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Esen Erdoğan-Çiftçi

Health Perceptions and Labor
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Health Perceptions and Labor Force Participation of Older Workers

Gezondheidsperceptie en de arbeidsparticipatie van oudere werknemers

THESIS

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When you dance, your purpose is not to get to a certain place on the floor. It's to enjoy each step along the way.

Wayne Dyer

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Chapter 1

Introduction

The financial meltdown and ensuing economic crisis with which the 21st century has entered its second decade are creating acute problems for public finances in many advanced countries, especially in those whose demographic structure already embedded a “time bomb” in the form of unsustainable future pension claims. Now more than ever it appears that a necessary condition for long term stability is to raise people’s life time productivity, by increasing both real product per unit of time and working lives. These are not new problems, of course. Labor force participation among the elderly experienced a considerable decrease between the late 1970’s and the late 1990’s as a result of financial incentives encouraging early retirement and generous health and disability insurance systems. In response, a series of reforms of social security systems have been implemented in many countries over the last twenty years. These mostly consist in increasing the mandatory retirement age, adjusting the length of the working life to increased life expectancy, abolishing early retirement schemes, and tightening the eligibility conditions for disability and unemployment insurance (OECD, 2009).

Economists have paid attention to the effects of Social Security institutional arrangements on labour market dynamics in general and retirement in particular. The famous work of Gruber and Wise (2004) shows that marginal financial incentives toward exiting or staying at work play a crucial role in individuals’ labor market decision making. In practice, some recent reforms targeting the early exit from the labor force in countries such as the Netherlands (Euwals et al. 2011) or Spain (García-Gómez et al. 2011) have had a degree of success.

Empirical evidence on these issues, as the studies cited above provide, is necessary because increasing the mandatory retirement age or introducing financial incentives to discourage early labor market exit do not guarantee that people will actually work longer. If increased survival rates are associated with sufficient improvements in health during survival, then it is indeed possible to sustain longer working lives. However, even though there have been considerable improvements in health outcomes over the last decades, severe health inequalities still remain and have sometimes

widened. In particular, poorer and more disadvantaged people such as people in lower occupations as opposed to those in managerial professions are more likely to have a disability and suffer from ill health (Bajekal and Goldblatt, 2006). As health operates alongside pensions and benefits rules in determining labor supply, social security reforms may have differential effects on labor market behavior by health status. Individuals in good health may be able to continue working until older ages, while individuals in poor health may not be able to stay in the labor force for as long. Likewise, individuals with lower wealth or wages may need to continue working at old age even if in poor health while richer but equally unhealthy individuals may afford to retire early.

These considerations suggest the following research questions: does the effectiveness of the financial incentives embodied in social security rules vary according to the health status of individuals? And, in relation, does the propensity to exit the labour market after a health shock vary according to the wealth level?

In the work that follows, I present four empirical studies. The first two studies concentrate on the effects of health shocks, financial incentives and their interactions on the propensity to exit the labour market either through the retirement or the inactivity / disability route. They aim to provide evidence for the two research questions posed in the previous paragraph. The last two chapters look at two types of health perceptions that are commonly used in modeling several economic decisions, in particular decisions about labour force participation, and investigate the validity and formation of forward and backward looking health perceptions. The third chapter compares the validity of two alternative ways of measuring recent health changes by examining their ability to predict subsequent survival. The fourth chapter does the same for survival expectations but also goes on to examine their formation.

Chapter 1 is concerned with the relation between health shocks, financial incentives generated by the social security system on retirement and inactivity transitions. I use the theoretical insights of the Option Value Model (Stock and Wise, 1990) to analyse the role of health in the decision to leave work. I then postulate a hypothesis stating that the effects of financial incentives are subordinated to the health status of the individual. Subsequently I investigate empirically, using the Spanish sample of the European Community Household Panel, whether individuals who differ in health characteristics react differently to financial incentives when confronted with retirement decisions and find supporting evidence for the subordination hypothesis mentioned above.

In Chapter 2, I test the subordination hypothesis in the context of the Dutch labour market and, additionally, provide evidence related to the second of the research questions posed above, which I term the affordability hypothesis. Namely, I investigate whether individuals with higher pension

wealth can “afford” to withdraw from the labour market to a greater extent than the rest of individuals after sudden adverse health. The richness of the data set used, i.e. the administrative data from the main Dutch pension fund for health care workers, allows me to study these issues separately for men and women. Furthermore, in contrast to the data used in Chapter 1, these data contain actual pension benefits and objective measures of health shocks, thus providing a greater degree of robustness to the empirical findings. I find that for both males and females new adverse health events increase the likelihood of transitions into disability. When it comes to transitions into retirement, however, they only affect males. I also find that financial incentives to early retirement are very important in explaining early retirement rates but they also affect disability rates. I find that a new health event increases the probability of transiting into early retirement *only* among workers with high pension wealth. Furthermore, this differential with respect to workers with low pension wealth is not compensated by the larger effect exerted by disability insurance on the propensity to retire early among the former. Therefore, my results provide support for the *affordability hypothesis*. On the other hand, contrary to the findings for the Spanish labour market reported in Chapter 1, I find little empirical support for the *subordination hypothesis* in the context of the Dutch labour market.

In Chapter 3, I investigate the validity of prospective health changes, i.e. changes based on longitudinal information on health status data, versus retrospective measures of health changes, i.e. changes based on the responses given by individuals when asked to compare their current health to a reference point in the past. In particular I investigate their predictive power for subsequent actual mortality. I focus on the observed and reported changes in self-assessed health (SAH), since this is usually the only health measure available in many individual surveys. I pay attention to health shocks because these may be more important precursors of later outcomes than health levels. I exploit a unique panel data set taken from the cohort study GLOBE conducted in the Netherlands, which includes measures of health changes and follow-up mortality. I find that for a high proportion of reports prospectively measured health changes in SAH do not concur with retrospectively reported health changes. The evidence shows that information regarding recent health changes carries additional information to that contained in the reported health levels. My estimates show that both measures of health changes are predictive of mortality when controlling for levels of SAH and socioeconomic characteristics only. However, if I control for SAH, prior presence of chronic conditions, the onset of new conditions, I find that prospective health changes still predict longevity but retrospective changes fail to do so.

In Chapter 4, I focus on subjective survival probabilities, a more forward looking measure than the health changes considered in Chapter 3. An individual’s ability to process information and to form an understanding of an expected lifespan may be an important determinant of subsequent outcomes

such as retirement and savings. Therefore, to understand the formation and to assess the validity of these longevity expectations is interesting for economists as well as policy makers. I investigate the formation and the validity of subjective survival expectations using the Health and Retirement Study (HRS) for the United States. The focus is in particular, on the role of education and cognition by examining whether predictive ability varies with age, education and cognition. In accordance with earlier literature, I find that individuals' reported survival expectations predict their actual longevity. However, I find a stronger association of subjective survival expectations with mortality for the group of individuals with a higher cognitive function and higher education. Subjective survival expectations for low education and low cognition groups turn out not to be predictive of mortality. I interpret this result as evidence for the existence of a greater degree of measurement error in the formation of survival probabilities among individuals with low levels of education and the cognitively impaired. This heterogeneity in measurement error across education and cognition groups then leads me to examine whether there are corresponding differences in the way in which new information affects the revision of survival expectations. In this regard I find that individuals in the lower cognition/education groups modify their expectations in response to a considerably smaller number of new events than individuals in higher cognition/education groups. The less educated and cognitively less able are less likely to revise survival expectations downward in response to new information on objective risks, such as the onset of cancer or the occurrence of a stroke.

Finally, Chapter 5 summarizes all four studies and draws conclusions from the thesis.

PART I

Labor market participation of older workers

Chapter 2

Does declining health affect the responsiveness of retirement decisions to financial incentives?

Both the impacts of financial incentives and health on transitions into retirement and inactivity by older workers have been studied extensively in a variety of contexts but far less is known about their interaction. Guided by the option value framework, we use Spanish data from the *European Community Household Panel* to compare the impact of the public pension system incentives on the retirement behaviour of workers who experienced health shocks and those who remained in good health. Our evidence suggests that the impact on retirement of forward-looking incentive measures such as the peak value is conditional on being in good health. These findings imply that many of the currently proposed and enacted pension reforms aimed at modifying financial incentives may only be effective for people in sufficiently good health.

2.1 Introduction

The relationship between health, financial incentives generated by social security systems and labour market decisions of older workers is a salient topic of debate in OECD countries. Decreasing mortality rates and a prolonged life expectancy have resulted in an ageing population in general and an ageing workforce in particular. Despite substantial increases in longevity, the labour force participation rates of the elderly have steadily fallen throughout the twentieth century due to financial incentives encouraging early retirement and generous health and disability insurance systems. Since these retirement arrangements seem no longer sustainable, reforms such as capping early retirement schemes and raising the normal retirement age have been adopted over the last decade (OECD, 2009). If increased survival rates are associated with sufficient improvements in health during survival, then it is indeed possible to sustain longer working lives. In this case, reforms targeting the extension of working life through changes in financial incentives may be successful. However, workers with poorer health may be less sensitive to such changes. This paper focuses on whether individuals who differ in health characteristics react differently to financial incentives when confronted with retirement decisions.

There is a body of international evidence on the importance of financial incentives and health on retirement behaviour (Lindeboom, 2006a). However, only one paper, by Banks *et al.* (2007), explicitly considers the conditionality of financial incentive effects on health status. Examining financial incentives in the presence or absence of specific health problems for men and women in the U.K using two waves of the English Longitudinal Survey on Aging (ELSA), they find evidence suggesting that the (negative) impact of peak value accrual on the odds of retirement is only significant for those without health problems. This prompts what we will label a *subordination hypothesis*: financial incentives exert a stronger effect on the chances of retirement for healthy workers. If health is poor, or in the presence of a health shock, the financial incentives embodied in pension rules might be dampened. These financial incentives might therefore be subordinated to health. We will discuss this hypothesis in the context of the Option Value model (Stock and Wise, 1990; Coile and Gruber 2007) and specify and estimate an empirical retirement model for Spain that allows us to evaluate its merits.

We use data from the Spanish sample of older workers (50+) in the *European Community Household Panel* (ECHP). The Spanish case is of particular interest for the purposes of this research because in the considered period (a) the take up of private pensions was marginal, and therefore pension wealth can be estimated from the labour market histories of workers by simply applying the set of rules of the pay-as-you-go public system and (b) reforms enacted in 1997 introduced additional variation in pension rules. Moreover, the ECHP data contain information that permits the construction of a health stock measure and changes therein. Our health measure takes into account recent insights from the literature, like the desirability of using an indicator of health that reflects more than one dimension, as well as the need to consider the effects of the initial health stock and its changes in order to avoid the potential endogeneity of health in a labour outcomes equation.

The paper is organized as follows. In section 2.2 we review some of the recent literature on health and retirement decisions in both the US and Europe. Section 2.3 discusses the interaction of health shocks and financial incentives in the context of the Option Value model. Section 2.4 describes the data and our modeling strategy for financial incentives and health. Section 2.5 describes the econometric model and section 2.6 contains the discussion of its estimates. We conclude with a discussion of the implications of our findings.

2.2 Health, financial incentives and retirement

The literature contains many studies about the effect of financial incentives on retirement, among which the most well-known comparative work is reported in Gruber and Wise (2004) which includes recent case studies for twelve countries. These applications typically find a strong effect of financial incentives on retirement choices. For instance, in the case of Spain, Jimenez-Martin *et al.* (2004)¹ compute financial incentives from Social Security administrative records and find a significant effect on the probability of retirement for private sector workers, but not for either the self-employed or public sector workers. For Italy, after a detailed computation of financial incentives variables using administrative

¹ Jimenez-Martin *et al.* (2004) provide a thorough description of the functioning and historical evolution of the Spanish Social Security system, from which we draw when describing and modelling the pension arrangements.

data, Belloni and Alessi (2009) find that when employees become eligible for pension benefits, the change in financial incentives they experience is so great that their retirement probability increases by 30 percentage points.

Surveys of early work on the relationship between health and retirement (e.g. Currie and Madrian, 1999; Lindeboom, 2006a) do not discuss the conditionality of the effects of financial incentives on health. And more recent literature has focused on the relative importance of permanent or temporary health shocks versus a gradual deterioration of health in retirement decisions (Bound *et al.* 1999; Disney *et al.* 2006; Hagan *et al.* 2008; Roberts *et al.* 2010). A common finding in these studies is that changes in health play an important role in retirement decisions, with stronger effects for the inactivity route, and that adverse health shocks are important predictors of retirement. Lindeboom *et al.* (2006b) focus on the relationship between the onset of a disabling condition and employment outcomes and estimate that health shocks increase the likelihood of exit into the disability scheme by 138 per cent. Jiménez Martín *et al.* (2006) study older Spanish workers' labour force transitions following a health/disability shock and find that the probability of remaining in work decreases with both age and the severity of the shock, and that the probability of remaining in employment varies substantially with the type of health condition or disability. Another strand of the empirical literature has addressed the relative importance of health versus financial incentives. Kerkhofs and Lindeboom (1999) investigate the effects of health and financial incentives on three alternative exit routes in the Dutch labour market: early retirement (ER), disability insurance (DI) and unemployment insurance (UI). They show that health is the most important factor in explaining transitions into DI and UI schemes, while financial incentives are dominant when explaining transitions into ER schemes. Other papers that compare the effects of financial variables and subjective health status on retirement are Bound (1991), Dwyer and Mitchell (1999) and McGarry (2004). These authors tend to find that the effects of health are substantially stronger than those of financial incentives. In their review article of mostly US studies, Lumsdaine and Mitchell (1999) conclude that the impact of financial incentives on retirement is important, but can only explain half of the observed variation in retirement rates in the US.

The studies cited above therefore suggest that the financial incentives embodied in the Social Security rules are significant determinants of retirement. And so are poor health and

health shocks. Because health itself is obviously not a direct policy lever, the majority of policies aimed at extending working lives focus on altering the set of pension rules - the so called “parametric reforms” (OECD, 2009). Nonetheless, it is important to investigate whether health might alter the effectiveness of financial incentives. That is, in addition to making retirement more likely *ceteris paribus*, poor health or health shocks might dampen the incentives created by such financial incentives and reforms. This contingency would have interesting consequences, as it would imply that marginal reforms in pension rules will have a limited effect on unhealthy workers. Conversely, health improvements will have not only an independent effect on extending working lives, but they will also act through the modification of financial incentive effects.

To the best of our knowledge, the sole study that has shed some light on this conjecture is by Banks *et al.* (2007), who estimate a model for labour transitions for English workers between age 50 and the State Pension Age in England. Their model includes measures of financial incentives inspired by the option value model of Stock and Wise (1990), as well as different measures of health. Interestingly, Banks *et al.* (2007) find that peak value accrual –an incentive measure capturing the cost of opportunity of retiring today in terms of foregone future pension benefits- only exerts a significant effect on the likelihood of retirement for those in good health.

In the following section we provide a discussion of how health shocks are likely to influence the effect of the financial incentives embodied in the Option Value model of retirement. In subsequent sections we will specify and estimate an econometric model that accommodates the possibility that the effects on retirement of such financial effects vary with health.

2.3 Health shocks and retirement in the option value model

There is a considerable body of literature on the relationship between social security benefits and labour force participation (for a review, see Coile and Gruber, 2007). For retirement, Stock and Wise (1990) emphasized the trade-off between the level of

retirement benefits and the entire future potential income and wealth stream from working. Their option value (OV) model's main insight is that retirement models should consider explicitly the utility difference between retiring today and retiring at the date that optimizes utility.

Coile and Gruber (2007) have provided an empirical implementation of the OV model where individuals compare the value of retiring immediately with the value of continuing work and holding the option of retiring in the future. Individuals at work re-evaluate this comparison at each period. For our purposes, we consider the effects of health shocks and their interactions with financial incentives in the context of such a model.

Suppose individual i will receive wage income Y_s in year s as long as he continues to work. If the individual retires in year s , he will receive retirement benefits B_s . If we define R as the year of retirement, and benefits as $B_s(R)$, then the value of retiring at R is given by:

$$V_t(R) = \sum_{s=t}^{R-1} \rho_{s|t} \beta^{s-t} U(Y_s) + \sum_{s=R}^T \rho_{s|t} \beta^{s-t} U(B_s(R)) \quad (2.1)$$

where β is a subjective discount factor and $\rho_{s|t}$ is the probability of being alive at some future date conditional on being alive today. Coile and Gruber (2007) assume that the individual indirectly derives utility from real income while working, $U(Y_s) = Y_s^\gamma$ and utility from pension benefits received while retired $U(B_s(R)) = [kB_s(R)]^\gamma$ where k is a parameter to account for disutility of labour and γ is a parameter of risk aversion. The OV model assumes that in every period t , individuals compare the lifetime utility of retiring at time t with the lifetime utility of retiring at a future "optimal" date, R^* , the retirement date at which lifetime utility is maximized. That is, the individual evaluates

$$OV_t = V_t(R^*) - V_t(t); \text{ where } R^* \text{ solves } \max V_t(R) \text{ with } R \in \{t+1, \dots, T\} \quad (2.2)$$

Therefore, the OV at time t is:

$$OV_t = \left[\sum_{s=t}^{R^*-1} \rho_{s|t} \beta^{s-t} Y_s^\gamma + \sum_{s=R^*}^T \rho_{s|t} \beta^{s-t} (kB_s(R^*))^\gamma \right] - \sum_{s=t}^T \rho_{s|t} \beta^{s-t} (kB_s(t))^\gamma \quad (2.3)$$

which equals labour income up to R^*-1 , plus benefits accruing from R^* conditional on having retired at R^* , minus the benefits that would have accrued from the current period up to T if retirement was chosen contemporaneously. If, for *any* future date R^* , OV is still positive, the individual will continue working. If not, he will retire. So, the higher the OV, the lower the probability of retiring.

It is useful to rewrite Equation 2.3 as the sum of the following three summands:

$$OV_t = \sum_{s=t}^{R^*-1} \rho_{s|t} \beta^{s-t} (Y_s^\gamma) + \left[\sum_{s=R^*}^T \rho_{s|t} \beta^{s-t} [(kB_s(R^*))^\gamma - (kB_s(t))^\gamma] \right] - \sum_{s=t}^{R^*-1} \rho_{s|t} \beta^{s-t} (kB_s(t))^\gamma \quad (2.4)$$

The first summand measures labour income until R^* . Defining “pension wealth” (PW) as the expected present value of a worker’s stream of pension benefits at year t , should he retire at a given age h^2 :

$$PW(h, t) = \sum_{s=h}^T \rho_{s|t} \beta^{s-t} E_t(B(s, h)) \quad (2.5)$$

where $E_t B(s, h)$ is the pension expected at age $s \geq h$ in case of retirement at age h , we can easily see that the third summand is a measure of pension related financial incentives: i.e. (lost) “pension wealth” from not retiring until R^* .

The second summand in Equation 2.4 (between brackets) measures the “peak value” (PV) at R^* . Given PW, the peak value is defined as the maximum difference in PW between retiring today and retiring at the age at which the expected value of the PW is maximized³:

$$PV_t = \max_h \{PW_h - PW_t\}, \quad h = t+1, \dots, R \quad (2.6)$$

Equation 2.4 allows us to discuss the effects of income, growth of benefits, pension wealth and health shocks in the context of the OV model. From this equation we can see that, *ceteris paribus*, higher income will increase OV_t and therefore make retirement less likely. The second summand shows that the growth of benefits with a longer active life - as

² T is assumed to be 100 and the discount factor to be 0.95238 (discount rate of 5 percent) in the empirical analysis.

³ In the computation of pension wealth, R is a mandatory retirement age (which does not exist in Spain for the majority of workers), but we have assumed that R=70.

proxied by the peak value - will raise OV_t and make retirement less likely: the greater the difference between brackets, the more likely that the OV expression is positive. Finally, the third summand enters with a negative sign: the greater the lost pension wealth (as explained earlier, the sum of benefits in the third summand is the pension wealth that would be obtained between time t and R^*), the lower OV_t , and the more likely it is to retire at time t .

There are several ways through which a health shock can affect the individual's evaluation of future OVs. One is a direct effect on the future string of labour income, which may be reduced if the shock affects productivity. But this is not our main concern here. Instead we focus on the indirect effects that are channeled through the parameters that determine the peak value and pension wealth. Thus, a second possible effect, for instance, is an increase in the marginal disutility of labour by an increase in the parameter k in the model. A third effect is a possible update (downward adjustment) of the survival probabilities ρ_t . Finally, a health shock might also raise the time preference rate of the individual, and reduce the discount factor β . In this sense, it would reduce the value of future consumption to the individual and act in the same direction as the downward adjustment in survival probabilities.

First, consider the effect of a health shock on pension wealth (third summand in Equation 2.3). A health shock increases the disutility of labour (k). An increase in k boosts the value of the string of benefits and, via the effect of this summand, reduces future OVs. However, a health shock may also result in a reduction in the survival probabilities or an increase in time preference, and this will have a compensating effect. Therefore, the net effect of a health shock, *ceteris paribus*, on pension wealth is indeterminate and we cannot expect *a priori* the interaction of health shock with pension wealth to go in either direction.

There is also no *a priori* theoretical expectation on the sign of the interaction of health shocks with the peak value. Through the second summand in Equation 2.4, the increase in k would act in the direction of increasing future OVs (thereby reducing the chances of retirement), while the decrease in survival probabilities acts in the opposite direction (increasing the chances of retirement).

Notwithstanding the theoretical indeterminacy of the impact of a health shock on the effects of peak value and pension wealth on the likelihood of retirement, the OV model suggests that the effects of an increase in the disutility of labour are more likely to prevail in summands corresponding to near future periods than in those corresponding to the distant future. This is a consequence of the fact that survival probabilities and discount factors will in the limit drive the summands towards zero, and even more so after a health shock. As a consequence, while we cannot rule out the possibility that a health shock alters the chances of retirement in the same direction through its impact on peak value and pension wealth, it is *a priori* more likely that such a coincidence, if it exists, manifests itself in a shock which makes pension wealth more inciting to retirement and peak value less inciting to continue working rather than the opposite.

Whether and by how much health alters the effects of financial incentives created by pension rules are therefore empirical questions, as far as the OV model can predict. A hypothesis of subordination of financial incentives to health, stating that some or all of the effects of the financial incentives embodied in pension rules might be dampened by poor health or health shocks, is therefore plausible. Indeed the results of Banks *et al.* (2007) would seem to confirm such a hypothesis for the case of the peak value. In the following sections we will investigate this empirically using data from the Spanish labour market.

2.4 Data and variables

2.4.1 Data

Our data are taken from the public use files of the *European Community Household Panel (ECHP)*. The ECHP was designed and coordinated by Eurostat, the European Statistical Office and consists of a longitudinal survey based on a standardised questionnaire that involves annual interviewing of a representative panel of households and individuals 16 years and older in each EU member state. It includes respondents' demographic background, employment status, income, health status, social transfers etc. We use all eight waves available (1994-2001) for Spain. Self-reported labour market status is defined as 'employed' if the respondent is working full-time or part-time in paid employment, and

‘self-employed’ if in self-employment or working in a family enterprise. Old-age retirement status is also based on self-reports. In line with a number of other studies (e.g. Bound *et al.* 1999; Disney *et al.* 2006; Hagan *et al.* 2008), we use two retirement definitions: (i) the narrowly defined retirement status, corresponding to a self-reported retirement and (ii) an extended definition including, in addition, being reported as economically inactive or doing housework, but excluding those reporting themselves as unemployed. Retirement is taken as an absorbing or permanent state and any subsequent transitions back to work are ignored. We will refer to these two labour outcomes as “standard retirement” and “extended retirement” throughout the paper.

Since we are concerned with transitions to retirement, we will focus on individuals who are at least 50 years old at wave 1. The ECHP allows us to adopt the stock sampling with follow up (also known as “left-truncation” or “delayed entry”) set up (Lancaster, 1990, Jenkins, 1995). Namely, at the date of wave 1, i.e. 1994, we observe a sample of individuals in employment (either employed or self-employed). The panel structure of the data set implies that we may subsequently observe them at the rest of waves up to 2001. These individuals can stay in the labour force, retire or be lost to follow up. Transitions into other states as well as attrition are summarized in Table 2.1. The initial stock sample consists of 1449 employed individuals, 74% male, and 46.3 %, 32.9% and 20.7% in age groups 50-54, 55-59 and 60-64 respectively. The stock sample gradually reduces to 347 by the eighth wave, while the number of self-reported retired respondents increases from 42 in wave 2 to 302 in wave 8.

The main income variable used is the log of total household income. Other socio-demographic variables used in the analysis include: house ownership, educational attainment graded using the highest grade of education achieved on the 3 level ISCED scale - completed third level secondary education; completed second stage of secondary education; completed less than second stage of secondary education; a quadratic function of age; whether there are children under 12 living in the household; and whether the individual is married. Names and definitions of variables and their descriptive statistics are presented in Tables 2.2 and 2.3 for the estimation samples for both definitions of retirement.

Table 2.1: Labour market status by wave in stock sample

Wave	Employed	Self-employed	Retired	Unemployed	Inactive	Housework	Attrition	Total
1	937	512						1449
2	663	359	42	49	58	33	245	1204
3	544	310	121	71	46	36	321	1128
4	447	251	154	61	62	43	431	1018
5	352	225	201	53	62	40	516	933
6	288	200	233	38	82	41	567	882
7	240	183	262	24	74	41	625	824
8	197	150	302	20	90	35	655	794

Note: Verbeek and Nijman (1992) tests for attrition bias did not reject the null hypothesis of no attrition bias. Results can be obtained upon request.

Table 2.2: Variable labels and definitions

Variable	Description
Retired	=1 if individual reports to be retired
Extended retired	=1 if individual reports to be retired or inactive doing housework, 0 o.w
Age minus 50	Age variable minus 50
Age minus 50 squared	Square of age variable minus 50
Married	=1 if married
Male	=1 if male
High education	=1 if completed third level of secondary education
Middle education	=1 if completed second stage of secondary education
Low education	=1 if less than second stage of secondary education
House owner	=1 if respondent owns a house with/without mortgage
Children	=1 if there are children under 12 in the household
Initial health stock	Initial value of the health index in wave 1
Small health shock	Small acute health shock, binary dummy=1 if individual has <1 standard deviation increment in health index value between waves
Large health shock	Large acute health shock, binary dummy=1 if individual has ≥ 1 standard deviation increment in health index value between waves
Pension Wealth	Computed pension wealth (ten thousands of Euros)
Peak value	Computed peak value (Euros)
Household income	Total household income (Euros)
Public pension entitled two periods later	Dummy signalling $B(s,h)=0$ & $B(s+2,h)>0$
Public pension entitled next period	Dummy signalling $B(s,h)=0$ & $B(s+1,h)>0$
Public pension entitled this period	Dummy signalling $B(s,h)>0$
Public sector employee	=1 if respondent's sector of employment is a civil servant

Table 2.2: Continued

Private sector employee	=1 if respondent's sector of employment is within the private sector
Self-employed	=1 if respondent is self-employed
Housework	=1 if respondent is in housework category
Unemployed	=1 if respondent is unemployed
Inactive	=1 if respondent is inactive

Table 2.3: Descriptive statistics

Variable	Standard Retirement		Extended Retirement	
	Mean	Std. Dev	Mean	Std. Dev
Health index	-0.239	0.894	-0.136	0.753
Small health shock	0.205	0.404	0.203	0.402
Large Health Shock	0.112	0.316	0.105	0.306
Age	57.7	3.701	57.6	3.681
Married	0.844	0.363	0.844	0.363
Female	0.278	0.448	0.229	0.421
High Education	0.163	0.369	0.179	0.383
Middle Education	0.082	0.274	0.090	0.287
House Owner	0.908	0.289	0.904	0.294
Has Children un. 12	0.070	0.256	0.070	0.256
Pension wealth (in (2001) € 0000)	16.517	9.534	17.329	9.694
Peak value (in (2001) € 0000)	6.972	5.726	7.506	5.907
Log Household income	14.037	0.809	14.058	0.826
Retired	0.070	0.255	0.097	0.295
Public sector	0.168	0.374	0.196	0.397
Self employed	0.308	0.462	0.351	0.477
Unemployed	0.059	0.235	0.057	0.232
Housework	0.048	0.214		
Inactive	0.069	0.255		
N	4991		4267	

2.4.2 Modeling pension incentives

Spain has a mandatory pay-as-you go public pension system run by the Social Security. For the average Spanish person, receiving a pension usually means receiving a *public* pension, as less than 1% percent of Spanish retirees draw more than 10% percent of their annual income from a private pension plan. In addition, the Social Security system offers unemployment benefits, disability benefits and some non-contributory benefits. We will focus on contributory public pension benefits. These are not directly observable in the

ECHP. However, we are able to reconstruct them by exploiting information on individual labour histories, upon which we apply the set of rules governing benefits, penalizations for early retirement etc. Belloni and Alessi (2009) have shown that where true seniority is not observed directly from administrative records, there is scope for measurement error in computed Pension Wealth, which in turn may lead to downwardly biased estimates for the effects of financial incentives. We are aware of such possibility, but unfortunately, while there are Social Security data sets providing true seniority for representative samples of Spanish workers, these data sets do not contain information on health or health shocks. For these reason we resort to the ECHP, and we construct seniority as the difference between current age and the reported age of entry in to the labour market.⁴

The measures of pension wealth thus obtained incorporate variation from three sources. First, there are several Social Security regimes, each with different rules. Secondly, there is individual variation in the length and size of contributions to the corresponding regime. Finally, in 1997, a year covered by our data set, there was a reform in pension rules that affected some of the Social Security regimes.⁵ While the first two sources of variation cannot be considered strictly exogenous to retirement, the dependence of contemporaneous pension wealth on decisions taken long ago (i.e. which Social Security regime) and on the history of contributions over a relatively long period guarantee that pension wealth is predetermined. The additional variation afforded by the 1997 reform can be considered exogenous.

