neq r. \quad (6)$$

At this stage, the FE Poisson model can be estimated. The main advantages of this model come from the properties of (4) the strict exogeneity assumption. The FE estimator is consistent under (4) only, and the robust variance estimator is valid⁷ and allows for any dispersion from the Poisson distribution and arbitrary time dependence, so that (5) and (6) are not requested whenever (4) holds.⁸ The *Within* estimator of the FE Poisson

⁶ Notice that both FE and RE procedures hinge on the strict exogeneity assumption of X_{it} conditional on c_i . Since it is important to check whether $H_0: E(X_{it}c_i) = 0$ is true, an easy test can be implemented (Wooldridge, 2010: 18.7.4): (i) let W_{it} a subset of X_{it} which potentially fail the strict exogeneity assumption; (ii) include W_{it+1} as an additional set of covariates; (iii) under the null hypothesis of strict exogeneity, the coefficients on W_{it+1} should be statistically insignificant. See Blundell, Griffith & Windmeijer (2002) for count data models where the strict exogeneity assumption is relaxed.

⁷ When using short panels (T small), FE Poisson provides more efficient estimates than the RE model. In addition, ML estimation of the RE model is calculated using quadrature, which is an approximation whose accuracy depends partially on the number of integration points used.

⁸ Cameron & Trivedi (1998) note that one of the reasons for the failure of the Poisson regression in cross-section settings, is unobserved heterogeneity. Although neglected unobserved heterogeneity leads to over-dispersion and excess of zeros (Jones, Rice, Bago d'Uva & Balia, 2007), the use of FE Poisson with multiplicative unobserved heterogeneity in panel data is expected to work well whatever the distribution of the dependent variable: "Except for the conditional mean, the distribution of y_{it} need not be discrete; it could be continuous or have discrete and continuous features." (Wooldridge, 2010: 18.7.4) For instance, relaxing distributional assumptions (e.g. through Negative-Binomial modelling) did provide similar results as the FE Poisson.

model has very useful properties and is especially relevant for the analysis of the differences in the changes of the frailty index over time (differences in ‘slopes’ in Fig. 1).

Nevertheless, the FE Poisson model has two main drawbacks in our case: (i) individuals with $y_{it} = 0$ for all t are removed from the estimation sample because the conditional mean has an exponential form, and (ii) time-constant explanatory variables drop out of the equation – just like in the linear case. However, one may believe that people who never experienced frailty over the period 2004-2011 are of potential interest in the context of disability prevention strategies. The analysis would also benefit from some key variables like date of birth, gender and country of residence, and retrospective conditions. These latter variables would be “silent” in the FE model since the time-invariant effects are taken into account in c_i .

In order to overcome some of the restrictions inherent to the FE Poisson model, an improved version of the RE Poisson model is specified to decompose the individual fixed effects by means of time-invariant variables, and to include individuals for whom $y_{it} = 0$ over the period. Estimation of the standard RE Poisson model requires all the previous assumptions, specifically (5) and (6), plus the two additional assumptions below

$$E(c_i | X_{i1}, \dots, X_{iT}) = E(c_i) = 1 \quad (7)$$

$$c_i \text{ is independent of } X_{iT} \text{ and distributed as Gamma } (\delta, \delta) \quad (8)$$

Assumption (8) is the most controversial since it is likely that the unobserved fixed components c_i are correlated with the explanatory variables, $E(X_{it} c_i) \neq 0$, and therefore standard RE estimators will be inconsistent. It is though possible to “soften” assumption (8) by allowing c_i and \bar{X}_i to be correlated;

$$c_i = a_i \exp(\bar{X}_i \gamma) = \exp(\bar{X}_i \gamma + a_i) \text{ with } a_i = \exp(a_i) \text{ and } \bar{X}_i = \frac{1}{T} \sum_{t=1}^T X_{it} \quad (9)$$

In that case, the regression model becomes

$$y_{it} = \exp(X_{it} \beta + \bar{X}_i \gamma + a_i) + u_{it} \quad (10)$$

Equation (10) illustrates the implementation in a count data model of the Mundlak (1978) device. Estimation of β by ML is straightforward in the Mundlak model; it follows the standard procedure for a RE Poisson models in which the time averages of individual time-varying explanatory variables \bar{X}_i are included as additional regressors. Choice between the standard RE Poisson model and the Mundlak specification is also straightforward; a basic joint test of coefficients (Wald) for $H_0: \gamma = 0$ can be interpreted here as a Hausman test.

The main property of the Mundlak RE model is that the estimated coefficients of the time-varying explanatory variables are the same as those obtained with a FE model. In addition, the list of covariates X_{it} can now be extended to include time-invariant variables, and the estimation sample can be extended to individuals for whom $y_{it} = 0$ for all t . As a consequence, the Mundlak RE Poisson model is especially useful in our case because the *Within* estimator still can trace differences in frailty dynamics (‘slopes’) as in the FE model, while the *Between* estimator provides insights about the differences in the levels of frailty (‘gaps’) in the whole sample. Finally, the combination of the Mundlak RE Poisson Model and the unbalanced panel sample help considering a much larger set of individual situations within, and outside, the frailty process.

4.1.2. Strategy for robustness checks

The strength of the FE Poisson model rests on the fact that it provides consistent estimates and efficient robust standard errors under few assumptions (mainly strict exogeneity) and whatever the distribution of Y_{it} : “We must emphasise that, while the leading application is to count data, the FE Poisson estimator works whenever assumption [(4)] holds.” (Wooldridge, 2010: 18.7.4) Nevertheless, in order to gain confidence in the results provided by the previous Poisson models, alternative econometric options may be explored. In particular, one may not be totally satisfied (at least from a conceptual point of view) with a frailty index which is considered as a count variable. First, the response measure is bounded between 0 and 5, suggesting that a binomial distribution would provide additional interesting results (at least for the sceptics). Second, it could be that treating the frailty index as a continuous measure of physiological decline is excessively straightforward. Rather, one may acknowledge that the frailty index actually consists of six ordered categories (from 0 to 5) which depict the latent physiological reserve.

Let us consider first the frailty index as a ratio index counting the number of “successes” or “failures” at each of the five “trials” (i.e. criteria) considered in the Fried model. In that case, a new version of the frailty index would measure the rate of total “successes” or “failures” out of five trials, the response variable being then defined as $0 \leq y_{it} \leq 1$. Papke & Wooldridge (2008) considered a Pooled Fractional Probit model (PFP or PFProbit) to deal with this response variable in a panel setting. The functional form is given by:

$$E(y_{it} | X_{it}, c_i) = \Phi(X_{it}\beta + c_i) \quad (11)$$

where Φ is the the standard normal cumulative distribution function. Although the model is identifiable (through semi-parametric methods) under the strict exogeneity assumption only, we specify a conditional normality assumption *via* a Mundlak device – as we wish to compare the model with Poisson RE-Mundlak estimates. The individual fixed effect is given by

$$c_i = \psi + \bar{X}_i\xi + a_i, \quad (12)$$

with $c_i | (X_{i1}, \dots, X_{iT}) \sim \text{Normal}(\psi + \bar{X}_i\xi, \sigma_a^2)$ and $a_i | (X_i) \sim \text{Normal}(0, \sigma_a^2)$, and where $\sigma_a^2 = V(c_i | X_i)$. Following Papke & Wooldridge (2008), the RE regression model can now be written as:

$$y_{it} = \psi_a + X_{it}\beta_a + \bar{X}_i\xi_a + a_i + \varepsilon_{it} \quad (13)$$

where the subscript a denotes division of the original coefficient by $(1 + \sigma_a^2)^{1/2}$. Parameters ψ_a, β_a and ξ_a can be consistently estimated using a Pooled (Fractional) Probit analysis – a.k.a. Population Average Probit for Fractional Response – using the GEE method (Generalised Estimating Equations). In addition, extension of this framework to consider ordered response is straightforward.

Let us now consider that $y_{it} = j$ if $\mu_{j-1} \leq y_{it}^* < \mu_j$ with $j = 1, \dots, 5$. Under strict exogeneity (4) and the same conditional normality assumptions on the individual fixed effect as previously, the RE Ordered Probit (or OProbit) with a Mundlak device is defined by (*cf.* Greene & Hensher, 2010: 9.2.)

$$E(y_{it} | X_{it}, c_i) = \Phi(\mu_{oj} - X_{it}\beta_a - \bar{X}_i\xi_a) - \Phi(\mu_{oj-1} - X_{it}\beta_a - \bar{X}_i\xi_a). \quad (14)$$

Here again, the subscript a denotes division of the original coefficient by $(1 + \sigma_a^2)^{1/2}$. Both in the case of PFP and RE Ordered Probit, Xit may contain time-invariant variables, just like in the RE-Mundlak Poisson model. The expected results are believed to be very close to those obtained in the Poisson case since the FE Poisson regression is a special case of the PFP model and the RE Ordered Probit also is a special case of the PFP model (Wooldridge, 2010: 18).