Public contributory pensions are provided by the following three programs in Spain: (1) the “General Social Security Scheme” (*Regimen General de la Seguridad Social*, or RGSS) is the “default” regime for private sector employees but it also covers the members of cooperative firms, the employees of most public administrations other than the central governments and all unemployed individuals complying with the minimum number of contributory years when reaching 65; (2) the “Special Social Security Schemes” (*Regimenes Especiales de la Seguridad Social*, or RESS) cover the self-employed and professionals plus some groups of workers in certain occupations. The RESS includes five special schemes, but by far the largest is the one for the self-employed (RETA); and (3)

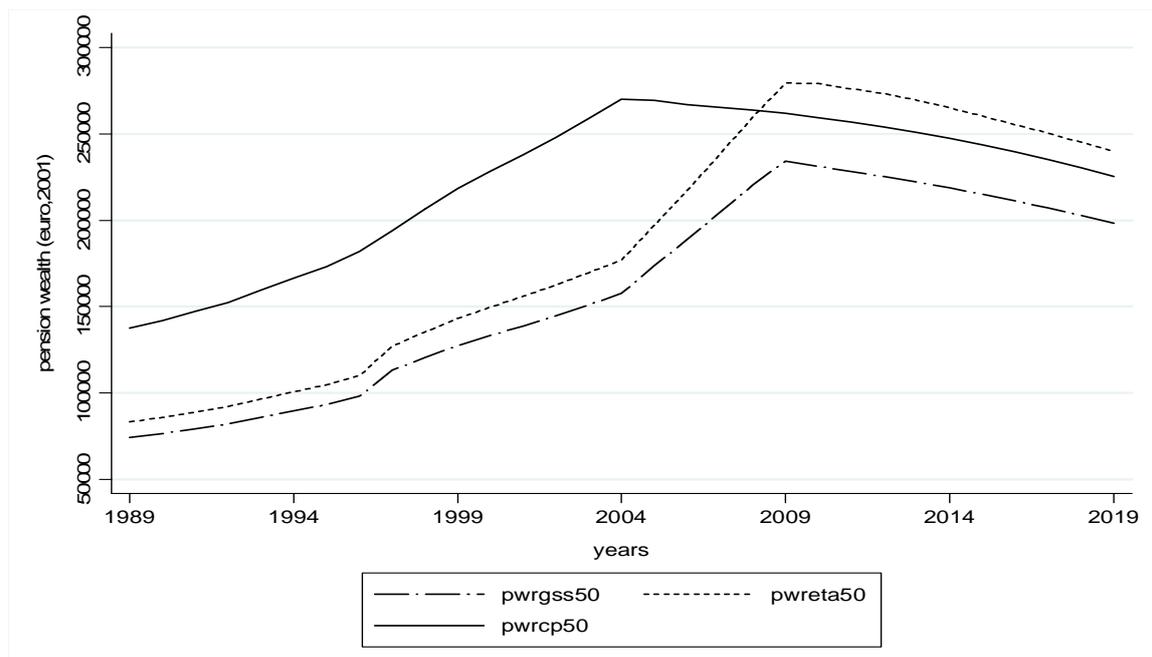
⁴ See Appendix for a detailed explanation of the assumptions in the computation of pension benefits.

⁵ The detailed description of this reform and changes in the rules which effects eligibility and amounts can be found in Appendix -Part 1.

the scheme for government employees (*Regimen de Clases Pasivas, or RCP*) includes civil servants employed by the central government, its local branches and universities.

For each of the defined pension schemes in the Spanish social security system, we compute pension wealth and the peak value which, as defined in section 3, captures the trade-off between retiring today and working until a year with a higher PW. Retirement incentives differ considerably across the three groups, as is illustrated for three prototypical individuals in Figure 1 (see also Appendix Part-2 for details on the estimation of pension incentives by age). Figure 1 presents pension wealth profiles for a man who is fifty years old in 1994 and has the following characteristics: a) Profile *pwr_{gss50}*: affiliated to RGSS regime, has worked since age 15, earns 12000 Euros a year, receives salary increases in line with inflation, has normal retirement age at 65, but can retire at age 60 with a penalty (b) Profile *pwr_{reta50}*: affiliated to RETA regime, same characteristics as the individual in Profile *pwr_{gss50}*, (c) Profile *pwr_{gcp50}*: RCP regime, same characteristics as the other two profiles, but early retirement is possible at age 60 without any penalty since he has been working for more than 30 years.

Before the age of 65 (age 60 for RCP), pension wealth increases because both the benefit base and replacement rate are rising (while the total number of periods that the worker receives a pension in the computation of pension wealth falls, the increase in benefit base and replacement rate is sufficient to compensate for this decrease). However, after age 65, the replacement rate is constant and although the benefit base is increasing, this does not suffice to compensate for the decrease in the remaining expected life span. Moreover, additional years of work add nothing to the expected pension amount and, as a result, pension wealth after age 65 decreases. Whether or not pension wealth increases depends on whether the rise in pension income from delaying retirement is sufficient to outweigh the fact that the pension would be received for one fewer year. In all three schemes, individuals are financially penalized for leaving prior to normal retirement age. There is typically no bonus for remaining in the scheme beyond the normal retirement age.

Figure 2.1: Pension wealth for stylized example, by pension scheme in 2001 euro⁶

Notes: *pwrss50* - Pension Wealth Profile for an individual that belongs to the RGSS regime. *pwreta50* - Pension Wealth Profile for an individual that belongs to the RETA regime. *pwrsp50* - Pension Wealth Profile for an individual that belongs to the RCP regime.

The patterns of pension wealth for the RETA and RGSS schemes are very similar. The RCP individual turns 60 in 2004, and has worked for more than 30 years, so he can retire with full pension in 2004. As a result, his profile peaks at age 60. Pension wealth in the RCP scheme is higher than in the private sector due to differing rules for civil servants. Their benefit base is higher than in other regimes and the replacement rate increases irregularly with seniority. While the rules are identical for RETA and the RGSS, their covered earnings are computed differently. The pension wealth profile is higher for RETA than for RGSS, because the legal minimum earnings in pension formulae applicable to RETA workers are higher than for RGSS workers. This rule attempts to neutralise the tendency for self-employed workers to underreport their income.

In addition to the computed pension wealth and financial incentives measures, we created three dummy variables indicating *when* the person could start drawing pension benefits: “public pension entitled after two periods” indicates that the person will not be able to draw pension benefits for at least two periods [$B(s, h) = 0$ & $B(s + 2, h) > 0$]; “public

⁶ Detailed descriptives of the pension wealth and incentive measures can be found in the Appendix .

pension entitled in next period” indicates that the respondent will be able to draw a pension from next year [$B(s,h)=0$ & $B(s+1,h)>0$]; finally, the dummy variable “public pension entitled this period” indicates the person is able to draw benefits currently or at some point during the current year [$B(s,h)>0$].

2.4.3 Measurement of health stock and health shocks

The relationship between observed and “true” measures of health has been a permanent concern for researchers. For lack of more objective data on health, most previous studies have used self-assessed health or other subjective reports of health limitations. This entails obvious potential problems of accuracy, endogeneity and justification bias in retirement and labour market transition models. Many recent studies have attempted to deal with these problems by constructing an underlying “health stock” for each individual and tracking longitudinal changes in this measure as a proxy for individual “health shocks” (eg, Bound *et al.* 1999, Disney *et al.* 2006, Hagan *et al.* 2008, Roberts *et al.* 2010). The latent health measure is then obtained as the predicted value of SAH, using supposedly more objective self-reported health indicators related to specific medical conditions and functional limitations as predictors. This is analogous to using the health indicators as instruments to ‘purge’ the measurement error from the SAH variable. This, of course, implicitly assumes that (i) SAH is the true variable belonging in the labour supply model and that (ii) the instruments can be legitimately excluded from such equation. While these “instruments” can be argued to be more objective self-reported measures of health than the usual five category SAH scale, it is not clear that they are valid instruments, in the sense that they cannot be argued to be entirely free from justification bias or measurement error themselves (Thomas and Frankenberg, 2002).

Our approach is based on the recognition that while some self-reported variables might be more objective than others, there is no single health variable in the ECHP (or any other similar survey) that can be considered the true health variable belonging in a labour outcomes model. All of these variables can only be considered indicators of true health, each capturing a different dimension of underlying unobservable health. In this sense there is an argument for not dismissing potentially useful information by opting for one or another variable as the “right” one for the model. Therefore, we construct a single health

indicator combining information from all health related variables included in the ECHP. This indicator can be thought of as a proxy variable for true unobservable health. Even if we were imperfectly proxying true health, the magnitude of the bias on the other explanatory variables in the retirement equation is smaller than if observed health were omitted. In addition, we still obtain the right sign on the effect of health (Wooldridge, 2002). Nothing prevents this proxy, of course, from being correlated with the error term in a labour transitions equation. In particular, there could be justification bias (an unobservable preference for leisure might be correlated with our index) or simultaneity bias. The longitudinal nature of our dataset allows us to use predetermined values for health which reduces our concerns about simultaneity bias.

The choice of health indicators from the ECHP consists of a set of self-reported health measures relating to limitation in daily activities, recent illness or mental problems and the history of inpatient hospital episodes. The definitions of the five variables used in the analysis are: (i) How do you rate your health in general? (SAH) (5 categories, very good to very bad); (ii) Are you hampered in your daily activities by any physical or mental problem, illness or disability? (3 categories; severely, to some extent, no); (iii) During the past two weeks, have you had to cut down things you usually do about the house, at work or free time because of illness or injury? (Yes/No); (iv) During the past two weeks, have you had to cut down things you usually do about the house, at work or free time because of an emotional or mental problem? (Yes/no); (v) During the past 12 months, have you stayed at least one night in a hospital? (Yes/no).

Because these health indicators are measured on ordinal or binary scales, which violate the standard multivariate normality assumptions, we use *polychoric* principal components analysis (PCA).⁷ The resulting index of latent health is a linear combination of the observed health-related variables. Table 2.4 presents the factor loadings of the observed variables used on our synthetic indicator of good health. It can be seen that the index is mostly driven by the “very poor” and “poor” categories of self-assessed health, by the “severe” category of health limitations, by the reporting of an illness or a mental health problem, and by the reporting of an inpatient hospital episode. Most health variables

⁷ Kolenikov and Angeles (2004, 2009) used a Monte Carlo simulation to show that failure to control for discreteness in variables leads to significantly inferior results. The standard PCA approach, especially when dummy variables are constructed from ordinal variables, has lower explained variance than the polychoric approach.

contribute substantially to the constructed health index, with the highest scores for self-reported health and mental health problems.

Table 2.4: Results of polychoric principal component analysis

Variable	Factor loading on health index
SAH	
Very bad	-1.3435
Bad	-0.8715
Fair	-0.4581
Good	-0.0489
Very good	0.5138
Illness	
Yes	-0.8724
No	0.0674
Mental prob.	
Yes	-1.0438
No	0.0104
Inpatient	
Yes	-0.6898
No	0.0419
Limitation	
None	0.3451
Some	-0.1676
Severe	-0.5668
% explained variance	0.5981

The identification of negative health shocks offers a convenient way to eliminate a potential source of endogeneity bias when the decision to retire may be related to a “health shock” i.e. a sudden sharp health deterioration (Disney *et al.*, 2006). Our two measures of health shock are based on the differences between two consecutive waves in an individual’s latent health index value.⁸ We created two binary indicators: a “small health shock” for a decrement of less than one standard deviation and a “large health shock” for a decrement of one or more standard deviations. This is similar to the concept of an acute

⁸ As a robustness check we also implemented the Bound (1991) and Kerkhofs and Lindeboom (1995) approaches to address issues associated with measurement error in SAH. This basically involves predicting SAH as a function of more ‘objective’ measures of health to define a latent health stock variable. We adopt an ordered probit (OP) and generalised ordered probit (GOP) model to allow for different thresholds when reporting SAH. Using these alternative health stock measures in the retirement models, we found the effects of health stock and health shocks to be very close in size and significance to our PCA based approach.

health shock as used by Riphahn (1999). Table 2.5 shows that deterioration in health is accompanied by the occurrence of both types of health shocks. Their prevalence increased across waves, occurring in 29.61 % of individual's in wave 2 and in 32.74 % in wave 8.

Table 2.5: Occurrence of small and large health shocks per wave

Wave	Health shocks (decrements)			Total
	Better or equal health	Somewhat lower latent health	Much lower latent health	
2	839 (70.38)	231 (19.37)	122 (10.23)	1192
3	699 (67.93)	200 (19.43)	130 (12.63)	1,029
4	566 (65.89)	183 (21.30)	110 (12.80)	859
5	508 (69.97)	134 (18.45)	84 (11.57)	726
6	430 (66.46)	132 (20.40)	85 (13.13)	647
7	362 (66.42)	98 (17.98)	85 (15.59)	545
8	306 (67.25)	89 (19.56)	60 (13.18)	455

Notes: Number of observed health changes and % of total (in brackets).

2.5 Models and estimation

As mentioned earlier we adopt the stock sampling with follow up with discrete time data set up.⁹ As shown by Jenkins (1995), this set up allows the estimation of hazard models using simple regression techniques for binary dependent variables. It is well known (Jenkins 1995, Lancaster 1990) that stock samples are not random. In our particular case this is so because we are sampling individuals who have lasted long enough in the labour force since they were first at risk of retiring. Although retirement before 50 in Spain is not common and serious bias is therefore unlikely, we account for this non-random selection by conditioning on survival up to the date of the stock sample. Such conditioning is achieved through the use of the reported age of entry into the labour market, which intervenes in the construction of all the variables related to pension incentives.

The left truncated nature of our data leads us to not consider hazard models with unobserved heterogeneity (frailty). As Jenkins (2005) warns, with left truncation “the convenient cancelling results used to derive an easy estimation method for discrete time

⁹ Recent examples with time discrete data include Disney *et al.* (2006), Hagan *et al.* (2008), and Roberts *et al.* (2010).

models no longer apply” if there is unobserved heterogeneity. There are methods to obtain consistent estimations for left truncated samples with unobserved heterogeneity (Meghir and Whitehouse, 1997), but for the sake of simplicity we rely on the richness of our explanatory variables to minimize the impact of any remaining unobservables and leave the testing of more sophisticated specifications including frailty for further work. We now proceed to a brief formal exposition of the econometric models we are about to estimate.

Duration in a labour market state, s , can be modelled using a hazard function (h) representing the instantaneous probability of leaving the state at time t , conditional on survival in the state until time t . A discrete-time representation of the continuous-time hazard rate can be defined as:

$$h_{it}^s = Pr[T_i = t | T_i \geq t, x_{it}] \quad (2.7)$$

where x_{it} is a vector of covariates that may vary with time, t , and T_i is a discrete random variable representing the time at which the end of the spell occurs.

With left truncation and no unobserved heterogeneity, the likelihood contribution for each individual is conditioned on survival from date first at risk through to stock sampling date (Jenkins, 1995). As shown by Jenkins (1995), in the discrete time case with no unobserved heterogeneity, the time periods prior to selection into the stock sample can be ignored; therefore one can work with the periods over which respondents are at risk after the stock sampling point.

The sample log-likelihood function of the observed duration data can then be simplified by defining a dummy variable $y_{it} = 1$ if $t=T_i$ and the individual is non-censored; and $y_{it} = 0$, otherwise. Accordingly, for individuals remaining in the labour market state of interest, $y_i = 0$ for all periods, while for those who exit the state, $y_{it} = 0$ for all periods except the period in which the exit occurs, when $y_{it} = 1$. The log-likelihood can then be written in a form familiar for the analysis of a binary variable y_{it} , where the unit of analysis is the spell period (see e.g. Allison, 1982; Jenkins, 1995):

$$\log L = \sum_{i=1}^n \sum_{k=1}^{t_i} y_{ik} \log \frac{h_{ik}}{1-h_{ik}} + \sum_{i=1}^n \sum_{k=1}^{t_i} \log(1-h_{ik}) \quad (2.8)$$

To complete the specification of the likelihood we need to choose the functional form for the hazard rate. We define a complementary log-log (*cloglog*) hazard rate, as it has the convenient property that it is the discrete counterpart of an underlying continuous-time proportional hazard model (Prentice and Gloeckler, 1978) such that:

$$h_{it} = 1 - \exp\{-\exp[\theta(t) + \lambda'(z_{it} - z_{it-1}) + \mu z_{i1} + \eta' s_{it-1} + \gamma'[(z_{it} - z_{it-1}) * s_{it-1}] + \beta' X_{it-1}]\},$$

$$i = 1, \dots, n, \quad t = 1, \dots, T \quad (2.9)$$

Where z_{it} is the health stock (and the changes in z_{it} are represented by dummy variables for small and large health shocks), z_{i1} is initial health stock in wave 1, s_{it-1} is a vector containing peak value and pension wealth, and x_{it-1} is a vector of predictors including socio-demographic and socio-economic characteristics (such as family and household characteristics, marital status, age, household income, type of job, proximity to drawing pensions). To assess whether the effects of financial incentives towards retirement differ when the individual has suffered a health shock, we introduce interaction terms between the small and large health shocks and the financial incentives variables. This is measured with the interaction vector $[(z_{it} - z_{it-1}) * s_{it-1}]$. When dealing with non-linear models, the interpretations of — and inference on — interaction terms require special attention (cf. Ai and Norton, 2003; Mullahy, 2008). First, the partial effect of an interaction term could be non-zero even if the directly estimated coefficient of the interaction term is zero. Second, we cannot rely on standard tests on the coefficients of the interaction term to test the statistical significance of the interaction effect. Third, the interaction effect is conditional on the independent variables and may have different signs for different values of the covariates. Therefore, to determine the magnitude of the interaction effects, it is necessary to compute the cross derivative (for continuous variables) or differences (for categorical ones). We used a bootstrapping procedure to obtain standard errors for the interaction terms.

2.6 Results

Table 2.6 presents the partial effect estimates for both definitions of retirement, standard and extended. For each model, the results are presented as partial (marginal) effects¹⁰, which measure how much the probability of retirement (extended retirement) is expected to increase or decrease for a unit change in an explanatory variable. Before examining the parameter estimates, it is useful to note that the unconditional probability for retirement (extended retirement) is 6.4% (9.1%).¹¹ Our results show that, as expected, healthier workers are *less* likely to retire: the initial level of health shows a strong and significant negative effect on the probability of retirement in both models. Indeed, workers whose health stock is one standard deviation below the mean are around 1% (2%) more likely to enter into retirement (extended retirement). The health shock effects also differ across the two definitions of retirement. Neither of the shocks - small or large - shows any significant effect on standard retirement. For the extended definition of retirement, however, the occurrence of a large health shock does significantly increase the probability of retiring by about 4 percentage points.

We find a significant positive effect of pension wealth on extended retirement (and a likewise positive although not statistically significant effect for retirement) and a significant negative effect of peak value for both retirement transitions. Whether these are downwardly biased estimates of the true effects, as the evidence by Belloni and Alessie (2009) would suggest for studies where true seniority is not available, cannot be tested with our data set. Nonetheless, in contrast to some of the studies cited by these authors as examples of downward bias, our estimates conform to expectations derived from the insights of the Option Value model.

Greater expected pension wealth increases the odds of extended retirement, while a financial reward to delaying retirement, in the form of a higher benefit level, increases the chances of continuing work. Pension wealth can be interpreted as an income effect, which

¹⁰ As an alternative, one can report exponentiated coefficients which measure the proportional effect on the underlying (instantaneous) hazard of a one unit change in a given variable (ratio by which the retirement probability changes for a unit change in a given variable). Due to the interpretations of interaction effects for financial incentives and health shocks, we report partial (marginal) effects and elasticities.

¹¹ That is, there are 6.4 exits into retirement (9.1 into extended retirement) for every observed 100 person-years at risk.

is consistent with the idea that leisure is a normal good. Pension accruals (or their peak values) are sometimes interpreted as “price effects”, as they reflect the relative cost, in terms of pension wealth, of not delaying retirement, and our estimates show a significant effect in the expected direction.

Table 2.6: Partial effects for models of retirement and extended retirement transitions

	Standard Retirement		Extended retirement	
	Partial Effects	S.E.	Partial Effects	S.E.
Initial health stock	-0.0099***	0.0031	-0.0199***	0.0046
Small health shock	0.00258	0.0059	-0.0043	0.0057
Large health shock	0.00206	0.00678	0.0413***	0.0116
Pension wealth	0.00035	0.00039	0.00118**	0.00057
Peak value	-0.00361***	0.00101	-0.00528***	0.00104
Small Shock *Pension Wealth	0.00179**	0.00075	0.00166**	0.00071
Large Shock *Pension Wealth	-0.00078	0.00097	-0.00637**	0.00251
Small Shock *Peak Value	0.00339**	0.00170	0.00335**	0.00162
Large Shock *Peak Value	0.00389*	0.00207	-0.00051	0.0025
Log household income	-0.0079**	0.0043	-0.0078	0.0053
High education	-0.0305***	0.0102	-0.0256*	0.0144
Middle education	0.0001	0.0136	-0.0166	0.0157
Female	-0.0127	0.0086	0.0285**	0.0119
Age minus 50	0.0248***	0.0073	0.0011	0.0053
Age minus 50 squared	-0.0002	0.0003	0.0007***	0.0003
House owner	0.0160	0.0107	0.0240*	0.0127
Has children	0.0143	0.0155	-0.0079	0.0174
Public pension entitled next period	0.0224***	0.0066	-0.0096	0.0088
Public pension entitled this period	0.0244*	0.0129	-0.0025	0.0155
Public sector employee	-0.0211**	0.0108	-0.0090	0.0140
Self-employed	-0.0345***	0.0080	-0.0155	0.0107
Unemployed	0.0425***	0.0158	0.0983***	0.0217
Housework	-0.0559***	0.0091		
Married	-0.0039	0.0100	0.0113	0.0113
Nr of Observations	4991		4267	

Notes: 1. Statistical significance at 1% level = ***, 5% = ** and 10% = *. 2. Average partial (marginal) effects are reported for dummy (continuous) variables. 3. We obtained cross partial derivatives to compute average partial effects for interactions and use bootstrapping to compute standard errors.

The size of the impact is greater for peak value: while the elasticity of the odds of retirement with respect to pension wealth is 0.090 (0.224 in the model for extended retirement), that with respect to peak value is -0.393 (-0.434 in the model for extended retirement). Another way to express the effect of these financial incentives is by considering the impact on the probability of retirement of a hypothetical increase in pension wealth or peak value. For instance, 10,000€ of additional pension wealth (about 6% of the average pension wealth in our sample) raises the probability of extended

retirement by 0.11% and retirement by 0.035%. An equivalent increase in peak value (in this case amounting to 14% of the average peak value in the sample) decreases the probability of extended retirement by 0.52% and retirement by 0.36%.¹²

The two variables indicating the time of entitlement to a public pension are strongly significant in the standard retirement model (amounting to an increase of 2.4% or 2.2% in the odds of retirement for those who can start drawing benefits either in the current year or during next year), but not in the extended retirement model. These results confirm that, even when controlling for pension wealth, people closer to the age of being able to draw pension benefits are more likely to retire than those who will not be able to draw such benefits for at least two more years. This raises some interesting questions about the potential presence of liquidity constraints (or illiquidity aversion) that are worth addressing in future research.

For other covariates, we find that higher income reduces the likelihood of retirement. This is consistent with the predictions of the OV model, as exposed earlier, and the effect is large in magnitude, as the elasticity with respect to household income is -1.732 (-1.202 in the extended retirement model, although the estimate is not significant). The probability of retirement increases with age.¹³ *Ceteris paribus*, women are significantly more likely to leave the labour market through the extended retirement route.¹⁴ The exit likelihood also differs by type of job. The self-employed are less likely to retire, *ceteris paribus*, whereas the unemployed are more likely to retire in comparison to those employed. Public sector employees are less likely to retire than those in the private sector. Being a house owner increases the probability of extended retirement. Higher education is associated with a lower probability of retirement. All remaining covariates were insignificant in both retirement models.

We now focus on the interactions between health shocks and financial incentives. In particular, we examine whether the effects of pension wealth and peak value differ

¹² Belloni and Alessie (2009), in the context of a comparable theoretical and empirical specification for the retirement decisions of Italian workers, find that increasing pension wealth by 10000 € would increase the odds of retirement by 0.095%. The corresponding figure for 10000 worth of extra peak value is a reduction of 1% in the probability of retirement.

¹³ We tested for a cubic polynomial in age in some specifications but the cubic term was never significant.

¹⁴ As a robustness check, we included interaction terms between female and financial incentives and health variables, but did not find any significant gender differences in retirement effects.

between workers in good health (i.e. who do *not* incur a health shock) and those in poor health (who *do* incur a health shock). Starting with the standard retirement model, we find that the marginal effects associated with the interaction of peak value with the occurrence of a large or a small health shock (estimated at 0.00389 and 0.00339 respectively) fully counteract the stand-alone marginal effects of peak value (estimated at -0.00361) on the retirement probability. This means that the small (elasticity of -0.393) but significant negative effect of peak value on the odds of retirement vanishes for workers who have endured a health shock. In the case of extended retirement, we find that the elasticity of peak value is more than halved (from -0.434 to -0.158) for those who experienced a small health shock. In contrast, the estimate for the interaction of peak value with a large shock in this model is insignificant and has a smaller order of magnitude. In the case of the interaction of pension wealth with the occurrence of health shocks, we find that small shocks *increase* by a statistically significant amount the average marginal effect of pension wealth. The corresponding elasticity increases from 0.224 to 0.539 in the model for extended retirement. On the other hand, larger health shocks significantly decrease the positive marginal effect of pension wealth for extended retirement from 0.0011 to -0.0051.

We now turn to examining the estimates for the interaction of financial incentives with health shocks. In the case of pension wealth, small health shocks contribute to reinforcing the main effect of making retirement more likely. The evidence is less clear cut in the case of large shocks because its point estimate is not significant in the retirement model and surprisingly negative and significant in the case of extended retirement. In the case of peak value, health shocks have a relatively larger impact. For standard retirement in particular, we find that the full effect of peak value vanishes among workers who have endured a health shock, regardless of whether it is a large or a small one. For extended retirement we also find that the effect of peak value is significantly dampened in workers who have endured a small health shock.