4.2. Models specification

Three sets of variables have been retained with regard to the models to be estimated: time-variant, time-invariant, and retrospective covariates which can be analysed here as specific time-invariant data as far as their time-range does not extend beyond 2004 (start date of wave 1). Descriptive statistics are given in the appendix.

4.2.1. Time-variant covariates

These variables focus on three domains of social policy. First, income adequacy is especially relevant in the present context. Recent reforms in Europe lead to less generous public pensions, “notwithstanding a deliberate policy of large increases in minimum income benefits in many countries, leading to a remarkable convergence of relative benefit levels”⁹ (Goedemé, 2012). How do older people in a frailty process handle the economic consequences of this context? Income adequacy is assessed from the four response items to the question “Thinking of your household’s total monthly income, would you say that your household is able to make ends meet:” “with great difficulty”, “with some difficulty”, “fairly easily”, or “easily.” The first response is used as the reference category for the three other binary variables.

Second, the empirical literature suggests that improving home environments enhances functional ability outcomes (Whal *et al.*, 2009), and that these benefits are long-term, and they extend beyond the disabled person to help the health of other family members (Heywood, 2004). The measure of housing adaptation is derived from a positive response (yes/no) to the question “Does your home have special features that assist persons who have physical impairments or health problems?” Unfortunately, the question was only asked repeatedly to respondents who moved to another residence between the regular panel waves. For many whom remained in the same home over the period, the question was not asked at subsequent waves. The within information does not thus extend to all respondents and an alternative model specification should also be considered to analyse the between information.

Third, social isolation is associated with deterioration in health: the loss of a partner generally leads to worse health status – especially among older cohorts (e.g. Liu, 2012), and participation to social activities and other “social capital” variables have a causal beneficial impact on health (Folland, 2007 ; D’Hombres *et al.*, 2010 ; Ronconi, Brown & Scheffler, 2010 ; Sirven & Debrand, 2012). Social isolation is measured through 3 variables. (i) Living without partner since the last two years is a dichotomous variable (yes/

⁹ In addition, the reforms have been accompanied by measures aiming at encouraging the development of occupational and personal pension plan since household savings are considered too scarce (Börsch-Supan & Brugavini, 2011). In that perspective, Governments are encouraged at increasing and improving the ability of individuals to make sensible choices, e.g. by fostering individual preparedness, reducing the distortions embedded in pension formulae, or choosing an enhanced choice structure (Fornero, Lusardi & Monticone, 2012). Although public provisions are not designed to fully cover financial needs of older people, income adequacy remains a significant role of social policy.

no) derived from the response to the questions “In which year did you:” “get divorced” or “become a widow(er)?”, and the date of interview. (ii) Taking part in social activities is fulfilled from response to the question “Have you done any of these activities in the last month?” Any positive response to the following items “voluntary or charity work, educational or training course, activities of a religious organization, a political or community-related organization” was coded 1, and 0 otherwise. (iii) Because of the potential endogeneity with frailty, the response “Gone to a sport, social or other kind of club” was used separately to define a specific binary variable.

Additional time-variant covariates include a binary index of occupational status indicating whether the respondent is active occupied at the time of the survey. As one may expect to observe a *Healthy Worker Effect* (suggesting in our case that people with higher levels of the frailty index keep out or drop out of the labour market), the occupational status variable has been decomposed by the number of days on sick leave from the questions “In the last 12 months, did you miss any days from work because of your health?” and “About how many days did you miss?” Four categories were derived: never missed work, missed less than 20 days/year, missed 20 days/year or more, and a non-response category; the reference category remains the same: not being active occupied at the time of the survey. Notice that, dummy variables indicating the date of interview were also included to take into account differences in the time-spell between the regular panel waves.

4.2.2. *Time-invariant covariates*

The usual fixed individual covariates include age (year of birth by decades since 1910), gender, education level (highest diploma obtained in three categories: none or primary, secondary, superior, and a non-response category), migration status (whether the respondent is born in the country of residence), and dummies for the country of residence (reference is France). Additional time-invariant retrospective covariates from SHARELIFE include the following:

- *Periods of ill health* or *Ever physically injured*: a binary index of health, taking the value 1 if the respondent reports any periods of ill health over the life-cycle (>1 year) or if she reports any physical injury over the lifecycle (>1 year). Physical disability in late life is indeed found to be associated with health factors in early- and mid-life (Freedman et al., 2008).
- *Financial Hardship*: a dummy indicating if the respondent encountered any periods of financial hardship throughout her life. Physical disability in late life is also found to be associated with financial strain as children and as adults (Szanton, Thorpe & Whitfield, 2010).
- *Health problems during childhood*: (i) retrospective self-rated value of health (SRH) at age 10 was defined as response to the question “(Looking back on your life,) was there a distinct period during which your health was poor compared to the rest of your life?” A binary variable takes the value 1 if the respondent reported that health during childhood was in general excellent or very good, and 0 else (i.e. good, fair, or poor, or spontaneously “Health varied a great deal”); and (ii) illnesses when child: fulfilled as a positive response to any of the questions “Did you ever stay in hospital more than three times within a 12-month period during your childhood” or “Did you ever miss school for a month or more because of a health condition during childhood”(yes/no). This set of covariates reflects what Hass (2008) called ‘the long arm’ of childhood health on current health status.

Table 5. Determinants of frailty – FE Poisson estimates

Dep. var. is Frailty	Baseline FE		Alternative specif.		by Euro-region		
	Poisson		Occupation	Health	North	Conti.	South
Indep. var.	(M1)		(M1.2)	(M1.3)	(M1.4)	(M1.5)	(M1.6)
Time-variant							
Make-ends-meet							
with great difficulty	Ref.		Ref.	Ref.	Ref.	Ref.	Ref.
with some difficulty	-0.077***		-0.079**	-0.056***	0.017	-0.102***	-0.080***
fairly easily	-0.098***		-0.096**	-0.056**	-0.018	-0.125***	-0.077**
easily	-0.123***		-0.117**	-0.084***	-0.038	-0.154***	-0.104*
Adapted housing	0.042		0.034	0.029	0.071	0.075	-0.214
Without partner ≤ 2 years	0.162***		0.164**	0.092**	0.194***	0.095	0.251***
Social activities	-0.074***		-0.077**	-0.047***	-0.091***	-0.082***	-0.038
Sport club, etc.	-0.102***		-0.103**	-0.070***	-0.111***	-0.114***	-0.042
Occupational status							
At work	0.101***			0.101***	0.075	0.107**	0.142**
Not at work	Ref.		Ref.	Ref.	Ref.	Ref.	Ref.
Decomp. Occup. Status							
Missing data wave 1			0.183***				
Never missed work			-0.063				
Sick leave <20 days			0.053				
Sick leave ≥20 days			0.304***				
Health measures							
Poor SRH				0.275***			
Chronic 2+				0.078***			
ADL 2+				0.197***			
Euro-D				0.538***			
Cognitive test				-0.510***			
Time dummies							
Wave 1	Ref.		Ref.	Ref.	Ref.	Ref.	Ref.
Wave 2	0.083***		0.109***	0.056***	0.138***	0.107***	-0.012
Wave 4	0.347***		0.371***	0.281***	0.364***	0.389***	0.262***
Obs.	26,608		26,608	26,218	7,922	12,535	6,151
Nb. indiv.	10,483		10,483	10,352	3,139	4,895	2,449
H0: Make-ends-meet $\beta=0$							
Chi ² (Wald)	19.38		18.17	10.39	1.51	12.78	7.78
p-value	0.000		0.000	0.016	0.680	0.005	0.051
H0: E(Ci,Xit)=0, Strict Exog.							
Chi ² (Wald)	7.280						
p-value	0.507						
H0: Sample Attrition is Exog.							
Chi ² (Wald)	7.88						
p-value	0.247						

Legend: * p<10%; ** p<5%; *** p<1%. Note: Robust standard errors used.

- *Parents' behaviour*: three dichotomous variables (yes/no) are derived from the response items to the question “During your childhood, did any of your parents or guardians:” “Smoke”, “Drink heavily”, or “Have mental health problems”. The implicit idea here is derived from recent work on intergenerational transmission of social inequalities in health (Tranno et al., 2010).

5. Results

Presentation and interpretation of the results is only detailed for Poisson estimates since robustness checks carried out with PFProbit and Oprobit provide very similar results – as expected. The latter are given in Table 6 for information only. The relationships between the frailty index and the various set of frailty determinants considered above are explored consecutively in a FE Poisson and a RE Poisson model with Mundlak device. The former is used to investigate differences in the dynamics of frailty between 2004 and 2011; some alternative specifications are compared, and the baseline model is estimated on different subsamples. The latter is brought into play to analyse the differences between individuals' frailty statuses. Special attention is hereafter given to the role of retrospective life-history events.