Our estimates mainly suggest that the effects of financial incentives on retirement vary with the occurrence of health shocks. In the case of pension wealth, there is a robust (i.e. significant and present in both models) exacerbation effect that makes retirement more likely when a small shock occurs, but the evidence is less robust in the case of large shocks. In the case of peak value, small and large shocks dampen its effect making retirement more likely in the retirement model, and small shocks do likewise in the

extended retirement model. These patterns are consistent with the *a priori* expectation that we have expressed in Section 3: despite the impossibility to predict the direction of the effects of health shocks on the chances of retirement through their impact on peak value and pension wealth, it is likely that a shock makes pension wealth more inciting to retirement and peak value less inciting to continue working.

2.7 Conclusion and discussion

Our study investigates the role played by health shocks, the financial incentives generated by pension schemes and their interaction in retirement decisions of older workers. In the context of an empirical version of the Option Value model, we explore the effect of health shocks on standard measures of financial incentives and pose a *subordination hypothesis*: the effects on the probability of retirement of forward looking financial incentives differ according to whether workers have suffered a health shock. Our theoretical discussion also suggests that — although other patterns for the impact of health shocks on such effects cannot be ruled out — it is likely that a health shock makes pension wealth more inciting to retirement and peak value less inciting to continue working.

We test these conjectures using data from the Spanish sample of the ECHP, with which we estimate models for two definitions of retirement —both old age standard retirement and an extended definition including inactivity- using empirical measures of health stock, health shocks, and financial incentives of the pension system. Our estimates show, in line with existing studies, that the stock of health is an important predictor of retirement. Also in line with theoretical predictions and existing estimates, we find that pension wealth increases the chances of retirement (elasticities of 0.090 and 0.224 for standard and extended retirement respectively) and peak value reduces the chances of retirement (elasticities of -0.393 and -0.434).

Our results show support for our *subordination* hypothesis: health shocks tend overall to make pension wealth more inciting to retirement and peak value less inciting to continue working. While their effect on the elasticity of retirement to pension wealth is modest, they completely wipe out the main effect of peak value for standard retirement. This finding is

in line with the results in Banks *et al.* (2007) who found the (negative) impact of peak value accrual on retirement to be significant only for those without health problems.

Our results have some interesting implications for the reforms of public pensions in Spain. A set of measures enacted in 2007 aimed at delaying retirement primarily through increases in the pension accruals after age 65, the statutory age of retirement for most workers at the time. Under these new rules, employees that continue working beyond the age of 65 would have seen the value of their state pension benefits increased by 2% per full year of work up until the age of 70 years.¹⁵ But given the low elasticity of retirement to peak value that we have estimated, it is expected that such measures will have little effect on delaying retirement. Moreover, our results suggest that these measures would be completely ineffective for workers who experience a health shock.

More recently, the poor state of public finances in Spain after the financial meltdown of the late 00's has prompted further reforms to the pension system. In 2011, the government and the trade unions agreed to raise the statutory age of retirement to 67. Our estimates suggest that such reform is likely to be more effective at delaying retirement, as one of the strongest explanatory variables in our models is the possibility to start drawing pension benefits.

To sum up, our findings highlight an important point: marginal changes to the financial incentives in pension systems will achieve less in terms of keeping older workers at work if these people are unable to respond to the incentives because of health problems. Therefore, such policies are to be considered in conjunction with appropriate health policies to contribute to the target of keeping older workers active for longer. This suggests that a potentially fruitful avenue for delaying retirement decisions would be to minimize the direct costs of working for individuals with some health deterioration. Technological advances that offer the possibility to work flexible hours and/or work from home should be exploited to their maximum potential in this sense. There is, finally, a positive message for

¹⁵ Also, if an employee had contributed to the pension scheme for 40 years at the age of 65, they would have received a higher annual increase of 3% in their pension benefits (LEY 40/2007, de 4 de diciembre, de medidas en materia de Seguridad Social).

the sustainability of the public pension system coming from the estimated effect of health. At face value our results suggest that a better health level does, *ceteris paribus*, reduce the likelihood of retirement. If technological progress and rising living conditions are to improve the average level of health, we should, *ceteris paribus* again, expect a reduction in the odds of retirement at any age. Of course, these gains –as far as sustainability is concerned- may be countered by other factors. An increase in the value of leisure, perhaps induced by higher levels of income, could well have larger effects than the likely effect of health improvement. We leave these issues for the future research agenda.

APPENDIX

Estimation of pension wealth

Rules of the RGSS (General Social Security Regime)

The RGSS is a pure pay-as-you-go scheme. Contributions are a fixed proportion of covered earnings, defined as total earnings, excluding payments for overtime work, between a floor and a ceiling that vary by broadly defined professional categories. Entitlement to an old-age pension requires at least 15 years of contributions. As a general rule, reciprocity is conditional on having reached age 65 and is incompatible with income from any kind of employment requiring affiliation to the Social Security system. Unless there are collective arrangements which prescribe mandatory retirement, individuals may continue working after age 65.

Computation of Pensions

In the computation of the pension benefits, we were somewhat constrained by the information available in the ECHP data (cf below). This forced us to make the following simplifying assumptions: (i) As data set covers private sector employees, self-employed workers and government employees, we simply assume that agricultural and domestic workers, sailors and coal miners also belong to the RGSS regime since the numbers of those individuals in these occupations are small in our sample. (ii) We only observe for each individual the age of the entry into the labour market and assume a continuous uninterrupted working career thereafter and compute seniority as the difference between age and the age of entry in to the labour market (this may be a strong assumption for females in our sample, but because of the minimum pension rules in Spain, for those who have interrupted career this assumption should not generate measurement error) (iii) we assume that the individual's occupational status (whether private sector, self-employed or civil servant) does not change over time and remains as it is in year 1994; (iv) we observe the actual earnings from employment and self-employment in the ECHP. Contributions are a fixed proportion of "Covered earnings", excluding payments from overtime work,

between a floor and a ceiling that vary by broadly defined professional categories. Thus, covered earnings are computed by using the actual earnings (v) we only compute the retirement benefits in this study (not disability or unemployment)

Earnings Projections

For the RGSS, first we divide the individuals into two groups: high skilled versus semi and non-skilled based on the variable “Occupation in current job” in the ECHP. High skilled individuals belong to the following occupations: Legislators, senior officials, Corporate managers, Managers of small enterprises, Physical, mathematical and engineering science professionals, Life science and health professionals, Teaching professionals, Other professionals, Physical and engineering science professionals, Life science and health associate professionals, Teaching associate professionals, Other associate professionals. Semi and non skilled individuals are: Office clerks and Customer services clerks, Personal and protective services workers, Models, salespersons and demonstrators, Skilled agricultural and fishery workers, Extraction and building trades workers and Other craft and related trades workers, Metal, machinery and related trades workers and Precision, handicraft, printing and related trades workers, Stationary-plant and related operators and Drivers and mobile-plant operators, Machine operators and assemblers, Sales and services elementary occupations, Agricultural, fishery and related labourers, Labourers in mining, construction, manufacturing and transport.

The specification of the model for earnings projection represents an essential step in the estimation of pension wealth at the individual level. For the backwards projection of earnings (earnings before the year 1994), we have used the following formula by assuming that individual earnings grow at the annual average growth rate of aggregate earnings until 1994 (Spanish National Institute of Statistics).

$$Earn_{t-1} = Earn_t * (1 - Rate \text{ of Earnings Growth }_t) \quad (2.10)$$

Similarly, we projected (expected) earnings forward (i.e. after 2001) as follows:

$$Earn_{t+1} = Earn_t * (1 + \Delta CPI_{t+1}) \quad (2.11)$$

We have assumed zero real earnings growth (i.e. earnings growth equal to CPI growth) in the forward projection of earnings.

Covered Earnings:

Covered earnings are defined as actual earnings only if they lie within a legally specified interval. In other words, if actual earnings exceed (or fall below) the specified intervals, then covered earnings are set equal to the upper (lower) value of the interval. The legal intervals vary by year and professional category. We have used the following floor and ceiling categories in the computation of covered earnings: (i) Minimum earnings for the RGSS; (ii) minimum earnings for self-employed (RETA) differentiated for individuals younger and older than 51; (iii) maximum earnings for two categories in RGSS, high skilled versus semi and non-skilled workers; actual earnings are replaced by the legal limits, if they are higher or lower. By this way, we obtain an estimate of the covered earnings of each individual, in each regime.

Benefit Computation

In order to compute the yearly pension for our sample, we have to define the benefit base (*base reguladora*) in the first step. Benefit base BR_t is a weighted average of covered yearly earnings over a reference period that consists of the last 8 years before retirement;

$$BR_t = 0.125 \left[\sum_{j=1}^2 W_{t-j} \right] + 0.125 \left[\sum_{j=3}^8 W_{t-j} (I_{t-2} / I_{t-j}) \right] \quad (2.12)$$

where W_{t-j} and I_{t-j} are earnings and the consumer price index in the j-th year before retirement.

If the eligibility conditions are met, the individual who retires at age 65 receives the initial yearly pension P_t : $P_t = \alpha_n BR_t$, where α_n is the replacement rate. It depends on the number of years of contribution “n” and on the age of the retirees and equals :

$$\alpha_n = \left\{ \begin{array}{ll} 0, & \text{if } n < 15, \\ 0.6 + 0.02(n - 15), & \text{if } 15 < n < 35, \\ 1, & \text{if } n > 35 \end{array} \right\}, \quad (2.13)$$

for age equal or larger than 65 until 1997. As of 1997, a pension reform was implemented as follows. From 1997 onwards, the number of reference years was increased by one every year until 2001, to reach a total of 15 years. Moreover, the replacement rate rules were changed to the following:

$$\alpha_n = \left\{ \begin{array}{ll} 0, & \text{if } n < 15, \\ 0.5 + 0.03(n - 15), & \text{if } 15 < n < 25, \\ 0.8 + 0.02(n - 25), & \text{if } 25 < n < 35, \\ 1, & \text{if } n > 35 \end{array} \right\} \quad (2.14)$$

Early retirement

The normal retirement age in Spain is 65, but early retirement at the age of 60 is permitted as a general rule for individuals that became affiliated to the Social Security System before 1967. However, a financial penalty is incurred by those who retire early between the age of 60 and 65; the replacement rate is reduced by 8 percentage points for each year under age 65. This means that the replacement rate rules for early retirees are as follows depending on age and the year which the individual started working:

$$\alpha_n = \left\{ \begin{array}{ll} 0, & \text{if } \text{age} < 60, \\ 0, & \text{if } 60 \leq \text{age} < 65 \text{ and start working after 1967} \\ 1 - 0.08(\text{age} - 60), & \text{if } 60 \leq \text{age} < 65 \text{ and start working before 1967} \\ 1, & \text{if } \text{age} \geq 65 \end{array} \right\} \quad (2.15)$$

As of 1997, workers who retire after the age of 60 with forty or more contributive years are charged a penalty of only 7 percent for each year under age 65. We have made the following assumption in the computation of incentives before the age of 60. When a person stops working between 55 and 59, his/her pension is computed considering earnings until that age, even if he/she starts receiving the pension at age 60.

Regime for Government Employees (RCP)

In this section we describe the main differences with the RGSS scheme. Public servants are divided into five categories by their schooling level, age and skills. A. High skilled, high educated, older than 23 (for collage graduates). B. High skilled, high educated, younger than 23 (for people holding certain kinds of college diplomas). C. High skilled, middle

educated (for high school graduates). D. Semi or non skilled, middle educated (for junior high school diplomas). E. Semi or non skilled, low educated (for lower education levels). For each of these categories, the budget law defines every year a theoretical SS wage (*haber regulador*) which is used to compute the pension wealth.

The basic yearly pension of a civil servant is computed using the same formula as for the RGSS, $P_t = \alpha_n BR_t$, but the replacement rate for the RCP depends (approximately, since the number of years worked has changed frequently over time) on the age and number of years on work as follows:

$$\alpha_n = \left\{ \begin{array}{ll} 0, & \text{if } age < 60 \\ 0, & \text{if } n < 15 \text{ and } age \geq 60 \\ \min(1, 1 - 0.0366(35 - n)), & \text{if } 15 < n < 30 \text{ and } age \geq 60 \\ 1, & \text{if } n > 30 \text{ and } age \geq 60 \end{array} \right\} \quad (2.16)$$

The differences with respect to the general scheme are various. The entitlement to a pension requires at least fifteen years of contributions, the replacement increases irregularly with seniority. RCP allows for early retirement at age 60, without any penalty for public servants with at least 30 years of service. Unlike the general scheme, the RCP imposes mandatory retirement age at age 65. The rules of the RCP scheme were not affected by the 1997 reform.

Regime for the Self – Employed (RETA)

While the social security tax rate is the same for RGSS and RETA covered earnings are computed differently, as the self-employed are essentially free to choose their covered earnings between a floor and a ceiling legislated annually. As mentioned in Jimenez-Martin et al. (2004), because of the strong progressivity of Spanish personal income taxes, a suspiciously large proportion of self-employed workers report earnings equal to the legislated floor. For individuals reporting earnings below these legal limits we simply assume that covered earnings are equal to the legal minimum (separately for those over and under 51). For all others, we have equated covered to reported earnings. Pension benefits computation for self-employed then uses the same formulae as for the general regime. A crucial difference with respect to the RGSS is that, under RETA, reciprocity of an old age pension is compatible with maintaining the self-employed status. Current Spanish legislation allows the self-employed to begin drawing retirement pensions without retiring,

at least as long as they keep managing their own business. As a result, the opportunity cost of retiring for the self-employed is not measured by the loss of future earnings but instead, by the fact that contributions cannot longer be accumulated to increase future pensions and that marginal income taxes must be paid on pensions. This means that the maximization of the social security payoff is a very reasonable objective function for the self-employed.

Maximum and Minimum Pensions

Pensions are subject to a ceiling set annually and roughly equal to the ceiling on covered earnings. If the computed old age pension is below a minimum, then a person is paid an annually set minimum pension. Minimum pensions are higher for those who are older than 65. In addition, the minimum pension for the RCP regime is higher than the RGSS and RETA regime.

Main assumptions in the computations:

1. We assume that the individual's pension wealth is unaffected by whether the individual has a spouse or not.
2. We assume that no person has access to early retirement before age 60 if he/she has not filled sufficient years of contributions. The first of these assumptions is largely innocuous. While marital status affects the size of pension benefits in some circumstances, the impact is very modest. The second assumption is forced by data limitations, as workers in some dangerous occupations can retire at earlier ages, but we cannot tell whether the workers qualify for such treatment. In any case, the incidence of such possibility is very small in the population of workers.

In order to compute pension wealth we need estimates of survival probabilities which we have taken (for 1994) from the life tables in the Human Mortality Database¹⁶.

¹⁶ Web Site: <http://www.mortality.org>

2. Additional tables

Descriptives of pension wealth, accrual and peak value

Table 2.7 shows the median of pension wealth, accrual and peak value incentives, as well as the first and ninth decile of accrual and peak values in our sample.

Table 2.7: Pension Wealth, Accrual and Peak Value Incentive Measures for all regimes (in 2001 Euros)

Age	Obs	Median PW	<u>Accrual Value</u>			<u>Peak Value</u>		
			P10	P50	P90	P10	P50	P90
55	551	138,185	3,667	7,446	17,323	12,934	62,885	161,880
56	578	139,800	3,593	7,906	18,374	17,784	61,763	153,644
57	625	132,891	3,505	7,172	16,628	15,282	57,679	144,002
58	591	130,839	3,411	6,914	15,847	14,581	53,135	137,008
59	550	126,569	3,276	6,684	16,544	12,102	51,420	128,990
60	512	120,014	-694	13,937	32,536	2,629	56,013	123,953
61	496	129,367	-1,373	12,258	30,770	897	41,390	101,909
62	437	137,101	411	11,581	33,015	895	32,058	81,165
63	397	150,824	-1,055	9,906	24,781	390	19,551	60,289
64	388	155,373	-1,294	7,853	22,390	-809	9,152	35,843
65	301	159,009	-6,410	-1,420	6,142	-6,217	-831	14,106
66	227	150,181	-8,032	-1,395	4,760	-8,032	-1,206	10,100
67	159	146,647	-7,058	-2,157	1,994	-7,058	-2,157	2,957
68	118	140,280	-5,658	-2,814	502	-5,658	-2,814	502
69	75	122,982	-6,195	-2,566	455	-6,195	-2,566	455

The median PW starts off at 138,185 Euros and peaks between 64 and 65 years of age at 159,009 Euros. Remember that PW reaches its maximum value just at the age which the worker is allowed to retire (normal retirement age). The median of pension wealth decreases from age 57 to 60 and rises again after the age of 60 until 65. A negative accrual can be interpreted as a tax on further labour force participation. The 10th percentile of the accrual value is positive until age 60 and turns negative above 60. Again this is consistent with the fact that pension wealth cannot decrease by postponing retirement one year for ages under 60. The median accrual is positive until age 65 and becomes negative after age 65: the increase in the pension for each additional year is too weak to compensate the loss of one year of pension. The accrual after age 60 is negative in 10% of the distribution. For

those individuals, the implicit tax rate on continuing working can be higher; if these individuals are also on low income, the effect on incentives for early retirement may be strong.

The median peak value and accrual value show similar profiles. However, from age 55 to 64, the median peak value is much higher, reinforcing retention incentives in that age range.

Chapter 3

Early retirement and disability:

Effects of new adverse health events and pension incentives

We examine how new adverse health events and financial incentives affected the likelihood of a transition into early retirement and disability for older female and male Dutch health care workers in the period 1999-2003. We also study their interaction by testing a subordination hypothesis (are financial incentives being subordinated after a health shock?) and an affordability hypothesis (can wealthier older workers afford to retire earlier after a health shock?). Unanticipated hospitalizations are used as health events and linked to information on retirement decisions and incentives from administrative records of the pension funds of health care workers. Not surprisingly, we find that health events have greater impact on entry into disability while financial incentives affect early retirement exits. In addition, pension wealth and peak value incentives also explain disability entry. This suggests that in those days disability acted as an alternative retirement route. We find more support for the *affordability hypothesis* – wealthier individuals are more likely to retire for health reasons – than for a *subordination hypothesis* - health events hardly reduce the impact of pension incentives.

3.1 Introduction

Due to the generous early retirement (ER) and disability insurance (DI) systems prevailing in the 80s and 90s in the Netherlands, labour force participation of older workers decreased substantially. Due to the fiscal implications of the aging population, these ER and DI arrangements were no longer sustainable. In order to ease the financial burden imposed on the Social Security System, several reforms have recently been proposed, including the abolishment of ER schemes, a rise of the normal retirement age and tightened eligibility criteria for DI (Euwals et al, 2010b). The effectiveness of such policies depends on whether and how workers can respond to financial incentives such as pension wealth in the presence of health problems (Gruber and Wise, 2004). In particular, financial incentives may have different effects on retirement transitions for individuals who experience health shocks or deteriorations (Banks et al, 2007; Erdogan-Ciftci et al, 2011). Erdogan-Ciftci et al (2011) have labelled this the *subordination hypothesis*. It asserts that financial incentives have a stronger effect on retirement decisions of healthy workers but are severely dampened when a health shock occurs. In addition, while financial incentives are important in determining the timing of retirement, they may be less important in explaining exits from work which might be considered less voluntary such as disability. The effects of financial incentives on claiming DI benefits have however received less attention in the literature compared to their effects on the ER route.

There is a consensus that financial incentives from social security and pension plans are important determinants of the transitions into retirement and they have contributed to the decrease in labour force participation of older workers (Lumsdaine and Mitchell, 1999; Gruber and Wise, 2004; Lindeboom, 2012). ER financial incentives may however have an effect not only on the probability that an older worker transits into early retirement, but also on other pathways. Kerkhofs et al (1999) estimate a competing risk model for transitions from employment to early retirement, disability and unemployment for the Netherlands for the years 1993-1995. They find that ER eligibility reduces the hazard of DI and unemployment insurance (UI). Duggan et al (2007) evaluate the effect of the reduction in the generosity of Social Security retired worker benefits in the US in 1983 on the enrolment in the disability scheme¹. Their estimates indicate that a \$5000 reduction in the

¹ The official name of the US DI scheme is Social Security Disability Insurance (SSDI).

old-age social security benefits induced a 0.39 percentage points increase in SSDI enrolments. This reform can explain one-fourth of the enrolment increase for females and one-third for males. In our analysis we evaluate the effect of ER incentives on transitions into ER and DI².

Ill-health has been found to increase the likelihood of ER and DI claims at older ages (Bound et al. 1999; Currie and Madrian, 1999; Kerkhofs et al 1999; Disney et al. 2006; Lindeboom 2006a, Lindeboom and Kerkhofs, 2009). Most studies focusing on the effects of health on labour supply rely on subjective health measures, which will lead to an overestimation of the impact on employment when health problems are overreported as justification for inactivity. The use of self-reported health indicators to instrument reported work incapacity may also cause bias in the same direction (Bound, 1991). Previous studies typically had to rely on crude health controls, such as changes in self-reported health status, or other subjective health measures rather than actual medical events not available in socioeconomic surveys (Bound et al. 1999; Disney et al. 2006; Banks et al. 2007; Hagan et al. 2008; Roberts et al. 2010). It is therefore not easy to disentangle the underlying causal mechanism between ill- health and labor supply due to the reverse causality (work effects health) and unobservables that relate to both health and work. Therefore, in order to identify the effect of health on ER and DI outcomes, we need independent variation in health status. Adverse health shocks gathered from hospitalization data provide a source of unanticipated variation in health status, which can be used to identify the causal effect of the health on work status (Lindeboom 2006a, 2006b).

The evidence on the effect of a new health event on labour supply using hospitalizations as an exogenous measure of health changes is limited. Møller Dano (2005) uses road accidents recorded for a 10% sample of the Danish population and finds a significant effect on employment only for males, for whom the employment rate decreases by around 10% after an accident and does not recover in the following six years. Halla and Zweimüller (2011) restrict attention to accidents experienced on the way to and from work, which they argue are less likely to induce selection problems. They find negative effects on employment (4 percentage points on average) and on earnings, conditional on remaining in employment. The effects are larger for individuals that are less attached to the labour

² UI at older ages in the Netherlands hardly qualifies as a pathway (Euwals et al, 2011). We do not investigate this route in this paper.

market, such as females and blue-collar workers. García-Gómez et al. (2011b) use sudden illnesses represented by acute hospitalizations for the Dutch population to identify the causal effects of health shocks, on employment and income. They show that, on average, an acute hospital admission lowers the employment probability by seven percentage points and results in a 30% loss of personal income for those entering disability insurance two years after the shock.

There is a large body of literature that explores the role of ill-health and financial incentives on transitions out of employment. However, the interaction of these two dimensions is poorly understood. On the one hand, the effect of financial incentives may depend on health status. In particular, financial incentives alone may not be sufficient if individuals cannot work at older ages due to shocks or deteriorations in health (Banks et al, 2007; Erdogan-Ciftci et al, 2011). Erdogan-Ciftci et al (2011) label this as *subordination hypothesis* and show that financial incentives exert a stronger effect on the chances of retirement for healthy workers and that, in the presence of a health shock, the financial incentives embodied in pension rules are dampened. Banks et al (2007) also find that the opportunity cost of retiring today in terms of foregone future pension benefits only exerts a significant effect on the likelihood of retirement for those in good health. This paper also tests this *subordination hypothesis*.

On the other hand, the labour supply response following an adverse health shock may also depend on the pension wealth of older workers. Banks (2006) show striking differences in labour force exit routes in the UK: older poor individuals are more likely to enter DI while the older rich enter retirement. García-Gómez et al (2011b) look at the effects of health shocks on employment by income groups and show that a health shock is both more likely to occur and to have a larger relative impact on employment and income at the bottom of the income distribution. Our contention here is that the increase in the likelihood of non-employment following a sudden health event would be higher among those individuals with higher pension wealth as they can “afford” to withdraw from the labour market. To the best of our knowledge, this is the first paper that investigates this *affordability hypothesis*.

We use a particularly rich dataset that links benefit information from the second largest pension fund in the Netherlands (the health care pension fund PFZW), hospital admission

records and socio-demographic information from municipality registers for a five year period. It contains very accurate information on pension and early retirement rights of employees, i.e. the exact pension wealth and marginal incentives. In addition, we exploit three sources of exogenous variation to identify the effect of financial incentives: i) reduction in early retirement replacement rates over successive birth cohorts ii) individual variation in the length and size of contributions to the scheme and iii) eligibility conditions depending on the year of birth and tenure. Moreover, to circumvent the health reporting problem and to exploit health variation that is arguably exogenous to labour market outcomes by virtue of being unexpected, we identify a subsample of hospital admissions that are likely to correspond to a new unexpected health problem if the individual did not incur a hospital admission in the previous year.

The paper is organized as follows. In the next section, we discuss ER and DI schemes in the Netherlands and in the health care sector. Section 3.3 presents the pension incentives in the health care sector pension fund. Section 3.4 presents our modeling strategy for the effects of financial incentives and health shocks for ER and DI transitions. Section 3.5 describes the data. Section 3.6 contains the results. We conclude with a discussion of the implications of our findings.

3.2 Early retirement (ER) and disability insurance (DI) in the Netherlands in 1999-2003³

There are two important components in the Dutch pension system. The first is the mandatory state pension which is financed on a pay-as-you-go basis and which pension benefits are not related to working history neither means-tested. In addition to the state pension, most employees can expect a mandatory occupational pension after sixty-five, which is capital funded and mostly defined benefit. There are also early retirement schemes for some workers under sixty-five (cf. below). In addition, the Social Security system in the Netherlands offers unemployment benefits, disability benefits and social assistance which expire when someone turns sixty-five.

³ This section is largely based on Euwals et al (2010b) and Trevisan and Euwals (2011).

3.2.1 Early retirement of health care workers

During the 1980s, many sectors introduced early retirement schemes independent of the capital-funded occupational pension system from age sixty. Under this scheme, a worker could retire and receive early retirement benefits of about 80% of the last earned wage in gross terms and the net replacement rate was usually higher. The schemes were financed on a pay-as-you-go basis and were highly actuarially unfair; an additional year of work would not lead to an increase in the replacement rate. As a result, the implicit tax rate on continuing working for one more year was about 100% (de Vos and Kapteyn, 2004), implying that the benefit level hardly increases when retirement is postponed. Empirical studies typically find that early retirement schemes contributed to the falling participation rates of men aged 55 to 64 in the past (Gruber and Wise, 2004, Euwals et al 2010a).

In the 1990s, it became clear that the pension and welfare system would not be sustainable in the longer run. A series of reforms followed in the next decade with two main goals: to remove the implicit tax on continuing to work and to limit the costs in order to guarantee the sustainability of the system. Therefore, unions and employer organizations agreed to gradually switch to an actuarially fair early retirement system. From January 1, 1999 onwards, health care workers could no longer access the actuarially unfair benefit. A transitional early retirement scheme, OBU⁴, was introduced for workers close to sixty who would have qualified for the old scheme. In particular, workers born before 1949, who did not receive a disability benefit, and who had been working for ten years in the sector became eligible for OBU-benefits. Replacement rates in the OBU scheme were as follows: workers born in 1948 get 71%, workers born in 1947 get 72%, workers born in 1946 get 73% and so on. Finally, workers born before 1939 get a replacement rate of 80%. During the period of early retirement, members continued to accumulate old-age-pension-rights. In addition, the scheme offered the opportunity of part-time retirement at age 58 or 59. Workers who had access to an early retirement benefit at age 60 could retire at age 58 (59) and receive 50% of the benefit during ages 58 to 61 (59 and 60). From age 62 (61) onwards they received the full 100% of the benefit.