5.1. Differences in the dynamics of frailty

Table 5 displays the estimated coefficients from FE Poisson models. The regressions are based on a sample of 26,608 observations over a time period of two or three waves (unbalanced panel), and individuals for whom $y_{it} = 0$ for all t are removed from the sample. The strict exogeneity assumption is tested in the baseline model (M1) and the Wald test indicates that in our case this assumption strongly holds. Consequently, the FE estimator has here some nice properties; it is consistent whatever the distribution of y_{it} is, and the robust standard-errors are valid – as indicated in the previous section. Notice that, time dummies appear to be judicious here (coefficients are significant and positive), indicating that values of the frailty index increase over time, and correcting for the time spell between regular panel waves.

Estimates from model M1 provide the following results. First, individuals report increasing financial difficulties as they become frailer. There is indeed a gradient in the coefficients associated with items of the variable and the joint-test (Wald) indicates that the coefficients are simultaneously and significantly different from zero. This finding concurs with the Health Capital model and suggests that household resources could be inadequate as the frailty process evolves. Second, although the coefficient on housing adaptation displays the expected sign, it is statistically insignificant. As stated previously, this result may be associated with the fact that the question is only time-variant for individuals who changed homes between the waves. On the other hand, when the variable is treated with a between estimator in the RE-Mundlak model (i.e. without a time-average control for this specific variable), the coefficient displays a positive sign ($IRR = \exp(0.232) = 1.261$) and is highly significant ($p < 1\%$) – results not displayed here. This indicates that higher values in the frailty index are associated with adapted housings. Third, social isolation matter: (i) the recent loss of a partner has an important effect in contributing to the increase of the frailty process, the Incidence Rate Ratio is $IRR = \exp(0.162) = 1.176$; while (ii) participation in both types of social activities prevents from increases in frailty (or may even contribute to a reversible pathway). Finally, the coefficient on the occupational status displays a counter-intuitive

Table 6. Determinants of frailty – Poisson, PFProbit and OProbit estimates

Dep. var. is Frailty	Poisson				Pooled Fractional Probit GEE-Mundlak ^a	Ordered Probit RE-Mundlak
	FE ^a		RE-Mundlak ^b			
	(M1)	(M2)	(M3)	(M4)		
Time-variant						
Make-ends-meet						
with great difficulty	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.
with some difficulty	-0.077***	-0.074***	-0.076***	-0.077***	-0.064***	-0.133***
fairly easily	-0.098***	-0.087***	-0.088***	-0.089***	-0.070***	-0.139***
easily	-0.123***	-0.108***	-0.107***	-0.109***	-0.091***	-0.187***
Adapted housing	0.042	0.078	0.075	0.065	0.058	0.129
Without partner ≤ 2 years	0.162***	0.157***	0.157***	0.160***	0.122***	0.244***
Social activities	-0.074***	-0.072***	-0.071***	-0.075***	-0.054***	-0.102***
Sport club, etc.	-0.102***	-0.105***	-0.105***	-0.108***	-0.078***	-0.141***
Occupational status						
At work	0.101***	0.080***	0.081***	0.084***	0.078***	0.172***
Not at work	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.
Time dummies						
Wave 1	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.
Wave 2	0.083***	0.073***	0.056***	0.072***	0.054***	0.103***
Wave 4	0.347***	0.295***	0.291***	0.311***	0.231***	0.448***
Time-invariant						
Female				0.621***	0.428***	0.830***
Birth cohort 1950				Ref.	Ref.	Ref.
Birth cohort 1940				0.005	0.015	0.021
Birth cohort 1930				0.276***	0.211***	0.402***
Birth cohort 1920				0.710***	0.565***	1.132***
Migrant				0.082***	0.057***	0.108***
Education						
None or primary				Ref.	Ref.	Ref.
Secondary				-0.059***	-0.047***	-0.082***
Superior				-0.064***	-0.047***	-0.092***
Missing				0.030	0.008	0.047
Country (ref. France)						
Austria				-0.185***	-0.119***	-0.251***
Germany				-0.258***	-0.173***	-0.346***
Switzerland				-0.219***	-0.156***	-0.267***
Sweden				-0.245***	-0.173***	-0.312***
Netherlands				-0.231***	-0.154***	-0.311***
Denmark				-0.030	-0.025	-0.058
Belgium				-0.063***	-0.040**	-0.095***
Spain				0.066***	0.066***	0.165***
Italy				-0.064**	-0.034*	-0.087**
Retrospective						
Periods of ill health						
Adulthood				0.369***	0.270***	0.530***
Childhood-SRH at 10				-0.188***	-0.124***	-0.232**
Childhood-illnesses				0.130***	0.088***	0.158***
Periods of fin. hardships				0.066***	0.045***	0.094***
Parents' behaviour						
Smoke				0.003	0.001	0.008
Drink				0.096***	0.069***	0.143***
Mental health				0.076**	0.052*	0.106*

Continued on next page...