⁴ OBU stands for ‘*Overbrugginpensioen*’ (‘Bridge pension’).

The OBU-scheme was abolished from January 1, 2006. Since then, early retirement is integrated into the capital-funded occupational pension system. Early retirement before age 65 is still possible and the special fiscal treatment in case of retirement before age 65 stays in place as long as the pension benefit is adjusted actuarially fair.

3.2.3 Disability Insurance

All employees in the Netherlands are covered by Disability Insurance (DI), regardless of their work history. Workers are entitled to (partial) DI benefits if they have a degree of disability, based on reduced earning capacity, of at least 15%⁵. The scheme distinguishes between fully and long-lasting disabled and temporarily and/or partially disabled. Individuals who are both fully and permanently incapacitated qualify for earnings-related benefits with a replacement rate of 75%. Individuals who are partially disabled or temporarily fully disabled may either qualify for earnings-related benefits or benefits which are based on the minimum-wage level, depending on the remaining work capacity and employment history.

During the period under analysis here, DI benefits were paid after a waiting-period of one year⁶. Until then, the employer is responsible for financing sick pay, which is equal to 70% of the gross wage. However, collective bargaining agreements usually ensure that sick employees receive up to 100% of their net salary (Burkhauser et al., 2008). Replacement rates are defined in terms of previous net salary excluding overtime or bonuses (so actual replacement rates on disposable income may be below 100%) and vary by firm or sector (van Vuren and van Vuuren, 2007).

DI pays benefits in two phases. In the first, which lasts up to six years depending on age at onset of disability⁷, the recipient receives a percentage of the previous wage. The percentage is based on the severity of the illness or injury up to a maximum of 75% if the individual is assessed as 80% disabled or more. The partially disabled, who represent around 20% of all recipients (OECD, 2009), receive *pro rata* benefits and are allowed to

⁵ The minimum disability level required for entitlement to DI was increased from 15% to 35% in 2006, which is outside our observation period.

⁶ The waiting period was extended to two years in 2004.

⁷ More specifically, the entitlement period ranges from 0 years for those under 33 to 6 years for those over 58 years of age.

work to close the earnings gap. Two thirds of those awarded partial benefits work, and for them the benefit acts as a wage subsidy (García-Gómez et al., 2011). After this first phase, the benefit is no longer set only in relation to previous wage and the severity of disability but is equal to the minimum wage plus an addition increasing in age and previous wage. In all cases, this follow-up benefit is constrained to be lower than that paid in the first phase, but it can be paid until the individual reaches the age of 65. Individuals can choose whether to insure against the difference between initial and follow-up benefits, and in most cases this is part of the collective bargaining agreement (De Jong, 2008), which implies that in those cases there is no difference between the first and second phase.

3.3 Pension incentives for health care workers

In modeling retirement and disability, we follow the option value retirement model of Stock and Wise (1990), which assumes that individuals compare the expected present value of retiring immediately (in utility terms) to the expected present value of continuing to work and holding the option of retiring in the future. Thus, for each period that the individual continues working, this decision is re-evaluated. Adoption of this model involves the computation of the option value variable (or some close counterpart) and estimation of its effect on retirement. As shown by Stock and Wise in their original and subsequent work, and by Coile and Gruber (2007), the option value is a comprehensive measure of future retirement incentives. In this paper we consider the “peak value” model, one of the versions of the option value model, in which individuals compare the total discounted income that they receive if they would retire today to the total discounted income that they would receive if they would retire in the year in which they would receive the highest discounted benefits. Pension wealth is interpreted as the income effect consistent with the idea that leisure is a normal good. Therefore, the higher the pension wealth, the higher the probability of a labour market exit. The Peak Value on the other hand, is interpreted as a price effect as it reflects the relative cost, in terms of pension wealth, of not delaying retirement. We also evaluate the effect of the “implicit tax on work” which compares earnings and pension benefits. The higher the implicit tax on work, the higher is the probability of early exit from the labour market.

Pension Wealth (PW) is calculated as the net present value⁸ of the pension benefits that an individual retiring at age t would receive:

$$PW_{it}(R) = \sum_{s=R}^{64} \gamma_t \delta^{(s-t)} B_t^{ER}(R) + \sum_{s=65}^T \gamma_t \delta^{(s-t)} B_t^P(R) \quad (3.1)$$

where s is the current age, R is the retirement age, δ is the discount factor⁹, γ is the survival probability¹⁰, B_t^{ER} are the early retirement benefits, and B_t^P are old age pension benefits.

The peak value is the maximum difference in PW between retiring at a future age and retiring at the current age:

$$PV_{it} = \max(PW_{iN} - PW_{it}) \quad N = t+1, \dots, M \quad (3.2)$$

where M is the mandatory retirement age. The implicit tax of postponing retirement from age t to age $t+1$ is given by:

$$IT_{it} = -\frac{PW_{i,t+1} - PW_{it}}{W_{i,t+1}} \quad (3.3)$$

where $W_{i,t+1}$ is the wage at age $t+1$.

Three sources of variation help us to identify the effect of financial incentives on early retirement behaviour. First, the replacement rate of the OBU scheme varies by year of birth; it decreases by one percentage point per year of birth. Second, there is individual variation in the length and size of contributions to the scheme. Third, individuals born after 1948 (regardless of their work history in the sector) are simply not eligible for OBU.

⁸ See Euwals *et al.* (2010b) for further details on calculation of NPV and analysis of actuarial fairness. Computations of pension wealth and marginal incentives are based on Trevisan and Euwals (2011).

⁹ The discount factor is 3% for all individuals (hence discount rate is 0.9708).

¹⁰ Survival probability tables were provided by PFZW.

3.4 Models and estimation

We are interested in the effects of health shocks and financial incentives on two different labour market transitions: early retirement and disability. We study these transitions separately. Thus, our sample of interest consists of those individuals who are working (active) in the first wave of the survey and we follow these until they first become early retired, disabled or are censored.¹¹ We ignore transitions back to work; both retirement and disability are considered as absorbing states.¹²

Duration in a labour market state can be modelled using a hazard function representing the instantaneous probability of leaving the state at time t , conditional on survival in the state until time t . A discrete-time representation of the continuous-time hazard rate can be defined as:

$$h_{it} = \Pr[T_i = t \mid T_i \geq t, x_{it}] \quad (3.4)$$

where x_{it} is a vector of covariates that may vary with time, t , and T_i is a discrete random variable representing the time at which the end of the spell occurs.

With left truncation, the likelihood contribution for each individual is conditioned on survival from date first at risk through to the stock sampling date (Jenkins, 1995). As shown by Jenkins (1995) in the discrete time case with no unobserved heterogeneity, the time periods prior to selection into the stock sample can be ignored, and therefore one can work with just the periods that respondents are at risk after the stock sampling point.¹³

The sample log-likelihood function of the observed duration data can then be simplified by defining a dummy variable $y_{it} = 1$ if $t=T_i$ and the individual is non-censored; and $y_{it} = 0$,

¹¹ Censoring occurs if an individual drops out of the survey or remains in the survey but fails to exit employment by the end of the survey period. We censored observations that leave employment using a different route. This is 8.9% (2.1%) for females and 7.9% (1.5%) for males for ER (DI) route.

¹² We don't observe many transitions back to work or to another state from retirement or disability in our sample. For instance, percentage of transitions: i) from ER to employment is 0.03%, and ii) from DI to employment is 0.008% for females.

¹³ With unobserved heterogeneity, the cancellation of the periods before the stock sampling date result no longer holds, and one has to consider the information about what happened before the stock sampling date. In this case, the unconditional survival function becomes an integral over an expression that depends on the individual heterogeneity component (which requires integrating out the heterogeneity). This raises some complications in the estimation and studies that control for it in the left truncation case are rare (Meghir and Whitehouse, 1997).

otherwise. Accordingly, for individuals remaining in the labour market state of interest, $y_{it} = 0$ for all periods, while for those who exit the state, $y_{it} = 0$ for all periods except the period in which the exit occurs, when $y_{it} = 1$, after which the observation drops from the sample. The log-likelihood can then be written in a form familiar for the analysis of a binary variable y_{it} , where the unit of analysis is the spell period (see e.g. Allison, 1982; Jenkins, 1995):

$$\log L = \sum_{i=1}^n \sum_{t=1}^{T_i} y_{it} \log \frac{h_{it}}{1-h_{it}} + \sum_{i=1}^n \sum_{t=1}^{T_i} \log(1-h_{it}) \quad (3.5)$$

To complete the specification of the likelihood we need to choose the functional form for the hazard rate. We define a complementary log-log (*cloglog*) hazard function, as it has the convenient property that it is the discrete counterpart of an underlying continuous-time proportional hazard model (Prentice and Gloeckler, 1978) such that:

$$h_{it} = 1 - \exp\{-\exp[\theta(t) + \beta'X_{i,t-1} + \alpha'S_{i,t-1} + \phi'Z_0 + \gamma'W_{i,t-2}]\} \quad (3.6)$$

where $S_{i,t-1}$ is the health shocks between $t-2$ and $t-1$, Z_0 includes controls for initial health, $W_{i,t-2}$ is the vector of financial incentives and pension wealth in wave $t-2$ and $X_{i,t-1}$ is the vector of predictors which include socio-demographic and socio-economic characteristics.¹⁴ We model the log of the integrated baseline hazard ($\theta(t)$) as a step function, by specifying dummy variables to represent each period individuals are at risk by using dummy variables for each year of age. This corresponds to a semi-parametric specification of the discrete-time duration model.

In our analysis, we condition on a number of socio-demographic characteristics, $X_{i,t-1}$, such as family and household characteristics, marital status, age, number of hours worked and wages that can be viewed as proxy variables of labour market attachment. As we cannot assume that we observe the natural starting point of the duration episode, we also condition on the number of years the individual has been working in the sector. In addition, we include initial and past health information (proxied by hospital care use). We assume

¹⁴ Labour market status in our data set is the status on January 1st of each wave. Therefore, transition into retirement or disability occurs between wave $t-1$ and t . So, we condition on the health shocks occurred between $t-2$ and $t-1$ and financial incentives at $t-2$.

that this set of explanatory variables contains the relevant information to explain the duration distribution.

3.5 Data

Our main data source is administrative data from the second largest pension fund in the Netherlands, the health care sector pension fund PFZW¹⁵ (formerly PGGM). PFZW provides pension arrangements to more than 2 million employees in the health and social work sector. The occupations in PFZW vary from nurses to medical doctors, and include all kinds of supporting activities like administration. Our data covers the period 1999-2003. We have access to individual information on gender, date of birth, working hours, wages, tenure, and pension and early retirement entitlements.

The PFZW data have some unique features. First, most individuals are women due to nursing being the most common type of job in this sector. Second, around 80% of females and 30% of males work part-time. In our analysis, we estimate empirical models separately for men and women as we expect the effects of health shocks and financial incentives to differ between men and women for transition into retirement and disability (Van Houtven and Coe, 2010; Colie, 2004a; Colie, 2004b; Belloni and Alessi, 2009). We select individuals who are active (in employment) and aged 55-63 in the first wave and follow them over a period of four years when they are at risk of becoming early-retired or receiving disability benefits.

In the PFZW data we observe the actual pension rights, the salary and seniority in the pension fund used to compute the pension benefits. This removes most of the measurement error encountered in surveys data which could lead to downward biased estimates for the pension incentive effects as shown by Belloni and Alessi (2009).

¹⁵ Pensioen Fonds Zorg en Welzijn. For details, see www.pfzw.nl/about_us/.

Figure 3.1: Median Pension Wealth by age for females and males

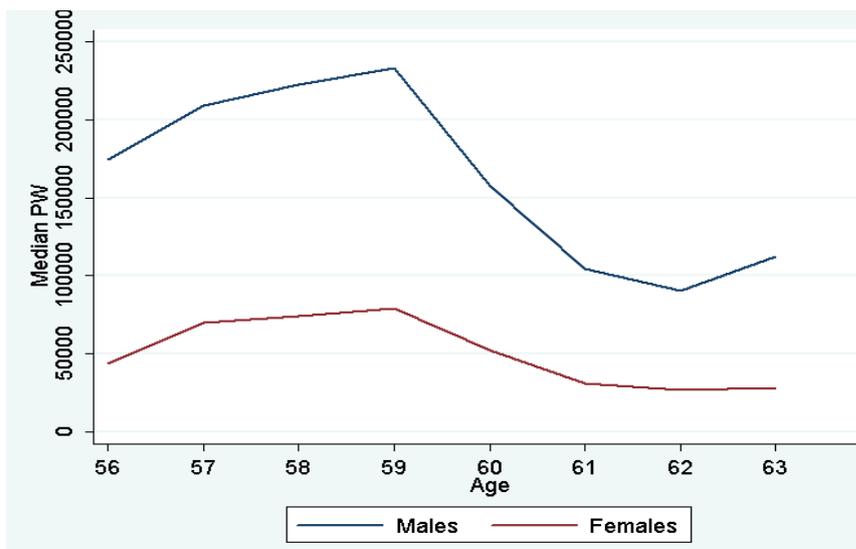
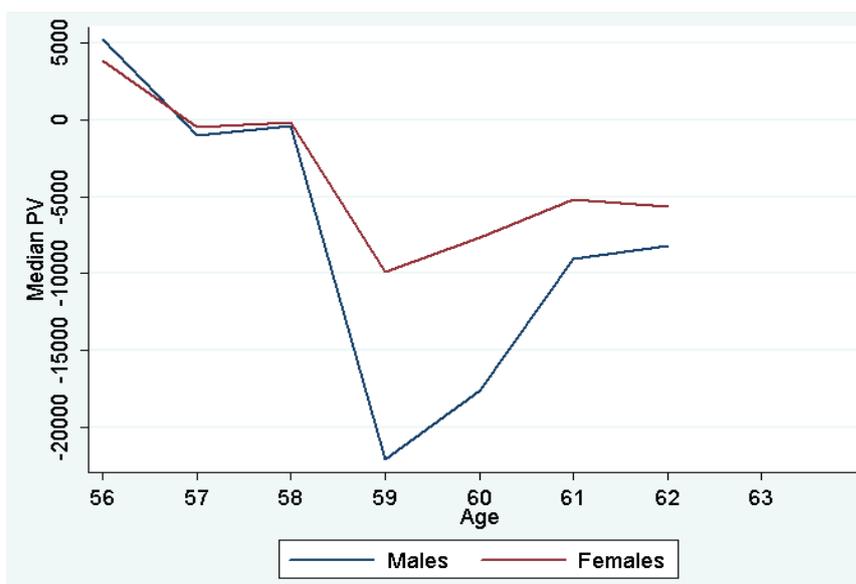


Figure 3.2: Median Peak Value by age for females and males



Figures 3.1 and 3.2 present median pension wealth and peak value for each possible early retirement age and illustrate that retirement incentives differ between males and females, most of whom work part-time. Median PW starts off around 50,000 Euros for females and 175,000 Euros for males. Until age 59, pension wealth increases because both the benefit base and replacement rate are rising. Pension wealth reaches its maximum value at age 59 - one year before the age which the worker is allowed to retire at age 60. Median PW decreases between age 59 to 61 for females, reflecting that additional years of work do not add to the expected pension amount. For males, median PW starts to rise again after age

62, while for females it remains constant after 61.¹⁶ A negative accrual or peak value can be interpreted as a tax on further labour force participation. The median peak value turns negative at age 58 and stays negative as the scheme offers the opportunity of part-time retirement at age 58 or 59.¹⁷

PFZW pension data for 1999-2003 were linked to three other administrative data sources by Statistics Netherlands: (1) the municipality register (GBA) which provides demographic information on birth date, marital status, household size, local migration and mortality; (2) the administrative employment register (SSB) with individual information of all employees in the Netherlands, including working hours, wages, and employer information¹⁸; and (3) the hospital discharge register (LMR) which includes information on diagnosis, length of stay of all hospital admissions.

Labour market status is missing in the PFZW data for some individuals in some periods. Attrition occurs: (i) when a participant has submitted an application for retirement.; (ii) if the address is unknown; (iii) when the insured becomes a so-called “sleeper” (i.e. no longer employed in the health care sector); or (iv) if the authority which administers their pension entitlements at PFZW has arrears of payment¹⁹. Attrition is problematic as individuals may still be employed, or may be retired. Fortunately, from the national employment register (SSB) we can recover whether the individual is employed and use this information to reduce the number of missing observations.

Table 3.1 shows the labor market transitions of females and males by wave for the stock sample of individuals aged 55 to 63. At a given period, individuals can be in one of four states: employed, early retired, disabled or censored. An observation is censored if the labour status is missing; the individual becomes older than 63 or dies, or transit to other labour market status. Most of the attrition is due to individuals becoming older than 63. The initial stock sample consists of 30,537 women and 9,598 men. The share of OBU early

¹⁶ Whether or not pension wealth increases depends on whether the rise in pension income from delaying retirement is sufficient to outweigh the fact that the pension would be received for one fewer year.

¹⁷ We observe the decrease in the peak value before age 60, since the maximum of the difference in pension wealth between now and the normal retirement age is decreasing. Closer to the normal retirement age, individuals' current wealth is getting closer and closer to the wealth at the normal retirement, so the maximum of the difference is decreasing.

¹⁸ See Euwals, et al (2010b) for a more detailed description of SSB and GBA

¹⁹ Euwals et al (2010) provide a detailed description of attrition in PFZW dataset. We follow them to reduce the number of missing observations.

retired (disabled) females increases from 4.35% (0.80%) in 2000 to 21.63% (2.46%) in 2003. For males, the number of OBU early retired (disabled) increases from 5.47% (0.46%) in 2000 to 21.02% (0.99%) in 2003. At the end of our observational period, 33.80% of females and 37.37% of males are still in employment. In the empirical analysis, we look at the first transition from employment, into OBU early retirement and disability states. We observe very few transitions back to work and so we take retirement and disability as absorbing states and ignore any subsequent transitions.²⁰

Table 3.1: Labor market transitions by wave for the stock samples

	1999	2000	2001	2002	2003
Females					
Employed	30,537	28,183 (92.29%)	22,619 (74.07%)	15,718 (51.47%)	10,322 (33.80%)
Early-retired	-	1,327 (4.35%)	2,953 (9.67%)	4,351 (14.25%)	6,604 (21.63%)
Disabled	-	243 (0.80%)	487 (1.59%)	658 (2.15%)	750 (2.46%)
Other labour status	-	101 (0.33%)	83 (0.27%)	116 (0.38%)	79 (0.26%)
Attrition	-	683 (2.24%)	4,395 (14.39%)	9,694 (31.75%)	12,782 (41.86%)
Total	30,537	30,537	30,537	30,537	30,537
Males					
Employed	9,598	8,823 (91.93%)	7,425 (77.36%)	5,144 (53.59%)	3,587 (37.37%)
Early-retired	-	525 (5.47%)	977 (10.18%)	1,337 (13.93%)	2,017 (21.01%)
Disabled	-	44 (0.46%)	68 (0.71%)	84 (0.88%)	95 (0.99%)
Other labour status	-	16 (0.17%)	29 (0.30%)	38 (0.40%)	20 (0.21%)
Attrition	-	190 (1.98%)	1,099 (11.45%)	2,995 (31.20%)	3,879 (40.41%)
Total	9,598	9,598	9,598	9,598	9,598

The hospital discharge register (LMR) contains information on both inpatient and day care patients of all general and university hospitals and most of the specialised hospitals in the Netherlands from 1998 to 2005. We observe (i) whether an individual entered the hospital, (ii) whether it was an unplanned admission, (iii) the admission and discharge date, and (iv) the main diagnosis. We construct measures of initial health status and new health events (proxying health shocks) using the LMR data.

²⁰ For instance, 0.03% of the females in our sample transit back from ER to employment and 0.008% from DI to employment.

We obtain indicators of the occurrence of new health problems from diagnostic information (ICD-9-CM codes). We select those codes that are likely to correspond to a new unexpected health problem if the individual did not go to the hospital in the previous year.²¹ This excludes diagnoses that can be first treated in outpatient care and those related to chronic conditions (for example, all the mental health problems and congenital diseases are excluded). Our measure of health shock takes the value 1 if the individual went into hospital to be treated for any of these diseases, and 0 otherwise. This selection of admissions makes it more likely that they are exogenous to labor outcomes. We test the robustness of the results using an indicator of whether the individual has an unplanned hospitalization in urgent need of treatment as an alternative measure. We control for the initial health status of the worker (i.e. at the start of the observation period) by using the information on number of hospital admissions and maximum number of hospital night stays in the first wave and the year before the first wave (1999 and 1998).

Table 3.2 shows descriptive statistics for men and women broken down by labor status in the next period. The health care sector employs many part-time working women, as can be seen from the average part-time factor for the females being around 60.²² The male average part time factor is much closer to 100, which means that most work full-time. Many women have low tenure in the sector, on average 9 to 11 years whereas males have around 20 years. The gross yearly wage ranges between 24 to 27 thousand Euros for females whereas for males it is between 37 and 42 thousand Euros. Other socio-demographic covariates used in the analysis include: marital status (married, single, widowed and divorced), household size, number of children, and Dutch nationality.

As expected, initial health (proxied by the number of hospitalizations and number of overnight stays) in 1998 and 1999 is highest for those who go into disability. Incidence of

²¹ We identify with the help of a medical doctor all diagnoses that could indicate new unexpected health events in the following groups: i) infectious and parasitic diseases (some codes within 001-139); ii) neoplasms (some codes within 140-239); iii) endocrine, nutritional and metabolic diseases and immunity disorders (some codes within 204-279); iv) diseases of the nervous system and sense organs (some codes within 320-389); v) diseases of the circulatory system (some codes within 390-459); vi) diseases of the respiratory system (some codes within 460-519); vii) disease of the digestive system (some codes within 520-579); viii) diseases of the genitourinary system (some codes within 580-629); ix) diseases of the skin and subcutaneous tissue (some codes within 680-709); x) diseases of the musculoskeletal system and connective tissue (some codes within 710-739); xi) injury and poisoning (some codes within 800-999).

²² A part-time factor equals 100 when the worker works 40 hours/week.

any new health event in the previous wave is 5.8% for disabled females and 8.9% for disabled males in our sample which is much higher than the incidences for the early retired: 2.5% for females, 3.3% for males. We also show the breakdown of acute admissions by (ICD9-CM) diagnosis. The diseases with highest incidence are cancer (especially among males), circulatory diseases, injuries and diseases of the genitourinary system.

Table 3.2: Descriptive statistics by labour market status in the next wave.

	Females			Males		
	Employed	Retired	Disability	Employed	Retired	Disability
Financial Incentives						
Pension Wealth	81,902.60	111,998.10	77,824.80	238,879.6	311,498.9	218,593.7
Peak value	-1,668.10	-10,649.00	-1,453.30	-2,060.7	-22,345.4	-687.5
Implicit tax	0.048	0.306	0.051	0.014	0.446	0.082
Initial health						
Number hospitalizations 98	0.095	0.097	0.225	0.098	0.107	0.267
Max number of nights 98	0.274	0.270	1.167	0.27	0.28	0.733
Number hospitalizations 99	0.106	0.105	0.306	0.107	0.113	0.253
Max number of nights 99	0.247	0.270	1.170	0.282	0.313	0.952
New health events/shocks						
Health shock (onset of any)	0.028	0.025	0.058	0.029	0.033	0.089
Infectious	0.000	0.001	0.000	0.001	0.001	0
Cancer	0.010	0.009	0.018	0.006	0.006	0.048
Nervous system	0.004	0.003	0.002	0.002	0.003	0.000
Circulatory	0.003	0.002	0.012	0.009	0.009	0.007
Stroke	0.001	0.001	0.005	0.003	0.002	0.007
Digestive system	0.001	0.001	0.005	0.001	0.003	0.000
Genitourinary system	0.003	0.002	0.004	0.000	0.001	0.014
Musculoskeletal	0.001	0.001	0.002	0.001	0.001	0.000
Injury	0.005	0.005	0.010	0.004	0.006	0.014
Other	0.002	0.002	0.007	0.002	0.002	0.007
Unplanned admission	0.028	0.025	0.068	0.028	0.025	0.068
Job characteristics						
Pensionable Wage	25,131.80	27,165.50	24,568.50	40,005.8	42,754.9	37,542.2

Table 3.2: Continued

Part-time factor	59	62.7	58.4	89.65	92.46	91.60
Tenure	9.34	11.67	10.1	20.10	25.10	20.40
Socio-demographic						
Age	57.6	59.13	57.6	57.70	58.97	57.60
Married	0.68	0.68	0.64	0.81	0.84	0.81
Single	0.09	0.1	0.11	0.07	0.06	0.10
Widow	0.07	0.07	0.05	0.02	0.01	0.01
Divorced	0.16	0.15	0.2	0.11	0.10	0.08
Number people in household	2.01	1.93	2	2.40	2.30	2.36
Number of kids	0.33	0.23	0.34	0.57	0.43	0.53
Dutch nationality	0.88	0.88	0.89	0.87	0.89	0.81
# of observations	78,094	7,683	1,030	25,297	2,456	146

3.6 Results

3.6.1 Effects of financial incentives and new health events

The results for the discrete-time hazard models for ER and DI transitions are presented in Table 3.3 for females and males. For each sample and model, results are presented as hazard ratios (exponentiated coefficients), which measure the proportional effect on the underlying (instantaneous) hazard of a one unit change in a given variable (the ratio by which the retirement probability changes for a unit change in a given variable).

The first two columns for each sample report results of the model of transition into ER using either peak value or implicit tax rate. The last two columns for each sample present the hazard ratios of becoming disabled for the same stock sample of employed females and males. A hazard ratio of one indicates that a variable has no effect on transition into ER (DI). A hazard ratio of more (less) than one indicates that a variable is associated with

Table 3.3: Effects in hazard ratios of health, financial incentives and job characteristics for early retirement and disability transitions

	Females				Males			
	Early Retirement		Disability		Early Retirement		Disability	
	Peak Value Model	Implicit Tax Model	Peak Value Model	Implicit Tax Model	Peak Value Model	Implicit Tax Model	Peak Value Model	Implicit Tax Model
Financial incentives								
PW/1000	1.005*** (0.0015)	1.006*** (0.0013)	0.994** (0.0029)	0.996* (0.0021)	1.001** (0.00035)	1.001*** (0.0003)	0.997* (0.0014)	0.997* (0.0013)
Peak value/1000	0.973*** (0.0029)	-	0.985* (0.0087)	-	0.992*** (0.0014)	-	0.998 (0.0062)	-
Implicit Tax	-	2.229*** (0.177)	-	1.370* (0.231)	-	1.824*** (0.201)	-	1.062 (0.277)
Health Shock and Initial Health								
Any new health event	1.05 (0.078)	1.0388 (0.077)	3.026** (0.535)	3.004*** (0.5312)	1.223* (0.13)	1.236* (0.134)	7.489*** (2.507)	7.482*** (2.511)
Max number of nights 98	1.003 (0.068)	1.0013 (0.0086)	1.058** (0.022)	1.058*** (0.024)	1.018** (0.0085)	1.018** (0.0092)	1.014 (0.0336)	1.014 (0.033)
Number hospitalization	1.012 (0.027)	1.0087 (0.0283)	0.953 (0.0787)	0.951 (0.079)	1.068 (0.0508)	1.057 (0.0516)	1.724*** (0.331)	1.724*** (0.331)
Max number of nights 99	0.987 (0.0078)	0.986 (0.0079)	1.028** (0.0099)	1.027*** (0.0099)	1.003 (0.00761)	1.0039 (0.0077)	1.051*** (0.015)	1.050*** (0.0155)
Number hospitalization	1.021 (0.0361)	1.027 (0.036)	1.226*** (0.07)	1.226*** (0.07)	1.051 (0.0435)	1.043 (0.044)	0.808 (0.143)	0.807 (0.142)
Job characteristics								
Wage/1000	0.948*** (0.0104)	0.948*** (0.0097)	1.012 (0.0219)	0.997 (0.0174)	0.991*** (0.0029)	0.988*** (0.0026)	1.008 (0.106)	1.006 (0.0065)
Part-time factor	1.009*** (0.0023)	1.007*** (0.0022)	0.993 (0.0049)	0.995 (0.0046)	1.002 (0.0015)	1.002 (0.0015)	0.998 (0.0069)	0.998 (0.0066)
Tenure	1.011* (0.0069)	1.008 (0.0066)	1.053** (0.0169)	1.046*** (0.0153)	1.041*** (0.0033)	1.037*** (0.0033)	1.016 (0.0183)	1.014 (0.0185)
# of observations	30,657	30,657	23,544	23,544	11,753	11,753	9,278	9,278

Notes: 1. Statistical significance at 1% level = ***, 5% = ** and 10% = *. 2. Robust standard errors of coefficients in parentheses.
3. Additional controls: age dummies, wave dummies, marital status (married, single, widowed, divorced), number of people in household, number of kids, Dutch nationality.

higher (lower) transition into ER/DI. All models include “onset of any health event” as a measure of health shock.²³

Effects of financial incentives

We consider alternative models with either pension wealth (PW) and peak value (PV) or PW and implicit tax (IT). We find significant effects for pension wealth (*PW*), peak value (*PV*) and implicit tax (*IT*) for ER transitions both for females and males. As expected, *PW* is associated with an increase in the hazard of ER. This can be interpreted as an income effect, which is consistent with the idea that leisure is a normal good. For females, an increase of 1000 Euros in *PW* increases the hazard of ER by 0.5%, and for males by 0.1%.

The *PV*, on the other hand, acts as a price as it reflects the relative cost, in terms of pension wealth, of retiring early. An increase in the *PV* significantly decreases the hazard of ER, both for females and males. The *IT* is also highly significant: a high *IT* on work leads to a higher hazard of retiring early the next wave. Females are found to be more sensitive to financial incentives compared to males. An increase of 1000 Euros in *PV* decreases the hazard of ER by 3.3%, while it decreases the hazard of ER by 0.8% for males. Similarly, an increase of the *IT* to work by 10 percentage points increases the hazard of ER by 8% for females and 6% for males.

Our estimated effects of *PW*, *PV* and *IT* conform to expectations derived from the insights of the Option Value model. Greater expected pension wealth increases the hazard of ER, while a financial reward to delaying retirement, in the form of a higher benefit level, increases the chances of continuing work. Generally, all financial incentive effects are estimated to be larger for women than for men even though the eligibility conditions for ER are the same for males and females.

Disability Insurance has traditionally been one of the pathways into (early) retirement in the Netherlands (de Vos et al, 2011). An individual decides to stop or continue working based on the comparison of the alternative exit routes and therefore, the availability and generosity of early retirement benefits are expected to play a role in the decision to transit

²³ The results using either “an unplanned hospitalization in urgent need of treatment” or onset of each new health condition separately are qualitatively similar. Results are available upon request.

into DI.²⁴ The results (columns 4 and 5 for females and 8 and 9 for males) show that the higher the pension wealth, the lower the DI hazard. In other words, the more beneficial ER is for older workers, the less likely they are to use other exit routes such as DI. This provides further evidence that DI was used at the beginning of the last decade as a substitute pathway into (early) retirement.

On the other hand, we find that the lower the PV and the higher the IT, the higher the DI hazard, although the effects are non-significant for males. These effects go in the same direction as in the ER hazard, but their magnitude is smaller. These two measures capture the price effect of the decision to continue or stop working. The results suggest that if the price paid to stop working today to go into early retirement is too high, it is also high to go into DI, and the individual would therefore be more likely to choose to continue working.

Effects of new health events and initial health status

For both male and female workers, we find that new health events have a larger effect on the DI hazard than on the ER hazard.²⁵ The hazard of disability is around 3 times larger for females who experience an adverse health event. New health events do not (significantly) raise the chances of going into ER for women, but they do for men. For males, new health events significantly increase the hazard of early-retirement: the hazard of ER transitions is 1.2 times larger for males who experience a health shock, compared to those who do not. This gender difference may be explained by higher pension entitlements among males. If the older worker qualifies for the OBU scheme, he/she is expected to withdraw from the labor market after a health event through the early retirement scheme as the replacement rates in OBU scheme (at least 71%) are higher than DI replacement rates (maximum 70%), and in both cases he/she continues to build-up pension rights.

Similar results are found regarding the effects of initial health. On the whole, a history of previous health events such as number of night stays and number of hospitalizations is also

²⁴ It is important to emphasize that we do not observe any financial incentives for the DI route. In particular, we do not observe social security wealth, only pension wealth.

²⁵ The hazard ratios for the effects of new health events are higher for males than females, but the large hazard ratio for males (around 7.48) could be due to the low number of males that transit into DI (178) and the low incidence of health shocks.

associated with an increase in the hazard of DI, but not ER, except for the number of hospital nights for males in 1998.

Effects of job characteristics and other socio-demographic variables

The results show that job characteristics also play an important role in explaining transitions out of employment. We find that higher wages are associated with a decrease in the hazard of ER and this effect is larger for females. The larger the number of hours worked (part-time factor), the larger the hazard of ER for females. A 10 points increase in the part-time factor increases the hazard of ER for females by 9%. The lack of a significant effect for males may be due to the low variation in the number of hours worked for males as they are mostly working close to full-time. Higher tenure in the sector significantly increases the hazard of retiring early for men, but not for women.

Wages and number of hours worked turn out not to have any significant impact on entry into disability entry, but tenure in the sector does significantly raise the hazard of becoming disabled for females. We find that women with larger tenure are more likely to enter ER and DI. A plausible explanation is that this is capturing the effect of the length of exposure to work-related stress given that a large proportion of females in our sample are nurses.

The effects of other covariates (not shown here) are stable across the different models but sometimes differ between females and males. For example, the hazard of becoming early retired or disabled increases with age for both. Marital status only appears to have an effect on females and the hazard of early retirement is lower for divorced and widowed compared to married females. Household size increases the hazard ratio of early retirement for female workers but not for males.

3.6.2 Interaction between financial incentives and new health events: do the effects of financial incentives on transitions into ER and DI differ between workers in good and poor health?

Erdogan-Ciftci et al (2011) argue that, theoretically, health shocks may have an impact on the effects of financial incentives by exacerbating the positive effect of pension wealth on the chances of retirement and dampening the negative effect of peak value on the chances of retirement. However, they also show that the direction of the effects of health shocks on the retirement impact of peak value and pension wealth cannot be theoretically signed *a priori*. In particular, health shocks may increase or decrease the effectiveness of financial incentives since after a health shock there is an increase in marginal disutility of labour, decrease in survival probability and reduction in discount factor.

Table 3.4: Interaction between health shocks and financial incentives for early retirement and disability transitions

	Females		Males	
	Early Retirement	Disability	Early Retirement	Disability
Covariates				
PW	1.005*** (0.0015)	0.994** (0.0030)	1.001** (0.0003)	0.997* (0.0017)
Peak value	0.973*** (0.0029)	0.990 (0.0087)	0.992*** (0.0014)	0.995 (0.0070)
Any new health event	0.891 (0.9884)	2.560*** (0.6782)	1.079 (0.1776)	5.196*** (2.533)
PW * Any new health event	1.002* (0.0011)	1.002 (0.0025)	1.000 (0.0005)	1.001 (0.0016)
Peak Value* Any new health event	1.007 (0.0120)	0.953** (0.0224)	0.992 (0.0063)	1.011 (0.0095)
# of observations	30,657	23,544	11,753	9,278

Notes : 1.Statistical significance at 1% level = ***, 5% = ** and 10% = *. 2.Standard errors in parenthesis. 3.Additional controls: age dummies, wave dummies, marital status (married, single, widowed, divorced), number of people in household, number of kids, Dutch nationality

We find little empirical evidence that a health shock modifies the effect of the financial incentives (Table 3.4). We only consider the interactions in the PV model, as IT model gives us similar results. Only for female ER transitions, we find that the PW effect

becomes stronger in case of a health event – the hazard ratio for the PW of those who experience new health event is increased to 1.007 compared to 1.005 for those who do not experience it. In other words, all else equal, every additional Euro in the pension benefit leads to a higher hazard of early retirement if the female employee has a health shock compared to those without a health event. This result is in line with Erdogan-Ciftci et al (2011) and could be due to the increase in the disutility of labour after a health shock boosting the string of benefits, which makes pension wealth more inciting to retirement. On the other hand, we do not find a differential effect of the PV on ER transitions for those who experience a health event.

The effect of the PW on the DI hazard is similar for healthy and unhealthy individuals. However, the negative PV effect is concentrated among those females who experience a health shock. The hazard of DI decreases by 5.6% if PV increases by 1000 Euro for females with a negative health event. This result suggests that the pension incentives to continue working or transit into DI are only relevant when the individual is likely to get access into DI, as it is after the occurrence of a new health event.

3.6.3 Do the effects of new health events on transitions into ER and DI differ by pension wealth?

As noted by Banks (2006), there are striking differences in labour force exit routes in the UK. Older poor individuals are more likely to enter DI while the older rich enter retirement. This suggests that individuals who can afford to retire withdraw from the labour market using early retirement, while those whose pension wealth is not sufficiently high need to find other exit routes. We build on this “affordability hypothesis” and investigate if the effect of a health shock differs by the amount of initial pension wealth. We split the sample between those with pension wealth in the first wave below and above the median. The results are shown in Table 3.5.

We find that new health events increase the hazard of ER for both men and women with higher pension wealth, but not for those with pension wealth lower than the median. An adverse health event increases the disutility of work for individuals with both high and low

pension wealth. However, only those with expected pension benefits high enough to compensate for the lack of earnings decide to go into early retirement. The effect of a health shock increases the DI hazard for both the high and low pension wealth group. Surprisingly, the effect is not larger – if anything it is smaller – for individuals in the lowest pension wealth group. This suggests that older workers in the Netherlands first decide whether to stop or continue working by comparing the expected stream of pension benefits with the loss of earnings and the disutility of work, and then choose the route that is most attractive given the financial incentives.

Table 3.5: Effects of new health events on early retirement and disability transitions by pension wealth and wage groups

	Females		Males	
	Early Retirement	Disability	Early Retirement	Disability
Effects of new health events				
Low wealth group	0.929 (0.106)	2.917*** (0.670)	1.102 (0.184)	5.164*** (0.0074)
	# obs: 16,759	# obs: 13,733	# obs:6,048	# obs:5,065
High wealth group	1.188* (0.119)	3.194*** (0.878)	1.324* (0.190)	14.699*** (8.662)
	#obs: 13,898	# obs: 9,811	# obs:5,705	# obs:4,213
Low Wage group	1.0852 (0.121)	3.168*** (0.827)	1.217 (0.187)	3.369** (1.891)
	#obs: 13,589	#obs:10,482	# obs: 5,878	# obs: 4,662
High Wage group	1.0287 (0.102)	2.849*** (0.661)	1.185 (0.178)	20.199*** (8.743)
	#obs: 17,068	#obs: 13,062	# obs: 5,815	# obs: 4,616

Notes: 1.Thresholds for high (low) wealth / wage groups are defined as higher (lower) than the median wealth /wage in the first wave. 2. Statistical significance at 1% level = ***, 5% = ** and 10% = *. 3.Standard errors in parenthesis. 4. 3.Additional controls: age dummies, wave dummies, marital status (married, single, widowed, divorced), number of people in household, number of kids, Dutch nationality

One could argue that the evidence presented above is not testing the affordability hypothesis but differential effects across skill groups. In order to rule out this alternative explanation, we did a similar analysis by hourly wage groups. Our contention here is that older workers with low hourly wages are those in the lowest skill groups, while those with highest hourly wages are in the most skilled jobs. We do not find any differential effects of

new health events by wages for ER transitions for either females or males. For the DI route, new health events significantly increase the DI hazard for both wage groups. The results for females suggest that older workers with low skills are slightly more likely to transit into DI after a health shock compared to high skilled workers. This reinforces the conclusion that the differences by pension wealth were not driven by differences in skills.

An alternative explanation could be that the effects are different because individuals in different wage and wealth groups experience different health events. We look at the distribution of diagnoses across these groups and we do not find any relevant difference in the type of diseases. Older workers with low pension wealth and with low wages are slightly more likely to experience an adverse health event and are in worse initial health. However, the distribution of health events among those who experience a health shock is similar among all wealth and wage groups.

3.7 Conclusion and discussion

This paper analyzes the role of adverse new health events and financial incentives for ER and DI transitions and explores their interaction in testing two main hypotheses: (i) a *subordination hypothesis*, i.e. health shocks may dampen the financial incentives created by pension systems (Erdogan-Ciftci et al, 2011); (ii) an *affordability hypothesis*, i.e. older workers with sufficiently high pension wealth are more likely to be in a position to afford to (early) retire after a sudden negative health event than workers with lower pension wealth. We make use of a unique dataset that combines administrative data with precise benefit information from one of the largest pension funds linked to hospital admission records in the Netherlands. We use unanticipated health events to aid identification of causal effect of health on ER and DI transitions. Exact knowledge of pension wealth and marginal incentives reduces the possibility of measurement error found in survey data based analyses (Gruber and Wise, 2004).

We find that the effect of new adverse health events is a dominant factor for DI transitions for both females and males, while incentives to retire early are strongest for ER. These results are in line with Kerkhofs et al (1999) and Erdogan-Ciftci et al (2011). However,

this does not mean that health does not influence retirement or that retirement incentives do not affect DI exits: we find a health shock also to increase the likelihood of early retirement for males, and early retirement financial incentives to affect transitions into DI.

In line with estimates in the previous literature (Gruber and Wise, 2004), we find that pension wealth and implicit tax on work increase the hazard of early retirement and a higher peak value reduces the hazard of retirement. Females are found to be more sensitive to financial incentives than males, which is consistent with previous findings (Belloni and Alessi, 2009; Hanel and Riphahn, 2006). Trevisan and Euwals (2011) have found low wage earners to be more sensitive to financial incentives. The females in our sample mostly work part-time and have lower tenure, and hence lower earnings. Females may then be more sensitive to financial incentives as they have on average lower wages. In general, the mechanisms underlying the differential sensitivity to financial incentives between males and females require further investigation as this could imply that different policies are needed to prevent older female and male workers to exit the labor market at younger ages.

Early retirement financial incentives also turn out to affect transitions into DI. The sign of the price effect, measured by the peak value and the implicit tax on work, is the same as for transitions into ER. Therefore, the higher the price paid today in terms of foregone future benefits and earnings, the lower the probability that the older worker stops working and transits either into ER or DI. Therefore, measures that provide incentives to delay retirement would also discourage workers to exit the labor market using other routes.

On the contrary, we find that pension wealth is significantly associated with a *decrease* in the hazard of DI. This is in line with evidence showing that ER and DI are potential substitute pathways into retirement in the Netherlands (Kerkhofs et al, 1999; de Vos et al, 2011). Several reforms have been introduced in the past decade in the Netherlands to tighten the DI eligibility criteria and reduce the financial incentives to choose an early retirement scheme. Euwals et al (2011) provide suggestive evidence implying that employment rates of older individuals have increased and that DI is no longer used as a pathway into retirement. A thorough evaluation of the effectiveness of these reforms remains a pending research question.

We find little empirical support for the *subordination hypothesis*. The pension wealth effect on retirement transitions becomes stronger in case of a health event only for females. On the other hand, we do not find a differential effect of the peak value on ER transitions for those who experience a health event. Erdogan-Ciftci et al (2011) and Banks et al (2007) found the (negative) impact of peak value on retirement to be significant only for those without health problems. The difference in the results can be due to cross-country institutional differences and/or the measure of health used. They both use self-reported health information. Our health measure is free of reporting bias, but it excludes health information related to illnesses that are normally treated as outpatient, like e.g. mental health problems.

While the occurrence of a new health event increases the probability that an older worker exits the labour market, the magnitude of the effect and the pathway chosen may depend on the resources the individual can enjoy if not working. Our results show that a new health event increases the probability of transiting into ER *only* among workers with high pension wealth, and this difference is not compensated by larger effects on the DI hazard among workers with low pension wealth. We rule out that these results are driven by differences in skill levels and therefore the ability to cope with adverse health events across groups, or by differences in the type of admission diagnosis. These results, therefore, *do* provide support in favour of the *affordability hypothesis*.

Unfortunately, data on DI financial incentives was not available and we could not investigate the relative effect of the incentives of the different exit routes. Kerkhofs et al (1999) explored the role of replacement rates of alternative exit routes (ER, DI and unemployment) and found the effects of ER incentives and eligibility to be strongest. Confirmation of these results with administrative data remains a pending research question.

Finally, we have examined decisions taken at the external margin. A majority of health care workers work part-time and they may decide to gradually transit into retirement. The extent to which financial incentives, health shocks and their interaction impact on decisions at the internal margin deserves further investigation in future work.

PART II

Health Perceptions and Their Formation

Chapter 4

Do self-perceived health changes predict longevity?

Researchers can rely either on retrospectively reported or on prospectively measured health changes to identify and quantify recent changes in respondents' health status. The two methods typically do not provide the same answers. We compare the validity of prospective versus retrospective measures of health changes by investigating their predictive power for subsequent mortality. Data from a cohort study conducted in the Netherlands are used to compare the ability of changes in self assessed health (SAH) – either reported retrospectively or measured prospectively in three waves (1991, 1993 and 1995) - to predict survival until 2004. We examine the relationship between health changes and mortality with a proportional hazard models controlling for individual unobserved heterogeneity, with and without control for pre-existing chronic conditions and the onset of new chronic diseases. For a high proportion of reports (39.8 %), prospectively measured health changes in SAH do not concur with retrospectively reported health changes. Our results show that both measures of health changes are predictive of mortality in the model controlling for levels of SAH and socioeconomic characteristics only. Controlling for SAH, prior presence of chronic conditions, the onset of new conditions and unobserved characteristics, we find that prospectively reported health changes still predict longevity, whereas retrospective changes do not. These results suggest that the collection of longitudinal information on health changes has advantages over the - easier and cheaper – option of retrospective collection of the same information.

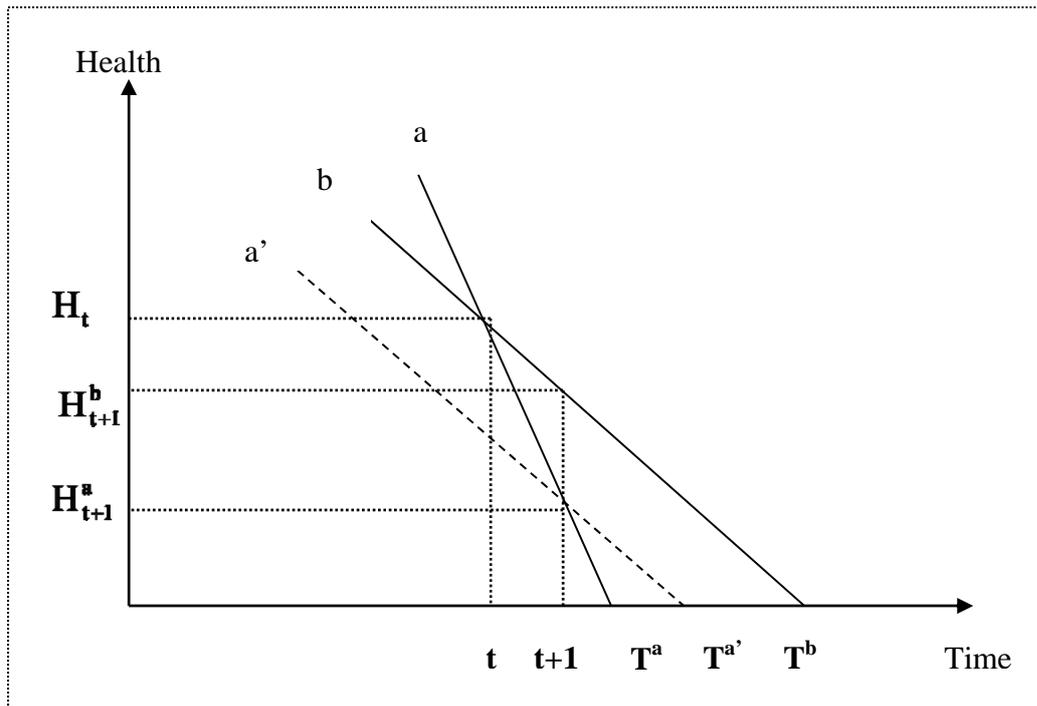
4.1 Introduction

It is well known that self-assessed health (SAH) at one point in time has substantial predictive power for behavior including medical care utilization (van Doorslaer et al., 2000, 2004), labor force participation (Bound, 1991), as well as for subsequent health outcomes, like survival (Idler & Benyamini, 1997; Van Doorslaer & Gerdtham, 2003; Mackenbach et al., 2002; Dowd and Zajacova, 2007; Huisman et al., 2007), even after controlling for other, more objective, health indicators. Much less is known about the predictive value of health *dynamics*, i.e. changes in SAH. In many instances, researchers are interested in such changes, especially the negative ones – often referred to as ‘health shocks’ – because these may be equally (or even more) important precursors of later outcomes as (than) health levels. They are also indicators of the degree of persistence of health status. The two questions that we seek to answer are: (a) do changes in health levels have predictive ability over and above the information contained in health level itself?; and, if so, (b) how can such changes best be elicited?

A priori, the answer to the first question ought to be affirmative, and this can easily be seen from the graph in Figure 1, which depicts health trajectories for two hypothetical individuals A and B. Clearly, the information about a difference in the level of health at time $t+1$ has predictive power for the likelihood of each person’s health falling below a critical level. If all else is equal, including the health level at t , then person A, with the lower health at $t+1$, is likely to reach the minimal critical health level sooner and exhibit shorter expected survival. In the particular case depicted, knowing that both persons started off at the same health in t will lead to very different predictions of future health paths than knowing that they were already in different health states at t and moved along parallel trajectories between t and $t+1$ (such as A’ and B). It seems therefore obvious that information on recent health changes does add to the information on health levels.

Regarding the second question, there are basically two main approaches to eliciting health changes from self-reports. The first and easiest option is to simply ask *retrospective* questions about health changes: respondents then rate their health compared to a reference point in the past.

Figure 4.1: Health trajectories of hypothetical individuals a, a' and b.



This health transition question asks respondents to rate their general health compared with a previous period, with three response categories: “better”, “same”, and “worse”. It represents a simple and straightforward way of obtaining health change information from cross-sectional surveys when there is no opportunity to follow respondents over time. However, it only provides a proper alternative to the prospective, longitudinal collection of health change data, if the information obtained is similar, if not identical. The prospective health changes can simply be obtained by computing the changes in SAH between two consecutive waves in longitudinal data. However, it has been shown that retrospectively reported health changes between point 1 and 2, assessed at point 2, do not always concur with prospectively assessed changes between point 1 and 2 in time. Benitez Silva and Ni (2008) discuss several reasons for the possible incongruence. First of all, the incongruence may occur due to the reporting heterogeneity bias and cut-point shifts in SAH¹. In particular, cut-point shifts of SAH, for a given individual, *over time* may be one possible source of bias in the prospectively health change measure. This means that, for a given true but unobserved health state, individuals may report health differently depending on their health expectations at two different points in time. In addition, health changes identified by

¹ See Kerkhofs and Lindeboom (1995) and Lindeboom and van Doorslaer (2004) for the discussion of reporting heterogeneity and cut-point shifts and Bago d’Uva et al (2008a, 2008b) for attempts at correcting using vignettes.

retrospective health changes may not be large enough to cause a category jump in SAH in the next period and may not show up in prospectively measured changes in SAH.

On the other hand, some biases have been reported regarding retrospective health change elicitation. The direct health change question forces individuals to provide a comparison of their current health with a different point in time. This may cause reliability and recall problems, and individuals may use different reference points in time when recalling the previous health state (Norman et al. 2001). There is also some evidence that retrospective self-reports of health are biased towards the respondent's present health state (Norman et al, 1997; Knox and King 2009) indicating that respondents with good health currently are more likely to report that their health has recently improved, and respondents with poor health currently are more likely to report that it has worsened. Given possible biases with both of the health change measures, the empirical question then becomes: which of the two change measures appears to perform better in predicting future hard health outcomes like mortality?

In spite of an abundance of studies demonstrating high predictive ability of SAH for mortality, only a few have done this for health changes. Ferraro and Kelley-Moore (2001), for instance, found that SAH predicted mortality risk over 20 years follow-up only when treated as a time-dependent covariate, highlighting the importance of using dynamic models when multiple observations are available. Han et al. (2005) studied the impact of SAH as a time-dependent covariate in Cox regression model among older people and found change in SAH to be a stronger predictor of mortality than SAH at baseline. Strawbridge and Wallhagen (1999) also used SAH as a time-dependent covariate and found that change in SAH was a significant predictor of mortality among women. More recently, Lyyra et al., (2009) showed that the use of SAH as a time-dependent covariate in a Cox regression model enables advantage to be taken of all the information in a longitudinal study design. Using data from the German Socio-Economic Panel, Schwarze et al. (2000) found that mortality was not only affected by the level of SAH but also by changes compared to a previous year. On the other hand, the only study we could find which analyzed simultaneously the effect of SAH and retrospectively reported health declines, found only the latter to be significant (Deeg et al, 1989).

While all of these studies do examine the relationship between SAH changes and mortality, none of them has compared the predictive ability of different measures of health change. One such direct comparison of the predictive ability of self-reported retrospective versus prospective changes was done by Benitez-Silva and Ni (2008) using data from the US Health and Retirement Survey (HRS) but for subjective survival expectations as outcome measure, not actual mortality. Their results have favored the use of retrospectively reported health changes instead of prospectively computed changes in SAH but the measurement of survival expectations has been shown elsewhere to be noisy and subjective itself (Bassett & Lumsdaine, 2001). We also believe their results stem in part from the inappropriate control for initial health (cf measures section below).

In this study, we exploit the simultaneous availability of four waves (1991-93-95) of longitudinal health data from the GLOBE study (Mackenbach et al., 1994) and a mortality follow-up until 2004 to examine and compare the validity of alternative measures of health levels and changes for predicting mortality. These health measures include the level of SAH, computed changes in SAH, retrospective assessments of health changes, a set of self-reported chronic conditions and changes in self-reported chronic conditions. They enable us to answer the two main questions of this study. First, is there any value added of including retrospective/prospective health changes for mortality prediction, over and above health levels? Second, are prospectively and retrospectively reported health changes equally predictive of subsequent mortality?

4.2 Methods

4.2.1 Data

Our data were taken from the longitudinal GLOBE study that was conducted since 1991 in a region in the Southeast of the Netherlands. The study is based on a cohort of non-institutionalized Dutch nationals, aged 15-74 years, living in the city of Eindhoven and surroundings. The GLOBE study is widely used and has contributed to the understanding of the explanation of socio-economic inequalities in health in the Netherlands (see e.g. Van

Lenthe et al., 2004, and van de Mheen et al, 1999). Study results were a main source of information in the development of policy measures aimed at the reduction of socioeconomic inequalities in health. More information on the design and objectives of the GLOBE study can be found elsewhere (Mackenbach et al., 1994). A baseline random sample of approximately 27.000 individuals (stratified by age and postal code) was drawn from the population registries of the participating municipalities. These individuals received a postal questionnaire in the Spring of 1991, which had a response rate of 70.1% ($n = 18973$).