Table 6. continued

Dep. var. is Frailty	Poisson				Pooled Fractional Probit GEE-Mundlak ^a	Ordered Probit RE-Mundlak
	FE ^a		RE-Mundlak ^b			
	(M1)	(M2)	(M3)	(M4)		
Constant		0.552***	0.507***	-0.300***	-1.003***	
$\mu=1$						-0.126**
$\mu=2$						1.180***
$\mu=3$						2.134***
$\mu=4$						2.985***
$\mu=5$						4.056***
In alpha		-2.471-***	-0.770***	-1.461***		
Rho						0.441***
Obs.	26,608	26,608	40,040	40,040	40,040	40,040
Nb. indiv.	10,483	10,483	17,501	17,501	17,501	17,501
Hausman test						
Chi ² (LR, Wald)	468.89	659.01	1,363.55	394.79	366.23	338.9
p-value	0.000	0.000	0.000	0.000	0.000	0.000
H0: Make-ends-meet $\beta=0$						
Chi ² (Wald)	19.38	17.24	13.95	14.68	18.15	19.07
p-value	0.000	0.001	0.003	0.002	0.000	0.000

Legend: * p<10%; ** p<5%; *** p<1%. Note: Coefficients for time-averages covariates in M2-M6 not displayed here. (a) Robust S.E. (b) Bootstrapped S.E. with 100 replications.

result (which seems to contradict the Healthy Worker Effect) as individuals at work have more chances to see their level of frailty increase over time than people who do not work. This effect is explored more in the details in the following model.

Model M1.2 displays an alternative specification of M1 in which the occupational status is decomposed for individuals at work, according to the number of days in sick leave. This does not affect other coefficients of the model and provides useful additional information. A dummy is inserted to control for the fact that the variable is only available since wave 2. Estimates from M1.2 suggest that the previous counter-intuitive result on the occupational status is driven by individuals reporting being at work at the time of the survey who, at the same time, experienced more than 20 days/year of sick leave. In other words, some people undergo health problems – among which an increase in frailty – while they are still at work.

Model M1.3 provides an alternative specification of M1 in which a set of health measures are inserted as covariates. The idea is to test whether results in M1 hold when the frailty index is “purged” from any influence of other health measures. It may be indeed that the frailty index captures an overall measure of health. In M1.3 the following binary controls (yes/no) are added: poor self-rated health, presence of 2 or more chronic conditions, presence of 2 or more ADL limitations, and presence of depression symptoms from the EURO-D scale. A continuous measure of cognitive performance (Bonsang, Adam & Perelman, 2012) is also added in the set of health covariates. The results reveal that the coefficients on financial difficulties (Wald Chi² = 10.39, p-value = 0.016) and other time-varying covariates remain comparable to those obtained in M1, despite the statistical significance of all the health measures in the right-hand side of the equation. This means that the socio-economic determinants are specific to the Fried frailty index and are not the result of a general health measure.

Models M1.4, M1.5 and M1.6 are replications of M1 on a subset of Euro-regions. It is striking that increased financial difficulties associated with the evolution of the frailty process are no longer significant in Northern countries (Denmark, Sweden, the Netherlands), and less significant in Southern countries (Italy, Spain). Although this effect can be due to the reduction in statistical power from smaller subsamples, the coefficients for these two Euro-regions are inferior to those for Continental countries (France, Belgium, Germany, Austria, and Switzerland). It is also noticeable that the loss of a partner does not seem to impact the evolution of frailty Continental countries. Finally, social capital variables appear to lose their explanative power in the case of Southern countries. Interpretation of these results is tricky because it brings into play different and interlinked institutional features in Europe, ranging from the family to the design of Social Protection Systems. Nonetheless, there is clear evidence that each Euro-region (and potentially, each country) should develop a specific social policy strategy for reducing frailty or maintaining life quality of people in a frailty process.

5.2. Differences in the levels of frailty

Table 6 displays estimates from the baseline FE model (M1) and the RE model with Mundlak specification (M2 to M4). As expected, the Hausman test (Likelihood ratio test for M1 and the Wald coefficients joint-test for time-averages in M2-M4) rejects the RE specification. Coherent with econometric theory, coefficients estimates for time-variant covariates are extremely similar between models M1 and M2, and extension of the sample to individuals with $y_{it} = 0$ over the period (N rising from 10,483 individuals up to 17,501) in M3, does not modify them either. The differences between the levels of frailty now take into account all the different pathways within and outside the frailty process. Model M4 also includes time-invariant covariates to operate partial decomposition of the until-then unobserved individual heterogeneity.

Estimates of time-invariant individual effects in model M4 confirm that women have higher levels of frailty than men (IRR = 1.861), and younger cohorts are unsurprisingly less exposed to frailty. Despite the socio-economic determinants taken into account in the time-varying explanatory variables, (i) migrants have a significant higher level of frailty (IRR = 1.085), while (ii) more educated respondents have significant lower levels of frailty. Cross-country comparison in the conditional levels of frailty indicates that only Spain has a frailer share of its population than the benchmark category – France. Model M4 also incorporates retrospective life-history covariates as time-invariant variables. The estimates concur with previous findings in the literature on the influence of childhood and adulthood health events and financial shocks on late life-health. In the detail, individual reporting health problems in early-life (retrospective self-rated health, or illnesses when child) or in adult-life have higher frailty levels. Other socio-economic factors in M4 provide interesting findings. The influence of periods of financial hardships still remain significant and positively associated with higher levels of frailty (IRR = 1.068), even after health measures over the life-cycle are controlled for. In addition, giving substance to the theory of intergenerational transition of inequalities in health, it seems that parent's behaviour have long term effects on their children – respondents confessing that their parents were heavy drinkers have higher levels of frailty (IRR = 1.101). Finally model M4 provides evidence of socio-economic inequalities (i) in the dynamics of frailty – drawing from *within* estimates comparable to those obtained in M1, and (ii) in the levels of frailty from the *between* estimates of time-invariant covariates.

6. Conclusion

Drawing on the theoretical economic model of health capital, and panel and retrospective data from SHARE, the analysis of some socio-economic determinants of frailty suggested insights for strategies to prevent, reduce, or accompany the process of loss of autonomy in the elderly population. Focus on variables that are relevant for social policy helped establish the presence of social inequalities in the frailty process. As suggested by the health capital theory, several indicators of social and economic status appear to be important determinants of frailty. Our findings also concur (i) with recent findings from epidemiology studies on the influence of education in lower levels of frailty, and (ii) more generally, with the literature on social health inequalities in the older population. Beyond this well-established literature in cross-sectional settings, we also found that the evolution of the frailty process goes along with increased financial difficulties of individuals to make-ends-meet. Moreover, the influence of periods of financial hardships in a life-long perspective is significant and positively associated with higher levels of frailty, even after health measures over the life-cycle (adult health, childhood health, and parents' risk behaviour) are controlled for.

These main results, together with other determinants of frailty, complement public health and medical approaches to disability prevention in Europe, and provide insights for social policy to improve older people's quality of life. First, the socio-economic gradient in frailty levels and dynamics suggests that there is room for Governments, if not in providing additional or more generous safety nets for the ageing population, at least in increasing people's ability to anticipate the consequences of physiologic decline at older ages (e.g. through fostering individual preparedness to make sensible choices in terms of savings, home adaptations, or investments in family and social networks, etc.). Second, although most of the research on frailty focused on interventions in a health care environment, empirical evidence in the general population indicates that specific areas of interest – usually unmapped in frailty interventions – should be considered. Our findings suggest that a significant share of the population aged 50 or more experienced a rapid increase in the frailty process during the last period of their working lives. This pleads in favour of more coordinated public policies of labour force participation of the older population with the disability prevention strategies. Third, more efficient prevention could benefit from better targeting of the sub-populations at risk of frailty – especially women, but also recently widowed persons, migrants, and less privileged elders in social and economic terms. Social actions such as participation to social activities could also be implemented in the early stages of the frailty process as a means to simultaneously maintain physical activity and prevent social isolation.

Finally, these results underline the legitimacy of Social Protection Systems in Europe to moderate the impact of health and economic shocks over the life-span and to maintain the reserve capacity individuals bring in late life. There is a potential for further research to unveil the yet under-acknowledged role social policy can play in increasing healthy life years. Special attention could thus be dedicated to the comparative efficiency of the various Welfare States regimes in Europe.