Two sub-samples drawn from the baseline sample were re-interviewed in the autumn of 1991 and in subsequent years. The first subsample, hereafter called the “healthy sample”, was randomly drawn from the baseline sample ($n = 2800$). These individuals were re-interviewed in 1993, 1995 and 1997. In the second subsample, which we call the “frail sample”, individuals who reported to have at least one of four chronic conditions (asthma, severe low back pain complaints, diabetes mellitus and heart disease) were overrepresented ($n = 2867$). The frail sample was re-interviewed annually in 1992-1995 and 1997. In our analysis, we only use data from years 1993 and 1995 in which both samples were interviewed (so, we exclude 1992 and 1994) and for which the main variables of interest (in particular retrospective changes) were available. This means that we further exclude data from 1997 as none of the samples was asked to report retrospective health changes in this year. For the same reason, we use the 1991 wave only to compute prospective changes in SAH between 1991 and 1993. In the analyses presented in this paper, we pool the healthy and the frail samples in order to increase the sample size. Dropping cases with missing values for at least one of the variables used in the analyses leaves us with a total of 6148 observations (3242, in 1993, and 2906, in 1995), of which 3238 belong to the healthy sample (1705, in 1993; 1533, in 1995) and 2910 to the frail sample (1537, in 1993; 1373, in 1995).

The GLOBE dataset was augmented through linkage to the national register of cause-of-death, until the end of 2004. During the follow-up period of 11 years, 8.6 % (282/6148) of the respondents in our sample died.

4.2.2 Measures

We use several measures of health levels and of health changes in our analysis. The main measure of health level is derived from the standard question: “How is your health in general?” with response categories very good, good, fair, sometimes good/sometimes poor and poor. Further measures of health levels are whether individuals suffer from a range of chronic physical and mental conditions, described in detail below.

The *retrospective health change* variable is the directly reported change in respondent’s health compared to a previous year (cf below). Response categories are “health remained the same”, “health has improved” and “health has worsened”. In 1995, respondents in the frail sample were asked to compare their health to that one year before, while for the healthy sample the reference period was two years. In 1993, the reference period was two years for both samples. Since we use the pooled sample in our analysis, we tested whether our results are sensitive to consideration of a different reference period for 1995 for the frail sample. This was done by including in the model interaction terms between an indicator of these observations and the indicators of reported changes. These interaction terms were jointly not significant (p -value=0.395), supporting our use of the pooled sample. We compute *prospective health changes* (better, same or worse) as the difference between currently and previously reported SAH. Better or worse computed changes reflect jumps of one or more health categories between waves. The reference periods considered for the computation of prospective health changes are consistent with those described above for reported health changes. A set of interaction terms between an indicator of observations from the frail sample in 1995 and indicators of computed changes was also jointly not significant (p -value=0.334).

Several recent papers have favored the use of prevalence (Adams et al.,2003) or the onset of new chronic conditions (Smith, 1999; Smith, 2007; Wu, 2003) as arguably more objective indicators of health and health changes than those derived from self-assessed health and self-reported (prospective or retrospective) health changes. The GLOBE data also provide information on the presence of self-reported chronic physical and mental conditions, and whether they were diagnosed during the 12 months prior to the survey. We are therefore able to assess whether the association between self-assessed health - and

retrospective/prospective health changes - and mortality can be explained by prevalence and onset of these conditions. We use information on fourteen conditions: high blood pressure, back pain / problems, diabetes, heart diseases, stroke, cancer, ankylosis, rheuma, lung diseases, stomach diseases, nervous diseases, intestine diseases, skin diseases. Additional emotional and mental health information is taken from Nottingham Health Profile questions (we consider an indicator of presence of at least 1 emotional or mental problem). We use both prevalence and onset of new chronic conditions between waves.

Since we are interested in the effects of health changes (i.e., self-reported retrospective/prospective and onset of chronic conditions) controlling for health levels, we need to define these in a consistent manner. Note that the effects of a health change can be obtained in two different ways, as the effects of: *i*) current health, H_2 , controlling for previous health, H_1 ; or *ii*) a health change, $H_2 - H_1$, controlling for previous health, H_1 (it can be easily shown that $a.H_2 + b.H_1 = a.(H_2 - H_1) + (a+b).H_1$, i.e., that the effect of $(H_2 - H_1)$ in *ii*) equals the effect of H_2 in *i*). Since retrospective changes are a direct measure of changes, $H_2 - H_1$, and for comparative purposes, we opted for option *ii*), rather than *i*), to evaluate effects of all health changes. We have therefore considered previous health levels (self-assessed health and prevalence of chronic conditions) observed at the reference point in time for health changes (that is, two years previously for both the sub-samples in 1993 and for the healthy sub-sample in 1995; and the previous year for the frail sample in 1995), rather than current levels. If, on the other hand, the model included $(H_2 - H_1)$ and H_2 , then the effect of the former could not be interpreted as that of a health change because $a.(H_2 - H_1) + (a+b).H_1 = -b.(H_2 - H_1) + (a+b).H_2$, ie, the latter effect of $(H_2 - H_1)$ does not equal that of the other two options and will, in fact, be of the opposite sign if effects of current and previous health levels are of the same sign in *i*), which is a reasonable expectation.

We also included two indices based on activities of daily living (ADL). The first is an ADL index and is simply a sum score of problems with the following activities: move at the same floor, get in/out of bed, eat and drink, getting (un)dressed, washing face/hands, washing completely. The second is an index of mobility problems, which adds up indicators of difficulties with each of the following activities: walking down/upstairs, moving outdoors, leaving/entering house, sitting down/getting up from chair.

We also control for demographic and socioeconomic characteristics, like gender, marital status (unmarried, married, divorced, widowed), education (at most primary, low vocational, middle education, high education), working status (employed, unemployed, disabled, retired, housework, student, living from investments) and income (low, middle, high). GLOBE respondents reported their total monthly household income in 13 categories ranging from ‘0-1000 guilders’ to ‘above > 5800 guilders’ (the guilder – 1 guilder = 0.45€ - was the currency in The Netherlands during the period under analysis). We consider values under 1900 guilders as low income (first four categories), 1900-3500 as middle income (5th to 9th categories) and more than 3500 as high income. All demographic and socioeconomic characteristics are measured both in 1993 and 1995, except for education (measured only at the baseline). Descriptive statistics for all variables used in the analyses are shown in Table 4.1.

Table 4.1: Descriptive statistics of health measures and socio-demographic variables

Variable	Mean	St. Dev
<i>Survival Variables</i>		
Life time (number of years lived by 2004)	59	15.3
Death (1 if has died by 2004, 0 alive)	0.086	0.282
Age in 1993	53	14.89
Age at death	67	8.44
<i>Independent Variables</i>		
Current SAH very good	0.170	0.375
Current SAH good	0.572	0.495
Current SAH fair	0.165	0.371
Current SAH som. good / som. poor	0.080	0.271
Current SAH poor	0.013	0.114
Previous SAH very good	0.171	0.377
Previous SAH good	0.577	0.494
Previous SAH fair	0.162	0.368
Previous SAH som. good / som. poor	0.076	0.265
Previous SAH poor	0.014	0.115
Self reported health change better	0.088	0.283
Self reported health change same	0.805	0.397
Self reported health change worse	0.108	0.310
Computed health change better	0.192	0.394
Computed health change same	0.648	0.478
Computed health change worse	0.195	0.396
ADL index (0-6)	0.290	0.885
Mobility problems index (0-4)	0.430	0.931
Male	0.537	0.499
Primary or < primary education	0.170	0.376
Low vocational education	0.219	0.413
Middle education	0.405	0.491
High Education	0.200	0.400
Employed	0.402	0.490
Unemployed	0.038	0.190
Disabled	0.069	0.254
Retired	0.229	0.420
Housework	0.224	0.417

Table 4.1: continued

Living from investments	0.007	0.086
Student	0.029	0.168
Married	0.740	0.439
Divorced	0.055	0.228
Widowed	0.056	0.230
Single (Never Married)	0.149	0.356
High Income	0.384	0.486
Middle Income	0.445	0.497
Low Income	0.170	0.376
N	6148	

Notes: Descriptives for high blood pressure, back pain / problems, diabetes, heart diseases, stroke, cancer, ankylosis, rheuma, lung diseases, stomach diseases, nervous diseases, intestine diseases, skin diseases, NHP Mental Health Problems are not reported.

4.3 Statistical Analysis

To estimate the relationship between prospective and retrospective health changes and age at death (in full years), we use the Gompertz proportional hazards model for three reasons: (i) the restriction of the hazard to be constant over time in an exponential model is undesirable in our case as we expect the hazard rates to be varying over time, (ii) the Gompertz model allows to take into account the age effect through the shape of the baseline function², (iii) the Gompertz model is the most commonly used in the biological and medical literature modeling mortality (see Balia & Jones (2007) for a comparison favoring this over alternative models for mortality). Since we observe the same individuals over two waves, our data exhibits within-individual variation in the mortality determinants – health levels/changes and socioeconomic characteristics. This makes it possible to use two observations per individual, which is appealing in two respects. First, we almost double our sample size. We should note the necessary transformation in the dependent variable. This equals, for the first period, age at death as observed in that period, i.e.: actual age at death, if it occurred until the end of the period, and age by then, otherwise (Gutierrez, 2002). Similarly, in the second period, in which only survivors are observed, the dependent variable equals the age at death, if that occurred until the end of follow-up period, and age by then, otherwise (Gutierrez, 2002). Secondly, we are able to account for the effects of unobserved time-constant factors (e.g. childhood conditions, genetic factors, family history), which may influence the intrinsic propensity to die at any moment in time,

² This might also be achieved with a Weibull model, so we also consider this as a robustness check.

over and above the effects of observed time-varying factors. This acts as multiplicative effect on the Gompertz hazard, assumed to follow a Gamma distribution (Gutierrez, 2002).

The mortality hazard function for individual i at time t is given by:

$$\lambda_i(t) = \lambda_0(t) * \exp[H_{it-1}\beta + \Delta H_{it}\gamma + X_{it}\theta] * \eta_i$$

where $\lambda_0(t) = \exp(\mu t)$ is the baseline hazard for the Gompertz Distribution with shape parameter μ , and $\exp[H_{it-1}\beta + \Delta H_{it}\gamma + X_{it}\theta]$ is the proportional hazard with parameters β , γ and θ . X is a vector of individual specific time-varying covariates including gender, marital status, education, labor force status, income categories and ADLs. The vector H contains *lagged* health variables and ΔH includes health change variables. We account for individual-specific time-invariant characteristics (unobserved heterogeneity) by including the multiplicative term η_i , assumed to be distributed as Gamma $(1, \sigma)$. This is a common random effects specification, applied to a hazard model.

The association between retrospective/prospective health changes and mortality is first examined controlling for SAH level and socioeconomic characteristics only. We consider retrospective and prospective health changes in separate as well as joint models, and compare the magnitude and significance of their hazard ratios, as well as the overall fit of the models. Secondly, we control for a set of other - arguably more objective - indicators of health levels and changes: the prevalence and onset of new chronic conditions, and ADL and mobility problems indices. This makes it possible to assess whether the association between retrospective/prospective health changes can be explained by these additional indicators or, on the contrary, those measures of health changes carry extra information relevant for mortality.

4.4 Results

Table 4.2 presents a comparison between prospectively computed and retrospectively reported health changes. The proportion of cases reporting no health change is about 60% for the prospective measure, while the remaining cases report better or worse health in

equal shares. A more sizeable share (80%) reports the same health retrospectively (about 11% report and improvement and 9% a deterioration). Observations located on the diagonal represent congruent reports across measures (only 60.2% of cases). Incongruent reports (39.8%) could reflect noise in one (or both) of the measures. The most serious violation of congruency occurs when according to one of the measures health has improved whereas according to the other health has deteriorated (2.72%). On the other hand, in 8.97% of cases the prospective health measure identifies no change, while the retrospective measure does. Table 4.2 also shows the proportions of people who died during the follow-up period by prospective and retrospective health changes. These descriptive statistics do not, however, show a smaller risk for those whose health improved than for those whose health remained the same. Our hazard regression analysis will assess whether these relationships hold when controlling for a number of other mortality determinants, and considering full survival, rather than just a binary indicator.

Table 4.2: Comparison of prospectively computed with retrospectively reported health changes and proportions dying during follow-up through 2004 by category of health change.

Prospectively Computed Health Changes	Retrospectively Reported Health Changes			Total # of Observations	Proportion dying during follow-up
	Better	Same	Worse		
Better	3.05%	14.68%	1.36%	1175	9.10%
Same	4.35%	52.42%	4.61%	3775	7.07%
Worse	1.36%	13.35%	4.76%	1198	11.60%
Total # of Observations	540	4947	661	6148	
Proportion dying during follow-up	9.07%	6.46%	21.78%		8.6%

Hazard regression results for SAH levels and health changes are shown in Table 4.3 for three models considering either prospectively computed or retrospectively reported changes, or both simultaneously. The first row shows results of models controlling only for health changes, SAH levels and demographic and socioeconomic characteristics. A hazard ratio of one indicates that a variable has no effect on mortality. A hazard ratio of more/less than one indicates that a variable is associated with higher/lower mortality. As widely observed in the literature (Idler & Benyamini, 1997; Van Doorslaer & Gerdtham, 2003; Dowd and Zajacova, 2007; Huisman et al. 2007), we obtain a strong effect of SAH on longevity: being in the good to bad categories compared to the very good health category

significantly increases the hazard of dying. Over and above the SAH level, a prospective SAH change and a retrospective SAH changes also affect mortality, either when considered separately or jointly in the same specification. The specification considering prospective *or* retrospective changes separately estimates that: i) respondents with a *prospective* decline in their health are about twice as likely to die as those prospectively reporting an improvement (hazard ratio=2.13), and ii) for individuals who *retrospectively report* a health decline, the hazard of dying is 1.83 higher than for those reporting a health improvement. The mortality hazard for those remaining in the same SAH level (compared to the previous period) is significantly higher than for those moving into better SAH, while those who retrospectively report the same health are not more likely to die than those who report an improvement. The results of the specification including both prospective and retrospective changes are consistent with the separate models, except that the estimated effects of changes are slightly smaller and less significant. In sum, unconditional on more objective health information both health change measures predict mortality, but the effect is stronger and more significant for prospective changes.

The second row of Table 4.3 presents results for the full model which controls, in addition, or prevalence and onset of chronic conditions and ADL indices³⁴. As expected, the effects of SAH levels as well as of both measures of health changes on mortality are lower in the full model. However, only the effect of a retrospectively reported health deterioration becomes insignificant, while the hazard ratio for a prospectively computed health deterioration remains significant (CI: 1.15- 2.42). While the full model with retrospective changes fits the data as well as the one with prospective changes in SAH, the results do suggest that prospectively reported changes in SAH are better predictors (higher significance and hazard ratios). Goodness of fit of both models improves with the inclusion of prevalence and onset of chronic conditions: the Akaike Information Criterion (AIC) improves from 417.71 (422.84) to 355.60 (355.70) for reported (computed) changes.

³ We have also considered a version of the full model with prospectively computed health changes which includes current instead of lagged SAH to illustrate the issues discussed in the measures section, regarding the most appropriate control for health levels in this case. As expected, the resulting effects of prospective changes are of the reverse sign. We obtain (insignificant) hazard ratios of 0.961 and 0.990, respectively, for the categories “same” and “worse” of the prospective variable. These results are in line with those of Benitez-Silva and Ni (2008) where self-assessed health levels are specified in this way.

⁴ As a robustness check, we also estimated full models using a Weibull instead of a Gompertz distribution. This alternative specification fits the data slightly worse (with prospective health changes, BIC = 709.77, with retrospective changes, BIC = 710.02) and yields very close hazard ratios and significance for the effects of prospective and retrospective health changes. Results can be obtained upon request.

Table 4.3: Mortality effects of self-assessed health levels and prospective and retrospective changes: hazard ratios and 95% confidence intervals

Controls	Prospectively Computed Health Changes				Retrospectively Reported Health Changes				Retrospectively Reported and Prospectively Computed Health Changes			
	Hazard Ratio	95% CI	AIC	BIC	Hazard Ratio	95% CI	AIC	BIC	Hazard Ratio	95% CI	AIC	BIC
1. SAH and health changes only	SAH good	(0.84-2.12)	422.84	570.77	1.112	(0.71-1.74)	417.71	565.64	1.213	(0.76-1.91)	412.86	574.24
	SAH fair	(1.74-4.78)			1.978***	(1.22-3.22)			2.320***	(1.39-3.84)		
	SAH s om. g/p	(2.29-7.47)			2.308***	(1.33-4.01)			2.968***	(1.64-5.35)		
	SAH poor	(5.46-25.16)			5.358***	(2.65-10.83)			7.558***	(3.53 -16.17)		
	Prosp. Same	(0.99-1.94)			-	-			1.340*	(0.95-1.87)		
	Prosp. Worse	(1.45-3.13)			-	-			1.778***	(1.2 -2.61)		
	Retros p. Same	-			0.86	(0.55-1.35)			0.813	(0.511-28)		
	Retros p. Worse	-			1.833**	(1.11-3.03)			1.538*	(0.91-2.57)		
2. SAH and health changes plus prevalence and onset of chronic conditions	SAH good	(0.71-1.77)	422.84	570.77	1.025	(0.65-1.62)	417.71	565.64	1.108	(0.70-1.74)	412.86	574.24
	SAH fair	(0.97-2.83)			1.363	(0.81-2.30)			1.586*	(0.92-2.71)		
	SAH s om. g/p	(0.89-3.23)			1.255	(0.68-2.33)			1.588	(0.83 -3.02)		
	SAH poor	(1.43-7.90)			2.268**	(0.99-5.20)			3.100**	(1.31 -7.31)		
	Prosp. Same	(0.87-1.68)			-	-			1.205	(0.86 -1.67)		
	Prosp. Worse	(1.15-2.42)			-	-			1.568**	(1.06 - 2.29)		
	Retros p. Same	-			0.95	(0.60-1.50)			0.913	(0.58 - 1.43)		
	Retros p. Worse	-			1.542	(0.90-2.61)			1.369	(0.81 - 2.30)		

Notes: 1. Chronic conditions include: high blood pressure, back problems, diabetes, heart diseases, stroke, cancer, ankylosis, rheuma, lung diseases, , stomach diseases, nervous diseases, intestine diseases, skin diseases, mental health; 2. Other controls : male, education (low, middle, high), working status (unemployed, disabled, retired, housework), marital status (married, divorced, widowed), income (middle, high), number of ADL'S, number of mobility problems 3. All models are estimated with Comperitz Hazard Model, with gamma distributed shared frailty (heterogeneity) term. 4. *, **, *** refers to significance at 10%, 5% and 1% respectively. 5. AIC and BIC represents Akaike Criterion and Schwarz Criterion results for model selection.

The specification including *both* measures of health changes gives results which are consistent with the separate models, confirming the better predictive ability for longevity of prospective reports than the retrospective.

Table 4.4: Mortality effects of prevalence/onset of chronic conditions for models using prospective and retrospective health changes : hazard ratios and 95% confidence intervals

	Prospectively Computed Health Changes		Retrospectively Reported Health Changes	
	Hazard Ratio	95% CI	Hazard Ratio	95% CI
Prevalence (in previous period) of:				
High blood pressure	1.29	(0.94-1.76)	1.27	(0.92-1.76)
Back Pain / Problems	0.58***	(0.40-0.85)	0.59***	(0.40-0.87)
Diabetes	1.53 **	(1.03-2.27)	1.60 **	(1.07-2.39)
Heart Diseases	2.18 ***	(1.53-3.09)	2.19***	(1.53-3.15)
Stroke	1.07	(0.46-2.49)	1.14	(0.47-2.75)
Cancer	2.25 **	(1.21-4.16)	2.20**	(1.16-4.20)
Ankylosis	0.59 **	(0.39-0.88)	0.56 ***	(0.37-0.86)
Rheuma	1.01	(0.52-1.96)	1.02	(0.52-2.01)
Lung Diseases	1.30	(0.90-1.86)	1.27	(0.88-1.85)
Stomach Diseases	1.20	(0.59-2.42)	1.17	(0.57-2.42)
Nervous System Diseases	1.55	(0.72-3.37)	1.60	(0.70-3.66)
Intestine Diseases	1.06	(0.54-2.09)	1.05	(0.52-2.13)
Skin Diseases	0.95	(0.52-1.72)	0.90	(0.49-1.67)
NHP Mental Health Problems	1.25	(0.91-1.72)	1.22	(0.88-1.70)
Onset of (in current period):				
High blood pressure	0.53*	(0.27-1.07)	0.59	(0.30-1.18)
Onset Back Pain / Problems	1.30	(0.79-2.14)	1.26	(0.76-2.12)
Onset Diabetes	0.90	(0.36-2.25)	0.91	(0.36-2.31)
Onset Heart Diseases	1.01	(0.55-1.87)	1.01	(0.54-1.89)
Onset Stroke	1.30	(0.41-4.13)	1.55	(0.49-4.91)
Onset Cancer	5.70***	(2.97-10.95)	5.81***	(3.04-11.10)
Onset Ankylosis	0.74	(0.41-1.33)	0.74	(0.41-1.36)
Onset Rheuma	0.53	(0.20-1.39)	0.49	(0.18-1.31)
Onset Lung Diseases	3.00***	(1.75-5.14)	3.06***	(1.77-5.31)
Onset Stomach Diseases	1.52	(0.53-4.37)	1.52	(0.52-4.42)
Onset Nervous System Diseases	1.74	(0.52-5.84)	1.58	(0.46-5.45)
Onset Intestine Diseases	0.85	(0.31-2.38)	1.04	(0.39-2.80)
Onset Skin Diseases	1.17	(0.70-1.96)	1.09	(0.65-1.85)
Onset NHP Mental Health Prob.	0.95	(0.59-1.54)	0.92	(0.57-1.51)
ADL index	1.08	(0.94-1.23)	1.04	(0.91-1.20)
Mobility problems index	1.16*	(0.99-1.37)	1.18**	(1.01-1.40)
Log Likelihood	-125.85		-125.80	
AIC	355.70		355.60	
BIC	705.34		705.24	
N	6148		6148	

Table 4.4 shows further results of the full models, in particular, the association between prevalence/onset of chronic conditions and mortality. Having heart disease and cancer, suffering the onset of high blood pressure, cancer and lung diseases, and having more mobility problems all increase the risk of dying.

4.5 Discussion

In this paper we have examined (a) whether, over and above the level of health itself, reported changes in health still contribute significantly to better prediction of survival outcomes, and (b) how this predictive ability compares between retrospectively reported and prospectively measured changes in SAH over time. We answer these questions using data from a unique Dutch survey which couples longitudinal measurement of health reporting over several waves of a panel with mortality follow up over a period of 11 years. Our findings confirm our expectation that information regarding recent health changes carries additional information to that contained in the reported health level. This is even true after controlling for both the prevalence and the onset of a large set of chronic conditions.

Our study adds to the relevant literature in several respects. Firstly, the simultaneous availability of several waves of longitudinal subjective and objective health data, of retrospectively reported health changes and of a fairly long (11 years) mortality follow-up allowed for a proper comparative assessment of survival prediction. Secondly, by using appropriate survival modeling strategies for panel data, we were able to account not only for observed determinants of mortality but also for unobserved time-invariant factors (such as influence of childhood conditions, genetic factors or family history) which may influence the intrinsic mortality risk. Finally, we were also able to control for a battery of self-reported more objective measures of health levels and changes, often alleged to carry more valuable information about mortality risk.

We observe that for a large proportion of individuals, prospective health changes are not congruent with retrospective health changes. Reporting no retrospective health change at the same time as a prospective health change could be, for example, due to recall bias in the retrospective measure, or to the same true level of health being considered as better/worse when evaluated at a later period. But some incongruence could be merely a reflection of the different nature of the measures. For example, cases with no prospective health changes and with some retrospective health change could mean that health changes captured by the latter measure are not large enough to cause a category jump in SAH, suggesting that retrospective changes may be more informative than prospective changes.

In particular, prospective changes may suffer from floor and ceiling effects: for individuals in the lowest/highest category, this measure captures no change if their health deteriorates/improves further. To minimize this problem, we add indicators of whether individuals reported the top or bottom category in both periods in our models. These indicators were not significant in our models and so we report results of models that do not include them.

The main findings can be summarized as follows. First, our findings confirm our prior expectation that ratings of self-reported health do not entirely capture the information contained in changes (and *vice versa*): recent health changes do have predictive power for subsequent survival over and above the effects of current self-reported (subjective) health indicators, as well as over presence and onset of chronic conditions. This suggests that health variability itself, over and above the level of health around which this variability occurs, is a risk factor. Our results are consistent with those of previous studies showing that SAH and corresponding health trajectories have an independent effect on mortality (Schwarze et al, 2000; Ferraro and Kelley-Moore, 2001; Lyra et al, 2009).

Secondly, we find that both prospectively and retrospectively reported health changes predict mortality over and above SAH level in a model without controlling for any other more objective health information. However, even though the information criteria indicate a similar goodness of fit of the two models, prospectively reported changes in SAH over time do have higher predictive power for mortality risk than retrospectively reported health changes when controlling for prevalence and onset of new chronic diseases. Finally, in a model which controls for both health change measures simultaneously, prospective health changes predict mortality whereas retrospective changes do not. We therefore conclude that while the collection of retrospective health information is a cheaper and simpler alternative to the more expensive and cumbersome prospective collection of longitudinal health data, it does not have better predictive power. Moving from longitudinally measured prospective changes to retrospective changes collected in cross-sections leads to some loss of information about mortality risk.

A recent study favored the use of retrospective self-reported health changes, after showing that this measure predicts subjective longevity expectations whereas prospective SAH changes do not (Benitez Silva & Ni ,2008). Our results reveal different conclusions. First,

we showed that both measures are predictive of mortality if one does not control for any other objective health information. Secondly, retrospectively reported health changes do not predict mortality after controlling for objective health information, while prospective SAH changes do. This discrepancy is due to the different way in which we controlled for SAH levels while capturing the resulting effect of prospective health changes (as shown above in measures section and endnote 3). In addition, this could also partly be due to the use of a different outcome variable. In particular, there is some evidence to suggest that subjective longevity expectations are subject to bias (Bassett & Lumsdaine, 2001).

While our study offers a number of important advances over earlier research, it also has limitations. One obvious limitation is the use of all-cause mortality as our main outcome measure. Clearly, not all ill health is fatal and leads to higher mortality, while other causes of mortality (like accidents) are not health-related. We were unable to exclude mortality from causes clearly unrelated to prior health evolution. However, while such exclusion might further improve the predictive ability of SAH levels and changes, it is unlikely to affect the comparison between retro- or prospectively measured changes. As recently discussed by Jylhä (2009) and Huisman and Deeg (2010), self assessed health is influenced by health-relevant information, but also by psychological filters such as perception, interpretation and memory. Therefore, the formation of health perceptions may involve a number of cognitive processes, such as recall of relevant experience and evaluation of relevant information, which may be dependent on cognitive ability. Unfortunately, our data does not include measures of cognitive ability, and so we were unable to assess its role in explaining the predictive ability of health change measures for mortality. This is something for a future research agenda.

Chapter 5

Cognition and Education Differences in the Formation of Survival Expectations: Are Some Folks Better Able to Predict their Own Demise?

Decisions about retirement, pensions, savings and health behaviour are presumed to be contingent on longevity expectations. We use eight waves of the U.S Health and Retirement Study (HRS) to examine whether the predictive accuracy of subjective survival expectations with respect to actual mortality varies with education and cognition. Predictive power increases with both education and cognition. An important reason for this is that the less educated and cognitively able are less likely to revise survival expectations downward in response to new information on objective risks, such as the onset of cancer or the occurrence of a stroke. These systematic biases in the formation of survival expectations suggest low education and cognition groups may be making sub-optimal decisions with respect to their finances and health.

5.1 Introduction

Economic decisions such as those concerning retirement, savings and health behaviour are presumed to depend on expectations of longevity. Mistaken expectations could have terrible consequences. Inadequate retirement savings as a consequence of overly pessimistic expectations might result in an impoverished old age, while overly optimistic expectations in the presence of health risks will result in inappropriate adjustment of health behaviour and the premature loss of life. Identification of systematic biases in expectations is an important first step toward avoiding such calamities. Knowledge and cognition are two potentially important sources of bias. The formation of accurate survival expectations requires ability both to perceive environments, health risk factors and signals of health conditions and to accumulate and process information on the mortality risks associated with these factors. Perceptions of health risks and the effective use of related information are likely to vary with cognitive functioning. Education may impart health knowledge but more importantly it may develop information processing skills.