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8. Appendix

Table A1. Overall, between, and within frequencies of time-variant covariates
(Percentages)

Time-variant	Overall	Between	Within
Make-ends-meet			
with great difficulty	7.3	12.7	60.1
with some difficulty	21.4	35.1	62.4
fairly easily	35.4	55.6	63.5
easily	35.8	49.5	71.2
Adapted housing			
No	92.6	93.2	99.2
Yes	7.4	8.3	90.8
Without partner \leq 2 years			
No	98.2	99.7	98.6
Yes	1.8	3.9	45.4
Social activities			
No	66.6	81.2	83.5
Yes	33.4	46.5	69.2
Sport club, etc.			
No	71.8	85.1	85.4
Yes	28.2	40.9	67.0
Occupational status			
Not at work	73.4	80.2	91.0
At work	26.6	33.8	79.8

Table A2. Overall frequencies of time-invariant covariates
(Percentages)

Time-Invariant		Retrospective	
Sex		Periods of ill health	
Male	46.2	Adulthood	
Female	53.8	No	72.6
Birth cohort		Yes	27.4
1920	12.1	Childhood-SRH at 10	
1930	27.1	Less than very good	98.9
1940	38.8	Excellent or very good	1.1
1950	22.1	Childhood-Illnesses	
Migrant		No	93.2
No	93.1	Yes	6.8
Yes	6.9	Periods of fin. hardships	
Education		No	65.7
None or primary	46.2	Yes	34.3
Secondary	28.6	Parents' behaviour	
Superior	24.7	Do not smoke	35.5
Missing	0.5	Smoke	64.5
Euro-Region		Do not drink	91.3
North	31.8	Drink	8.7
Continental	47.2	No mental health pb.	97.4
South	21.0	Mental health pb.	2.6

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On the Socio-Economic Determinants of Frailty: Findings from Panel and Retrospective Data from SHARE

Une analyse des déterminants socio-économiques de la fragilité des personnes âgées à partir des données de panel et rétrospectives de SHARE

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Recent studies on the demand for long-term care emphasised the role of frailty as a specific precursor of disability besides chronic diseases. Frailty is defined as vulnerable health status resulting from the reduction of individuals' reserve capacity. This medical concept is brought here in an economic framework in order to investigate the role social policies may play in preventing disability or maintaining life quality of people in a disablement process.

Using four waves of panel data from the *Survey on Health, Ageing, and Retirement in Europe* (SHARE), a frailty index is created as a count measure for five physiologic criteria (Fried model) for respondents aged 50+ in 10 European countries, between 2004 and 2011.

The longitudinal dimension is explored in two ways. First, differences in frailty dynamics over a seven-year-time period are analysed through variables that are relevant for social policy (income maintenance, housing adaptation, and prevention of social isolation) in a panel model for count data with fixed effects. Second, the individual fixed effects are decomposed by means of a random effects model with Mundlak specification. SHARE additional retrospective data on life history (SHARELIFE) are then used to investigate differences in frailty levels.

The results reveal the presence of various sources of social inequalities over the life-course. Social Protection Systems thus appear to play a major role in accompanying, preventing or reducing the frailty process. Several policy implications are suggested.

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Les études récentes sur la demande de soins de long-terme ont mis en évidence le rôle de la fragilité en tant que précurseur de la perte d'autonomie, indépendamment des maladies chroniques. La fragilité est définie comme un état de santé vulnérable résultant de la diminution de la réserve physiologique de la personne âgée. Ce concept gériatrique est ici mobilisé en population générale et dans un cadre économique afin d'analyser le rôle des politiques publiques dans la prévention et l'accompagnement des personnes âgées dans un processus de perte d'autonomie.

A partir des données de panel et rétrospectives de l'enquête SHARE entre 2004 et 2011, nous étudions les déterminants socio-économiques de la fragilité en Europe. Dans un modèle à effets fixes, les différences individuelles dans la dynamique de la fragilité sont analysées au regard de trois piliers de l'action sociale : politique de soutien au revenu, lutte contre l'isolement social, promotion de l'aménagement du logement. Les différences persistantes dans les niveaux de fragilité sont explorées en utilisant les données rétrospectives sur l'histoire de vie (SHARELIFE) dans un modèle à effets aléatoires avec une spécification à la Mundlak.

Les résultats principaux indiquent la présence d'inégalités sociales de santé sous différentes formes et à différentes époques de la vie. L'importance des systèmes de protection sociale en Europe est reconnue comme un moyen d'accompagner, voire de retarder l'évolution du processus de perte d'autonomie. Plusieurs considérations de politique publique sont discutées.