In this paper, we use eight waves of the US Health and Retirement Study (HRS) of older Americans to examine whether individuals' ability to predict their own survival varies with education and cognitive functioning. We find that high school drop-outs and individuals with lower cognitive functioning report survival expectations that are less predictive of actual survival than the expectations reported by their higher educated and more cognitively able counterparts. There are two interpretations of this result, which are not mutually exclusive. As suggested in the previous paragraph, it could be that less knowledge and lower cognition impede the perception of risks and the formation of accurate expectations in response to them. The second explanation is that individuals with less education and cognitive capacity find it more difficult to express expectations in the form of probabilities, which is what they are asked to do in the survey. Questions about probabilities require comprehension of the concept of probability and interpretation of the meaning of the probability question (Schwarz and Oysermann, 2001). Even if the cognitive (in particular, memory) skills of the respondent are high, low education may restrict his/her ability to report probabilities.

Using the older AHEAD cohort of the HRS, Hurd et al. (1998) find that the less cognitively able are less likely to respond to the survival expectations question and, if they do, are more likely to give a 'focal response' (0, 0.5 or 1) or a rounded probability. Manski and Molinari (2010), who focus on rounding for survival expectations, show that rounding can seriously limit the information on the means of the true underlying probabilities given other covariates. With minimal assumptions on the rounding process, they find large and uninformative bounding intervals for the conditional expectations of interest for subjective survival expectations in the HRS. Kleinjans and Van Soest (2010) show that there is less rounding and non-response in the reporting of a variety of other subjective expectations among the better educated and higher cognition groups. Elder (2010) argues that subjective survival expectations predict economic outcomes only because they act as a proxy for cognition.

While the definition of measurement error with respect to subjective expectations data is far from clear (Elder, 2010), difficulties in expressing expectations in probabilities presumably generate noise, which will downwardly bias the predictive power of survival expectations with respect to actual survival. If the measurement error is greater for the lower educated and the cognitively impaired, then the estimated correlation between subjective and actual survival will be weaker for these groups. This has implications for empirical work, but it does not necessarily have substantive implications in terms of individuals' decisions. On the other hand, if survival expectations are less predictive of actual survival for the less educated/cognitively able because they form less accurate expectations in response to information, then this has profound consequences for the welfare interpretation of outcomes resulting from individual decision making.

To better discriminate between these two explanations, we examine directly how the incorporation of information in survival expectations varies with both education and cognitive functioning. Using the first two waves of the HRS, Hurd and McGarry (2002) demonstrate that survival expectations respond to longevity-relevant events including the death of a parent and the onset of a serious health condition, such as cancer. Following the framework for updating risk perceptions proposed by Viscusi and O'Connor (1984) and Viscusi (1985), Smith et al. (2001) find that new information in the form of the onset of serious health conditions and new limitations on activities significantly reduces reported expected longevity in the HRS. We add to these studies by examining whether individuals

of lower cognitive ability and education are less responsive to events that objectively provide new information on mortality risks. We find that high school drop-outs and individuals with less than average cognitive functioning generally do not revise survival expectations downward when they contract a new disease or health condition while the higher educated and cognitively more able do.

While certainly not decisive, this points to impaired ability to perceive and utilise information on mortality risks among the less educated and cognitively able. This has a number of important implications for policy relating to individual decisions concerning pensions, saving, health care and health behaviour. It also helps explain the well-established education gradient in health. An increasingly popular explanation for this gradient is that the knowledge and cognition deficit of the less educated impedes ability to process information on risky health behaviours, effective but complex new health technologies and the medical management of chronic conditions (Kenkel, 1991; Goldman and Smith, 2002; Lichtenberg and Lleras-Muney, 2006; Cutler et al. 2011). Lange (2011) finds support for the hypothesis in the fact that the less educated are less likely to incorporate objective risks of cancers in subjective assessments of those risks. They are then less likely to take preventive measures to reduce those risks. Our finding that low education and cognition reduces the likelihood of revising survival expectations downward when objective risks, such as taking a stroke, emerge, suggests that low education/cognition individuals are less likely to change behaviour and seek appropriate medical treatment in response to conditions that pose a serious threat to their longevity.

The paper is organized as follows. Section 2 explains the HRS data and variables used in the analysis. Estimates of the ability of subjective survival expectations to predict actual mortality and how this varies with education and cognition are presented in Section 3. In Section 4, we estimate how the formation of survival expectations responds to new information on objective risks to longevity and how this response differs by education and cognitive functioning. We conclude by discussing the implications of the results.

5.2 Data: Measures and Descriptive Analysis

Our data come from the first eight waves (1992-2006) of the Health and Retirement Study (HRS). The HRS is a national US panel study of cohorts born between 1931 and 1941 and their spouses (including younger) if married. We use two cohorts of the HRS study: the HRS cohort and the AHEAD cohort. Participants in the HRS cohort have been interviewed every two years since 1992. Individuals in the first wave of the HRS cohort range from 51 to 61 years of age while the AHEAD cohort is composed of participants born before 1924. Participants in AHEAD cohort were interviewed in 1993, 1995, 1998 and subsequently every two years. We took all background variables from the RAND HRS data files (St. Clair et al. 2009) and constructed those on cognitive functioning (on memory and arithmetic ability) from the raw HRS files (following Ofstedal, Fisher, and Herzog, 2005). We use cognition measures in this analysis from wave 3 (1996) onwards.

5.2.1 Measurement of Subjective Survival Expectations (SSE)

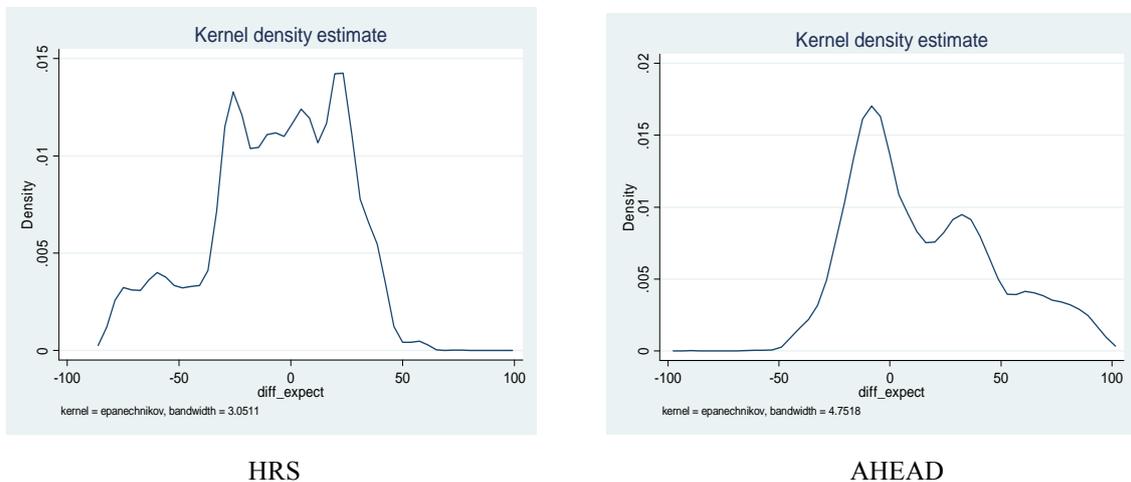
The HRS asks about the percent chance that a respondent will live to age T , where T depends on the respondent's current age. The target age T is 75 for respondents younger than 65.¹ We denote individuals' responses to this question as SSE_{75} . For those older than 65, the target age in waves 1-4 is 85, which we denote as SSE_{85} . In waves 5 to 8, respondents are asked the percent chance of surviving another 11 to 15 years, denoted as SSE_{11-15} . Specifically, those aged 65 to 69 are asked about the likelihood of surviving to age 80, those aged 70 to 74 are asked about the likelihood of surviving to age 85, and so on.

We deal with these variations by calibrating SSE_T with life table probabilities (O_T) for each age and gender. These life table probabilities are the implied probabilities from the Vital

¹ Respondents younger than 65 also responds to the question on the probability to live until 85 in waves 1-4.

Statistics life tables² that someone of the respondent's age and gender will live to be age T . We construct an individual specific measure of how far SSE_T deviates from life table probabilities at any age. This provides a summary measure of the extent to which individuals' expectations are above or below the actuarial average. We compute the measure SSE_{diff} using the difference between subjective SSE_T and O_T probability for given age and gender as follows: if age ≤ 65 , $SSE_{diff} = SSE_{75} - O_{75}$; if age > 65 in waves 1-4, $SSE_{diff} = SSE_{85} - O_{85}$; and if age > 65 in waves 5-8, $SSE_{diff} = SSE_{11-15} - O_{11-15}$). Figure 5.1 shows the distribution of this variable for the AHEAD and HRS cohorts. Table 5.1 shows the age specific average of these differences for females and males. On average, as also shown by Elder (2010), female individuals in the HRS cohort tend to understate their survival chances whereas males' reported SSE is closer to life table probabilities. The mean difference moves from negative to positive as the age of respondents' increases (Elder, 2010). In the much older AHEAD cohort, especially male respondents tend to overstate survival probabilities relative to life table data.

Figure 5.1: Distribution of difference between subjective and life table survival probabilities for HRS and AHEAD cohort



² Life table probabilities are published by National Center for Health Statistics. These probabilities are available within the HRS data. The probability of survival is calculated as the number surviving at age T divided by the number surviving to respondent's age, for males or females, as appropriate.

Table 5.1: Distribution of difference between subjective and life table survival probabilities (SSE_{diff}) by age and gender in HRS and AHEAD cohort.

HRS Cohort	Males			Females		
Age range	Mean	Std. Dev.	N	Mean	Std. Dev.	N
50-54	1.39	30.12	2642	-8.36	28.66	3346
55-59	-0.25	29.71	6542	-10.91	29.58	8227
60-64	-4.76	28.85	7895	-13.37	29.38	10052
65-69	-4.08	29.07	4779	-12.06	29.62	6299
70-74	7.64	30.85	2150	-3.23	30.88	2783
>75	12.12	32.87	79	1.05	32.98	114
AHEAD Cohort						
70-74	14.23	30.54	187	-5.16	33.76	26
75-79	14.88	31.63	774	4.86	32.51	969
80-84	20.26	32.47	1567	12.50	31.32	2458
85-89	20.00	30.41	885	18.62	32.62	1497
90-94	-0.03	8.72	9	28.03	34.81	12

Notes: Based on Wave 1 - Wave 8 data.

5.2.2 Measures of cognitive functioning and education

The HRS contains measures of cognitive functioning based on well validated measured tests. These assess cognitive functioning within several domains. First, episodic memory is assessed through a test of verbal learning and recall. The episodic memory task consists of learning a list of ten common words. The interviewer reads a list of 10 words (e.g., book, child, hotel, etc.) to the respondent, who is asked to recall as many words as possible from the list in any order. Then, immediate and delayed recall tests are taken. Immediate recall follows directly, while delayed recall is tested after some other tests are performed. There are four possible lists of nouns for this purpose. Respondents are asked a different list of words from the lists that they, and their spouse, had to answer during the previous wave. Different nouns were used for each of four successive waves. This is done in order to avoid that the respondent remembers the words from the previous wave. Total recall score is the sum of the number of target words recalled in the immediate and the delayed recall test (score ranging from 0 to 20). This cognitive function has been shown to be particularly affected by aging, with some studies even arguing that it is among the first to decline with aging (Souchay et al., 2000; Anderson and Craik, 2000; Prull et al., 2000).

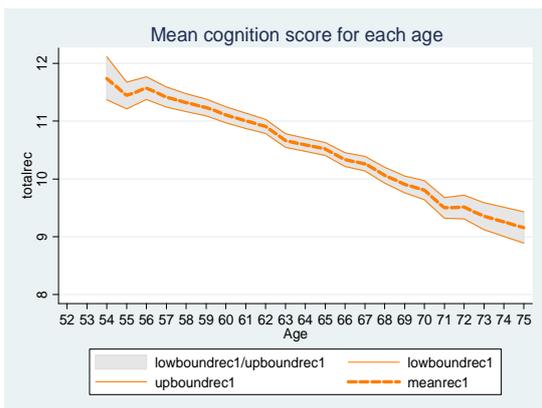
Working memory, i.e. the ability to process and store information simultaneously, is assessed through a serial 7's subtraction test. In this test, respondents are asked to subtract 7 from 100 and continue subtracting 7 from each subsequent number for a total of five subtractions. This test thus requires respondents to perform a basic arithmetic operation (subtracting 7), while memorizing the result from the previous subtraction that is required as an input in the following operation. The serial 7's subtraction test score counts each correct subtraction, leading to scores between 0 and 5. The second test related to arithmetic ability asks the respondents to count backwards as quickly as possible, starting from 20 for 10 continuous numbers. Respondents were allowed two trials for this exercise. Scores were recorded as 0 if incorrect or "don't know/unable to do" on both tries, 1 if incorrect on first try, but correct on second try, 2 if correct on first try).

Figure 5.2 to 5.4 show respectively the age-related evolution of total recall, serial 7's and backwards counting tests for the HRS and AHEAD cohort. The total recall score appears indeed strongly related to age, whereas the two other scores are less age-dependent. Younger respondents have a substantially higher level of cognitive functioning as measured by total word recall than older respondents. This is less pronounced for serial 7 subtractions and backwards counting for HRS Cohort. The serial seven score declines more with age for the AHEAD cohort and declines less with age for the backwards counting. Mean backwards counting scores for the HRS and AHEAD cohort are closer to each other whereas the younger HRS cohort has higher scores than AHEAD for the other two scores. In our analyses, we account for age-related cognitive decline by employing age-sex standardized scores for these tests.³ We construct a measure of arithmetic ability from the sum of the standardized serial 7 score and standardized backwards counting score. The second measure we use is the standardized total recall score.

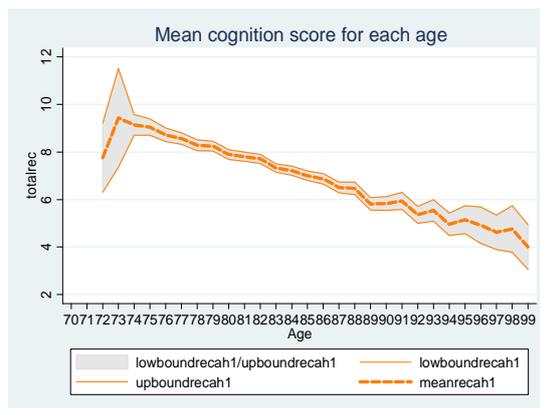
In order to examine the relationship between SSE and measures of cognition in the HRS, we classify individuals into one of three categories based on their standardized scores of total recall and arithmetic ability separately, as follows: i) *low cognition group*: those with age-sex standardised cognition below the mean observed in the first and last waves; ii) *high cognition group*: those with standardised cognition above the mean in first and last

³ Age and sex standardized test scores are generated by subtracting from the actual scores the age-sex specific means (which is done by taking residuals from a regression). We then standardize these residuals to have a variance of 1.

Figure 5.2: Age trend in Recall Score

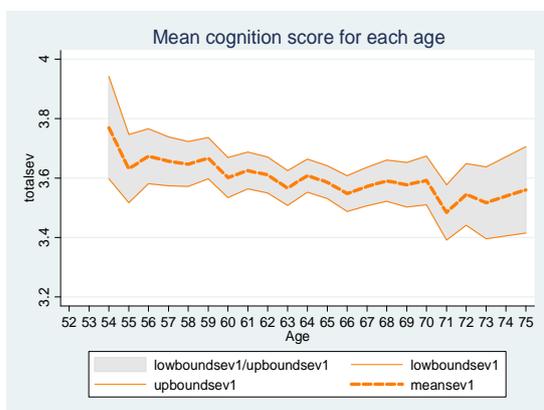


HRS (Waves 3-8)

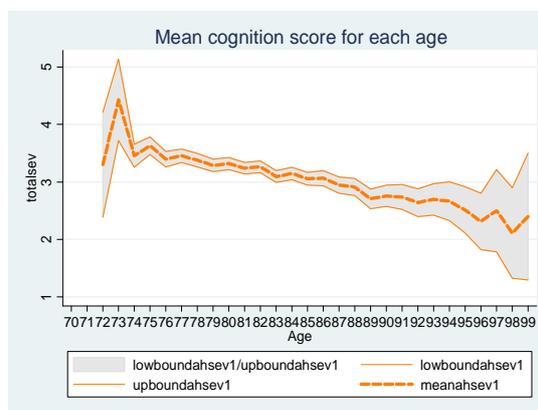


AHEAD (Waves 3-8)

Figure 5.3: Age trend in Serial 7 score

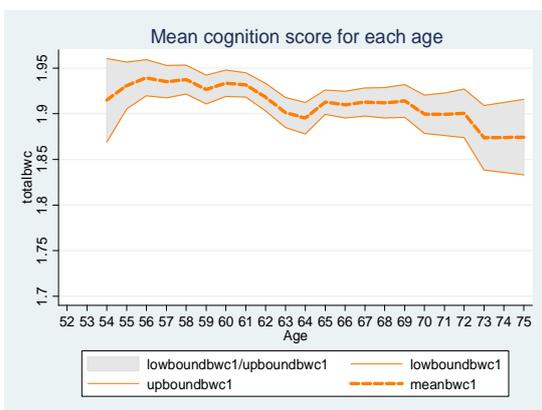


HRS (Waves 3-8)

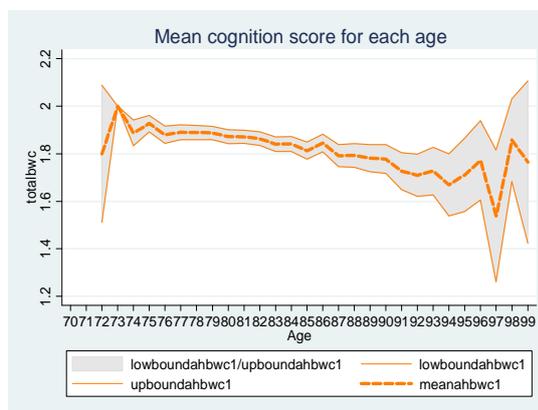


AHEAD (Waves 3-8)

Figure 5.4: Age trend in Backwards Counting



HRS (Waves 3-8)



AHEAD (Waves 3-8)

waves iii) *changing cognition group*: those moving from standardized cognition below the mean in first wave observed to above the mean in last and those moving from standardized cognition above the mean in first wave observed to below the mean in last wave (these two groups are combined due to the small sample sizes of each group).

Table 5.2 shows the association between SSE (adjusted by life table probabilities) and each dimension of cognition⁴ and education for HRS cohort. There is a strong positive gradient in SSE by cognition, in particular for the total recall score. SSEs are much closer to life table probabilities for those in the upper cognition groups, but further below the life table averages for the low and changing cognition groups.

We also investigate the number of missing observations in SSE by cognition groups. These missing observations may be related to cognition if less able individuals are less willing to answer this question. We indeed observe that the percentage of missing values is much larger for the lower cognition groups, which suggests that those with low cognitive functioning may have difficulty forming expectations, or at least answering questions on expectations. In the empirical analysis we take this into account by replacing the missing values with the mean and adding a dummy variable representing the missing observations. A reported probability of 50 percent may also indicate difficulty in expressing expectations in terms of a probability (Kleinjans and Van Soest, 2010). These ‘focal point responses’ are slightly higher in the low recall score cognition group. Differences in percentage of focal responses between low and high arithmetic groups are much smaller.

The categorical education groups in HRS are obtained using the number of years of education. We use the following groups: high school drop outs (0-11 years of education) and GED (General Educational Development)⁵ test group, high school graduate, some college (if the respondent has a high school diploma or GED and years of education over

⁴ Since survival chances of respondents in the much AHEAD cohort are extremely below the life table averages, this gradient is much less clear for the AHEAD Cohort.

⁵ General Educational Development (or GED) tests are a group of five subject tests which, when passed, certify that the taker has American high school-level academic skills. Only individuals who have not earned a high school diploma may take the GED tests. Common reasons for GED recipients not having received a high school diploma include immigration to the United States or Canada, homeschooling, leaving high school early due to a lack of interest, the inability to pass required courses or mandatory achievement tests etc.

12) and college and above (if respondent has bachelor degree or higher). In Table 5.2, we do a similar analysis for SSE_{diff} by education groups. The association between reporting survival probabilities close to life table probabilities and education is evident. On average, groups with higher education report survival chances closer to life table data and groups with lower education report survival chances lower than the life table data. Given the established education gradient in mortality (Cutler et al, 2011), the fact that SSE is further below the life table average for low education/cognition groups by no means indicates that they are more mistaken in their expectations. The percentage of non-response and focal 50 percent answers is much higher in the lower educated group.

Table 5.2: Distribution of difference between subjective and life table survival probability by cognition and education - HRS Cohort.

Recall Score	Mean	Median	Std.Dev	N	% not responding	% Focal response
Low cognition	-9.434	-7.092	30.34	7822	12.98	25.95
Changing Cognition	-4.77	-1.910	27.82	8309	9.86	24.55
High Cognition	-1.616	1.340	25.77	7536	7.69	23.27
Arithmetic ability score						
Low Cognition	-8.586	-5.831	32.05	4096	16.89	24.65
Changing Cognition	-5.725	-2.724	28.74	6165	10.94	24.60
High Cognition	-3.13	-0.381	26.24	11756	7.86	24.94
Education						
High school drop out and GED	-14.31	-14.12	34.75	10016	36.94	25.1
High school grad.	-7.96	-5.83	29.08	12703	17.55	26.9
Some College	-3.27	0.177	27.56	7666	15.69	23.6
College	-0.25	3.95	24.51	7167	12.90	20.2

Notes: 1. Waves 3-8 are used. 2. Low (high) cognition group consists of those below (above) the mean standardized score in first and last waves. Changing cognition group consists those moving from standardized cognition below the mean in 1st wave observed to above the mean in last and those moving from standardized cognition above the mean in 1st wave observed to below the mean in last wave. 3. Focal responses correspond to reported probabilities of 50%.

In Table 5.3, we break down the differences further and cross-tabulate by both education and cognition. There are more respondents in the higher educated group with a higher cognition score and their reports of survival chances are close to life table probabilities. Similarly, there are more low educated people in the lower cognition scores group and, on average; they report much lower probabilities of survival. Within-education level differences in SSE by cognition are smaller than within-cognition group differences by education. SSE is much lower in the low education/high cognition groups than it is in the

high education/low cognition groups. Therefore, the table shows that: i) there are clear education gradients, irrespective of cognition; ii) for the recall score (which is intended to signal dementia), there is a clear cognition gradient at all education levels; iii) for the arithmetic score, there is a cognition gradient at low but not at high education.

Table 5.3: Distribution of difference between subjective and life table survival probability by cognition for education groups in HRS Cohort

Education Groups	Recall score		Arithmetic Ability	
	Low Group	High Group	Low Group	High Group
High Sch. Drop out and GED	-14.33(34.19) N=2639	-10.45(31.19) N=601	-12.53(35.08) N=1696	-10.16(30.84) N=1230
High Sch. Grad.	-8.70(28.89) N=2865	-4.53(26.78) N=2265	-8.36(30.89) N=1445	-5.13(26.66) N=3850
Some College	-5.41(28.17) N=1380	-1.30(25.07) N=1933	-2.72(28.85) N=640	-2.78(26.13) N=2947
College	-3.79(23.44) N=938	2.60(23.13) N=2677	-0.216(25.88) N=315	-0.97(23.38) N=3729

Notes: 1. Standard deviation in parenthesis. 2. Compositions of changing cognition group is less homogenous with smaller sample sizes; therefore we only present the distributions for low and high groups.

5.2.3 Mortality Information

The HRS determines the vital status of respondents in any particular survey wave through tracking: a respondent is observed to be alive if s/he was interviewed or contacted directly by an interviewer during the wave, or was said to be alive by a spouse or partner, or was not reported dead. If no informative contact was made (if partners or family members could not be reached), the respondent's vital status is classified as unknown.⁶ As a result, there is a 12 year follow-up (from 1994 until 2006) for mortality available to investigate the hazard of dying. We construct our vital status variable as a dummy variable coded as 1 if the individual died during in the next wave of the follow-up period and 0 otherwise. In the HRS cohort, of 9814 respondents whose actual mortality status is known, 1422 (14%) were deceased by wave 8. In the AHEAD cohort, of 7575 respondents whose actual mortality status is known, 3884 (50 %) were deceased by wave 8.

⁶ In addition, HRS matches the respondent's records to the National Death Index for those who are reported as deceased or of unknown vital status during tracking.

5.2.4 Control variables

The HRS collects extensive information about health, economic status, work and family relationships. In our model explaining mortality as a function of SSE, we include several other explanatory variables. We control for health status using both subjective and more objective measures. We construct four binary indicators of self-assessed health (SAH) based on the response to the question, “Would you say your health is: excellent, very good, good, fair or poor?”. In addition, we construct binary indicators of whether individual’s SAH improved or worsened by three categories, two categories or one category or remained the same, based on the difference between current and lagged SAH. For physical health status, we use indicators of eight chronic conditions, namely: high blood pressure, diabetes, cancer, lung disease, heart disease, stroke, arthritis and psychiatric illness. In order to control for physical health changes, we also use information on the onset of (newly diagnosed) chronic diseases. Respondents’ depressive symptoms are measured with a subset of the 20-item Center for Epidemiologic Studies Depression Scale (CES-D) score. Finally, we control for the number of doctor visits and whether the individual was hospitalized or not. We further generate three ADL - IADL indices following the suggestions in Wallace and Herzog (1995) and only including questions asked consistently across waves: mobility, use of large muscles, and IADLs. Functional limitation is measured as a binary indicator for whether the individual has any health problems which limit his ability to work. We also include in our analysis information on individuals’ demographic characteristics, such as measures of age, gender, marital status, race, education, income, wealth and employment status (employed, unemployed, retired, disabled). Education is measured by the number of years during which the individual received formal education. Marital status takes the value one if the individual is married, and 0 otherwise. Additionally, parents’ mortality information has been collected in the HRS. We include binary indicators of whether the individual’s mother and father are deceased, the age of death of parents and an interaction term between the death indicator and age of death. Table 1 in the Appendix presents the descriptive statistics of the variables used in the analyses.

For the expectation formation model, we generate variables indicating new events between waves: onset of health conditions (high blood pressure, diabetes, cancer, lung disease, heart

disease, stroke, arthritis and psychiatric illness. health limitation), new divorce, newly widowed, changes in the ADL indices and doctor visits and new death of parents (interacted with the age of death of the parents).

5.3 Mortality Prediction

5.3.1 Model for Mortality

In order to assess the predictive power of subjective survival expectations, we model the transition to mortality as a function of SSE_{diff} and various covariates, using a discrete time hazard specification. As we observe the period during which the individual died, we calculate the age of death and model this using a discrete time duration model. The covariates include levels of and changes in health status and various socioeconomic characteristics, all defined in section 2.

We use data for a sample of all individuals who are alive in wave 1 ($t = \tau$, in the notation followed by Jenkins, 1995). At the end of the time period for which we have data, some people will still be alive (censored duration data, $\delta_i = 0$), and some will have died (complete duration data, $\delta_i = 1$). Each respondent i lives s_i years from the interval between the start of the observation period and the occurrence of death (observed age of death is $\tau + s_i$, if $\delta_i = 1$), or the end of observation period (ie, $\tau + s_i$ represents age at the end of the period, if $\delta_i = 0$). The discrete time hazard rate is:

$$\rho_{it} = P [T_i = t \mid T_i \geq t ; x_{it}], \quad (5.1)$$

where x_{it} is a vector of covariates which may vary with time and T_i is a discrete random variable representing the time at which the end of the spell occurs (age of death).

We need to account for the fact that we only observe individuals who are alive at the beginning of our observation period. Jenkins (1995), shows that the probabilities of observing the event history of each individual throughout our observation period depend only on ρ_{it} , $t = \tau, \dots, \tau + s_i$, and so the previous history can be ignored. Modelling this kind of

event history data is simplified by defining: $y_{it} = 1$ if $t = \tau + s_i$ and $\delta_i = 1$; $y_{it} = 0$ otherwise (ie, y_{it} is an indicator of the occurrence of death at age t). For those who are alive at the end of follow up, $y_{it} = 0$ at all observed periods. For those who die during the observation period, $y_{it} = 0$ at all periods except that of death, after which the individual drops from the sample. Then the analysis simplifies to a standard form for analysis of a binary variable y_{it} where the unit of analysis is now the spell period, i.e. actual estimation is based on a simple binary model for the panel data set. The data set consists of multiple rows of observations for each individual with as many rows as periods at risk, i.e. it has a standard unbalanced panel data format (Jenkins, 1995).

To complete the specification of the model, an expression for the hazard rate is required. We specify a complementary log-log hazard rate which is the discrete-time counterpart of the hazard for an underlying continuous-time proportional hazards model (Prentice and Gloeckler, 1978; Jenkins, 1995):

$$\rho_{it} = 1 - \exp\{-\exp[SSE_{diff(it)} + H_{i,t-1}\beta + \Delta H_{it}\gamma + X_{it}\varphi + \theta(t) + v_i]\}, \quad (5.2)$$

where $\theta(t)$ is the log of the baseline hazard integrated over the interval $[t-1, t]$, H is a vector of variables characterizing health status, ΔH contains the respective changes and X is a vector of other observed individual characteristics. We complete the specification by modelling $\theta(t)$ as a piecewise-constant function, by using dummy variables for age groups to the set of regressors. This leads to a semi-parametric specification of the discrete-time duration model. Our mortality model accounts for normally distributed unobserved heterogeneity (v_i) that is uncorrelated with the explanatory variables.

We perform the analysis on the relationship between SSE_{diff} and mortality with cumulative entry of the objective health variables and SAH in order to assess the extent to which individuals process the information on their objective or subjective health into SSE. We estimate three main models for each group of respondents. We first include only respondents' age, socioeconomic characteristics (income, wealth, education employment status, gender, race and marital status) and two measures of cognition – recall and arithmetic ability scores. In this model, without controlling for health, SSE captures all

information the individual has on his/her health. We then add respondents' health status (prevalence and onset of chronic conditions, ADLs/ IADLs indices, health limitation), other health measures (hospitalizations, number of doctor visits), parental longevity and socio-demographic traits. In this model, the SSE only captures the private health information that is not reflected in health indicators included. Lastly, we control for SAH in addition to the other health measures we have used in the second specification. We estimate separate models for the AHEAD and HRS samples, and for men and for women in the HRS sample. Finally, we investigate this relationship for different cognition, education groups, and for groups based both on education and cognition.

5.3.2 Predictive power of SSE for mortality

Table 5.4 reports the coefficient estimates and standard errors for SSE_{diff} for males, females and the full sample for HRS Cohort and the full AHEAD cohort sample.

The coefficient on SSE_{diff} is negative and statistically different from zero for males, females and the full sample in the first specification. Individuals who report higher survival probabilities are less likely to die. In this specification, without any further control for health information, the SSE_{diff} variable captures variation in expectations deriving from variation in health. This therefore reflects an individual's ability to process health information into mortality expectations. Both males and females appear capable of processing health information into SSE_{diff} . SSE_{diff} is also predictive of mortality even after adjusting for cognition scores, parental longevity, a full set of health status and health dynamics indicators and socioeconomic characteristics.

Respondents with a higher recall score are significantly less likely to die, while arithmetic ability score does not predict mortality. Memory appears to be the more important cognitive dimension for mortality. Controlling for cognition scores does not make the effect of SSE_{diff} on mortality disappear. The effect of SSE becomes insignificant only after controlling for SAH. There are a couple of implications of this for our results. Insignificance of SSE once conditioning on SAH does not mean that individuals cannot predict mortality. Our first specification shows that they can, while the second and third specifications are suggestive of the health information that is used in that prediction.

Table 5.4: Estimates of extent to which SSEdiff predicts mortality - HRS and AHEAD cohort

Other Controls		HRS cohort (Waves '96-'06)			AHEAD cohort (Waves '96- '06)
		Males	Females	Full sample	Full sample
Socioeconomic controls & Cognition	<i>SSE_{diff}</i>	-0.0119*** (0.0024)	- 0.0125*** (0.003)	-0.0120*** (0.0018)	-0.0024 (0.0015)
Socioeconomic controls & Cognition+ Health Conditions+ Parental longevity	<i>SSE_{diff}</i>	-0.0057** (0.0024)	-0.0054* (0.0029)	-0.0057*** (0.0019)	0.0000 (0.0015)
Socioeconomic controls & Cognition +Health Conditions + Parental longevity + SAH	<i>SSE_{diff}</i>	-0.0014 (0.0023)	-0.0022 (0.0029)	-0.0018 (0.0018)	0.0015 (0.0015)
	<i>Recall score</i>	-0.1318** (0.0675)	- 0.2251*** (0.0838)	-0.1787*** (0.0538)	-0.0819*** (0.0144)
	<i>Arithmetic Ability Score</i>	-0.0039 (0.0403)	-0.0228 (0.0480)	-0.0056 (0.031)	-0.0222 (0.0266)
	<i>V. Good SAH</i>	0.6255** (0.2649)	0.7775** (0.3790)	0.6609*** (0.2198)	0.2804 (0.1847)
	<i>Good SAH</i>	1.0856*** (0.2867)	1.2866*** (0.4036)	1.1630*** (0.2361)	0.5955*** (0.1923)
	<i>Fair SAH</i>	1.5626*** (0.3283)	1.8263*** (0.4676)	1.6676*** (0.2766)	1.0481*** (0.2162)
	<i>Poor SAH</i>	2.4025*** (0.3904)	2.4313*** (0.5601)	2.4178*** (0.3438)	1.2698*** (0.2640)
			N= 10658	N= 13526	N= 24184

Notes: 1. *, **, *** refers to significance at 10%, 5% and 1% respectively. 2.All models are estimated with panel cloglog. 3.Health Conditions include: high blood pressure, diabetes, cancer, lung disease, heart disease, stroke ,arthritis and psychiatric illness. Onset of (newly diagnosed) chronic diseases, Depression Scale (CES-D) score, number of doctor visits and hospitalization indicator. 4.Changes in SAH are also included over SAH levels. Reference category for SAH is excellent SAH. 5.Socioeconomic Controls: Male, Education, Work Status, Marital Status, Income, wealth. 6. Standard errors in parenthesis.

Cognition recall scores are predictive of mortality for the much older AHEAD cohort, while *SSE_{diff}* is not. The latter result is to be expected given that older individuals tend to severely overstate their survival chances and they have much lower cognition scores than the HRS cohort. This result holds even in the specifications that do not control for any health information. Therefore, AHEAD cohort is not considered in the remaining parts of our paper.

5.3.3 Heterogeneity of predictive validity of SSE by cognition and education

In this section, we look at the heterogeneity in the effect of SSE on mortality for cognition groups (defined in terms of both total recall score and arithmetic ability score) and education groups in the HRS cohort. As argued in the introduction, cognitive skills and education may impact the formation of survival expectations and therefore SSE may be less valid for people who are in the lower rather than the higher cognitive or educated groups.

SSE effects by cognition

Table 5.5 and Table 5.6 confirm that SSE_{diff} is only predictive of mortality for the lower recall group when no other health information is in the model. The effect of SSE is also much smaller than for the other two higher cognition groups. In the last two specifications, in which we control for health information, SSE_{diff} is not predictive of mortality for the lower recall group. For the higher recall group, SSE_{diff} is still significant when we control for other health information, but not when we control for SAH. If the low cognition group does not incorporate health information in survival expectations well, then we would expect that controlling for health has a lower impact for the low cognition group. The absence of any marked variation by cognition in the predictive power of SSE_{diff} after controlling for health does not tell us anything about differential ability to use information on health. On the other hand, comparing the results from the first specification for different cognition groups shows that the lower recall group does not incorporate health information in survival expectations well.

The recall score represents the cognitive dimension that indicates dementia and is much lower for the lowest cognition group. Recall score is only predictive of mortality (the higher the score, the lower the hazard of dying) for the low cognition group in all specifications, not so for the high cognition group. This shows that the impact of recall score on mortality is non-linear.

When we look at Table 5.6, for the lower arithmetic ability group, SSE_{diff} does not predict mortality in any of the three specifications, while for the middle and higher arithmetic ability groups it is at least as predictive as the “corresponding” recall groups. In the high arithmetic ability group, SSE_{diff} predicts mortality even after controlling for SAH. In sum, stronger differences in predictive ability of SSE_{diff} are found by arithmetic ability score than recall score.

Table 5.5: Predictive ability of SSE for mortality by cognition groups for *recall score* (Waves ‘96-‘06).

Controls		Low Cognition	Changing Cognition	High Cognition
Socioeconomic controls & Cognition	SSE_{diff}	-0.0067*** (0.0025)	-0.0181*** (0.0035)	-0.0173*** (0.0042)
Socioeconomic controls & Cognition+ Health Conditions+ Parental longevity	SSE_{diff}	-0.0033 (0.0025)	-0.0081*** (0.0031)	-0.0095** (0.0041)
Socioeconomic controls & Cognition + Health Conditions+ Parental Longevity+ SAH	SSE_{diff}	-0.0014 (0.0025)	-0.0022 (0.0032)	-0.0028 (0.0043)
	<i>Recall score</i>	-0.2921*** (0.0907)	0.0574 (0.0978)	0.1816 (0.1409)
	<i>Arithmetic Ability Score</i>	0.0026 (0.0386)	0.0593 (0.0619)	-0.1451* (0.0857)
	<i>V. Good SAH</i>	0.7411** (0.3702)	0.6124* (0.3397)	0.6407 (0.4064)
	<i>Good SAH</i>	1.1468*** (0.3883)	1.1662*** (0.3634)	1.0784** (0.4669)
	<i>Fair SAH</i>	1.4364*** (0.4242)	1.7324*** (0.4315)	1.9200*** (0.5724)
	<i>Poor SAH</i>	1.8573*** 0.4899	2.3453*** (0.5245)	3.4341*** (0.7391)
		N= 8064	N= 8514	N= 7606

(Notes: can be found in Table 5.4)

In this analysis, the predictive ability of SSE_{diff} in models without controls for health shows the ability to process information on health into survival expectations. We can see that this ability varies by cognition; the ability is higher in the highest cognition groups and low cognition group are less able to use that information. However, it is not clear if this indicates that low cognition cannot predict their mortality or that they cannot express expectations in terms of probabilities. Our models looking at the heterogeneous effects include dummies representing the focal responses and missing values for SSE and replacing each type of response by the mean value of SSE_{diff} . Doing so only slightly

improves the predictive ability of SSE_{diff} in the low cognition group. This is some evidence that low predictive ability of SSE for the lower cognition group is not very much dependent on the focal point 50 percent responses or non-response.⁷

Table 5.6: Predictive ability of SSE for mortality by cognition groups for *arithmetic ability score* (Waves '96-'06)

Controls		Low Cognition	Changing Cognition	High Cognition
Socioeconomic controls & Cognition	SSE_{diff}	-0.002 (0.004)	-0.019*** (0.004)	-0.017*** (0.003)
Socioeconomic controls & Cognition+ Health Conditions+ Parental longevity	SSE_{diff}	0.002 (0.004)	-0.010*** (0.004)	-0.010*** (0.003)
Socioeconomic controls & Cognition + Health Conditions+ Parental Longevity+ SAH	SSE_{diff}	0.004 (0.004)	-0.005 (0.004)	-0.007*** (0.003)
	<i>Recall score</i>	-0.180* (0.101)	-0.175* (0.098)	-0.120 (0.093)
	<i>Arithmetic Ability Score</i>	0.004 (0.048)	0.118* (0.070)	0.549*** (0.214)
	<i>V. Good SAH</i>	0.414 (0.510)	0.539 (0.384)	0.715** (0.333)
	<i>Good SAH</i>	1.271** (0.530)	1.325*** (0.417)	0.701* (0.378)
	<i>Fair SAH</i>	1.528** (0.600)	1.874*** (0.507)	1.331*** (0.447)
	<i>Poor SAH</i>	2.016*** (0.713)	3.095*** (0.661)	1.899*** (0.570)
		N= 4758	N= 6809	N= 12617

(Notes: can be found in Table 5.4)

SSE effects by education

Table 5.7 shows that, when controlling only for socioeconomic information, SSE_{diff} has a significant effect on mortality in all education groups except the lowest. The consistent association between survival expectations and mortality persists through all three of the specifications only for the highest educated group (college and above graduates). In all

⁷ Full results with focal response and non-response dummies are available upon request. The coefficient for the focal response is negative and significant only in the low cognition group, meaning that people who respond 50 percent is less likely to die, whereas people who report missing values are more likely to die.

specifications, estimated coefficients of SSE_{diff} increase with education level. SSE_{diff} does not predict mortality in the lowest education group in any specification.⁸

Table 5.7: Predictive ability of SSE for mortality by education (Waves '96-'06)

Controls		High school drop out + GED	High school grad.	Some college	College
Socioeconomic controls & Cognition	SSE_{diff}	-0.005 (0.003)	-0.0095*** (0.0030)	-0.0230*** (0.0055)	-0.0228*** (0.0042)
Socioeconomic controls & Cognition+ Health Conditions+ Parental longevity	SSE_{diff}	0.0001 (0.0036)	-0.0036 (0.0030)	-0.0102*** (0.0038)	-0.0157*** (0.0045)
Socioeconomic controls & Cognition + Health Conditions+ Parental Longevity+ SAH	SSE_{diff}	0.001 (0.004)	0.0001 (0.0030)	-0.0059 (0.0040)	-0.0108 ** (0.0047)
	<i>Recall score</i>	-0.223 ** (0.118)	-0.2088** (0.0841)	-0.1692 (0.1079)	-0.0592 (0.1235)
	<i>Arithmetic Ability Score</i>	0.077 (0.061)	-0.0681 (0.0451)	0.0232 (0.0769)	-0.0525 (0.0980)
	<i>V. Good SAH</i>	0.426 (0.569)	0.9552** (0.4210)	0.4452 (0.4109)	0.4316 (0.3919)
	<i>Good SAH</i>	0.696 (0.581)	1.4964*** (0.4430)	0.7732* (0.4504)	1.0335 ** (0.4384)
	<i>Fair SAH</i>	0.865 (0.636)	2.0294*** (0.4893)	1.0372*** (0.5445)	1.7408*** (0.5580)
	<i>Poor SAH</i>	1.312 * (0.636)	3.1089*** (0.5616)	1.5080*** (0.6629)	3.0383*** (0.7249)
		N= 5012	N= 8317	N= 5314	N= 5541

(Notes: can be found in Table 5.4)

⁸ We also checked whether non-focal responses (focal responses replaced by mean values of SSE and dummy for focal responses is included in models) predict mortality better for the lowest education group. Doing so did not improve the predictive ability of SSE for mortality in lower and other education groups. Results available upon request.

Table 5.8: Examination of differential effects of education by recall score cognition in the predictive ability of SSE (Waves '96-'06)

Controls		Low education & Low Cognition	Low education & High Cognition	High Education & Low Cognition	High Education & High Cognition
Socioeconomic controls & Cognition	SSE_{diff}	-0.0040 (0.0030)	-0.0120* (0.0067)	- (0.0070)	- (0.0061)
Socioeconomic controls & Cognition+ Health Conditions+ Parental longevity	SSE_{diff}	-0.0010 (0.0030)	-0.0048 (0.0070)	-0.0073 (0.0052)	-0.016** (0.007)
Socioeconomic controls & Cognition + Health Conditions+ Parental Longevity+ SAH	SSE_{diff}	0.0007 (0.0031)	-0.0011 (0.0080)	-0.0055 (0.0053)	-0.0072 (0.0056)
		N=5712	N=2945	N=2352	N=4661

(Notes: can be found in Table 5.4)

Table 5.9: Examination of differential effects of education by arithmetic ability score cognition in the predictive validity of SSE for mortality (Waves '96-'06)

Controls		Low education & Low Cognition	Low education & High Cognition	High Education & Low Cognition	High Education & High Cognition
Socioeconomic controls & Cognition	SSE_{diff}	-0.002 (0.004)	-0.009** (0.004)	-0.001 (0.014)	-0.023*** (0.005)
Socioeconomic controls & Cognition+ Health Conditions+ Parental longevity	SSE_{diff}	0.001 (0.004)	-0.007 (0.005)	0.002 (0.012)	-0.013*** (0.004)
Socioeconomic controls & Cognition + Health Conditions+ Parental Longevity+ SAH	SSE_{diff}	0.003 (0.004)	-0.006 (0.005)	-0.003 (0.013)	-0.007 (0.005)
		N=3681	N=5502	N=1011	N=7115

(Notes: can be found in Table 5.4)

SSE effects by education and cognition

Tables 5.8 and 5.9 present results broken down by both high/low education and high/low cognition. We can see that education has differential effects on the predictive ability of SSE_{diff} for mortality, depending on the cognitive domain. In Table 5.8, we see that SSE_{diff} predicts mortality for the groups of high educated & low/high cognition and for the low educated & high cognition group, but not for low educated & low cognition group when we do not control for health. At low education, the effect of SSE_{diff} increases in moving from low to high cognition but it only reaches margins of significance in the latter case. At high education, the effect is more significant and again increases in moving from low to high cognition. SSE_{diff} is always significant at high education irrespective of cognition. Also, the predictive effect is stronger at high education and low cognition than at low education and high cognition. These results suggest that education plays a more important role than cognition in building valid survival expectations. When we repeat this analysis for groups of education & arithmetic ability cognition groups, the highest effect of SSE_{diff} is found for the high education & high cognition group. Moving from low to high cognition in both low & high education groups increases the effect of SSE_{diff} strongly. However, the small sample size of the high educated & low cognition arithmetic ability score group limits the degree to which conclusions about the separate effects of education and arithmetic ability cognition can be derived from Table 5.9.

5.4 Survival Expectations Formation

In Section 3 we examined the degree to which subjective survival expectations act as good predictors of future mortality and whether there is heterogeneity in the predictive power by looking at sub-groups of respondents with respect to the level of education and cognition. Our results reveal that: i) SSE are less predictive for mortality for low educated (particularly) and low cognition groups and ii) education is more important than cognition in the predictive ability of SSE for mortality. In this section, we explore potential reasons for these findings by examining how expectations are formed. In particular, we will explore if expectations are updated with new information and how individuals integrate old and new information when updating their expectations. This is similar to Hurd and

McGarry (2002) and Smith et al. (2001) which were also interested in the update of expectations, but focusing on the general HRS sample or smokers in particular.

5.4.1 Model for SSE formation

In order to assess the effect of new information on longevity expectation formation, we estimate dynamic panel data models. A respondent's survival chances at time t are assumed to depend on his/her expectations at $t-1$ (old information) and the newly available information that arrived between period $t-1$ and t . Thus, the perceived longevity in wave t , is hypothesized to be a function of a respondent's initial longevity assessment in wave $t-1$, along with any new information that an individual receives that would motivate a revision in the longevity expectations. One should keep in mind that this specification does not allow testing for rational expectations, due to the informational requirements it imposes.⁹ A dynamic model is required and therefore, we estimate the following equation:

$$SSE_{diff(it)} = \beta SSE_{diff(i,t-1)} + \gamma \Delta Z_{it} + \theta W_t + \varepsilon_{it} \quad \text{with} \quad \varepsilon_{it} = v_i + u_{it} \quad (5.3)$$

ΔZ_{it} is the vector of onset of new events (health shocks, parental death), and W a vector of dummy variables representing the different waves of data to control for year fixed effects. The error term is divided into two parts, v_i is the individual fixed effect and u_{it} the observation specific error. By construction, $SSE_{diff(it-1)}$ is correlated with ε_{it} , which deems the OLS estimator inconsistent. The fixed-effects estimator is also inconsistent as the transformed lagged dependent variable is correlated with the transformed error term.

The first differences estimator is also inconsistent due to correlation between first differences of the error term and of the lagged dependent variable. We make use of the Blundell and Bond (1998) system GMM dynamic panel data estimator that uses a system of equations that instrument both levels (with differences) and differences (with levels). This is known as "system GMM". The data transformation for system GMM stacks the

⁹ Formal rational expectations test analyzes whether SSE at time t , are function of SSE at time $t-1$, conditional on the information set at time $t-1$, whereas we are interested in the arrival of new information, in particular changes in X_s .

first-differenced equation on top of the level equation; hence we have the following structure:

$$SSE_{diff(it)} = \beta SSE_{diff(i,t-1)} + \gamma \Delta Z_{it} + \theta W_t + \varepsilon_{it} \quad (5.4)$$

$$\Delta SSE_{diff(it)} = \beta \Delta SSE_{diff(i,t-1)} + \gamma \Delta(\Delta Z_{it}) + \theta \Delta W_t + \Delta \varepsilon_{it} \quad (5.5)$$

The system GMM estimator uses the levels equation (equation (5.4)) to obtain a system of two equations: one differenced and one in levels. By adding the second equation additional instruments can be obtained. In this system of equations, endogenous variables in the difference equation (lagged differences of SSE_{diff}) are instrumented with past lags of the levels, while the endogenous variables in the levels equation (lagged level of SSE_{diff}) are instrumented with past lags of the differences. Therefore, separate instrument sets must be specified for equations 5.4 and 5.5 in the system GMM approach. In case of no serial correlation of u_{it} , $SSE_{diff(i,t-2)}$, $SSE_{diff(i,t-3)}$, \dots , are valid instruments for $\Delta SSE_{diff(i,t-1)}$ in (5.4), and $\Delta SSE_{diff(i,t-1)}$ is a valid instrument for $SSE_{diff(it-1)}$ in (5.5). In, case u_{it} is MA(1), however, $SSE_{diff(i,t-2)}$ and $\Delta SSE_{diff(i,t-1)}$ are not valid instruments and further lags need to be considered. In order to determine the lags which can be used as instruments, we use the Arellano AR test of autocorrelation.¹⁰ Second, we test for the exogeneity of the instruments using Sargan-Hansen's test, which is robust to heteroskedasticity and autocorrelation, and which test statistic is asymptotically distributed as χ^2 with degrees of freedom equal to the number of restrictions.

We hypothesize that β and γ vary with education and cognitive functioning as people with different cognition and education levels may incorporate past information and new events into their current expectations differently. Therefore, we estimate Equation 5.3 by education and cognition groups.

¹⁰ We perform two specification tests for the validity of instruments. First, we test for the presence of first, second-order, (3rd and 4th order for the ones that we used more lags for instrumenting) autocorrelation in the differenced equation (but, note that first-order autocorrelation in the differenced equation is expected and does not signify an improper model specification). If there is autocorrelation in additional lags then our lagged instruments will be correlated with the contemporaneous error. Therefore, we run AR tests of various lags to determine which lags provide exogenous instruments.

5.4.2 Results for SSE formation and the role of new information

To examine the effect of new information on SSE, we estimate the effects of changes in all relevant factors on expectations using the system GMM estimator for our dynamic model. Any significant effect of the changes means that respondents change their SSE in response to unanticipated new events. There will be no significant effects if, on average, people are able to completely anticipate the new information or if they ignore the new information. Unfortunately, we cannot distinguish between the two interpretations. In the second part, we investigate how the formation of expectations varies with cognition and education.

Table 5.10 presents results for the full HRS sample, as well as for males and females separately. We find SSE_{diff} reported in the previous period always has a significant effect, and it is larger for males than females. Respondents therefore do indeed integrate their earlier expectation in their next period's expectation. The coefficient of the lagged survival probabilities is 0.803 for the full sample, 0.821 for males and 0.659 for females. This shows that controlling for new health events, on average, the SSE in this period is *only a fraction* of what it was in the last period (consistent with Sloan et al. (1999) and Smith et al. (2001)). However, the objective life table probabilities of surviving to a target age increase over time when getting closer to the target age. Therefore, one would expect that respondents update their survival probabilities upwards (not downwards) if they build their expectations according to the law of conditional probability. This implies that respondents either do not understand conditional probabilities or that they incorporate additional information about their survival prospects over and above that captured by the health indicators that causes them to revise their survival expectations downward. Another point worth mentioning here concerns the magnitude of the coefficient of the lagged dependent SSE. We find, after controlling for dynamics and unobserved heterogeneity, for the full sample and for males, a coefficient of around 0.8, whereas lagged dependency is found to be much lower in the previous studies (around 0.5 in Sloan et al (1999) and Smith et al. (2001)). Our result suggests that controlling for unobserved heterogeneity and dynamic nature of the expectations improves (improvement from 0.5 to 0.8) the modelling of longevity expectations. As it is shown by Blundell and Bond (1998), in dynamic models

with persistent series, Arellano and Bond (1991) and fixed effects estimators can seriously underestimate coefficient of lagged dependent variable, which is overcome by system GMM estimator.

Table 5.10: New information and Subjective Survival Expectations in HRS

Dependent Var: $SSE_{diff(t+1)}$	Full Sample		Males		Females	
	Coeff	Std. Err	Coeff	Std. Err	Coeff	Std. Err
$SSE_{diff(t)}$	0.803***	0.119	0.821***	0.168	0.659***	0.133
Recent death of dad	-30.936**	13.370	-50.302**	21.129	-11.240	13.353
Recent death of mom	-11.892	10.336	-19.335	14.921	-5.169	13.021
Mom age	0.041	0.026	0.042	0.031	0.071**	0.034
Dad age	0.017	0.021	0.022	0.035	0.031*	0.022
Death of dad x dad age	0.382**	0.154	0.600**	0.244	0.158	0.155
Death of mom x mom age	0.155	0.119	0.221	0.171	0.092	0.150
Newly widowed	0.561	1.370	1.024	2.761	0.283	1.445
Newly divorced	-1.070	1.749	-6.940***	2.864	1.984	2.047
<u>Onset of:</u>						
Health Limitation	-2.069***	0.741	-3.595***	1.101	-1.029	0.912
High Blood Press.	1.171	0.959	1.043	1.448	1.159	1.175
Diabetes	-0.476	1.351	-1.984	1.814	0.602	1.876
Cancer	-5.221***	1.448	-8.244***	1.881	-1.565	2.132
Lung disease	-4.901***	1.827	-6.709***	2.659	-3.550	2.291
Heart Disease	-1.929	1.201	-1.284	1.614	-2.774*	1.648
Stroke	-5.448**	2.170	-1.034	3.024	-9.051***	2.808
Psychiatric Disease	-4.130**	1.685	-7.344**	3.049	-2.709	1.844
Arthritis	-0.367	0.873	-0.996	1.272	0.138	1.100
Δ Doctor Vis.	-0.023*	0.013	0.009	0.018	-0.040**	0.018
Δ ADL Musc. Index.	-0.240	0.206	-0.176	0.318	-0.206	0.248
Δ ADL Mob.	-0.531**	0.240	-0.753*	0.402	-0.411*	0.274
	N= 24184		N= 10658		N= 13526	
Hansen test of overiden.	Pr>chi2= 0.527		Pr>chi2= 0.917		Pr>chi2= 0.369	
Test for Autocorrelation						
AR (1)	Pr>z= 0.000		Pr>z= 0.000		Pr>z= 0.000	
AR(2)	Pr>z= 0.000		Pr>z= 0.000		Pr>z= 0.000	
AR(3)	Pr>z= 0.070		Pr>z=0.0714		Pr>z=0.035	

Notes: 1. *, **, *** refers to significance at 10%, 5% and 1% respectively. 2. Standard errors in parenthesis. 3. To determine the validity of instruments, we conduct two tests. AR test (for autocorrelation in the error term) fail to reject the null at lag 3 at 5% level, therefore we start instrumenting at this lag. To determine if our instruments are exogenous, we calculate Hansen's J statistic. We fail to reject the null hypothesis that instruments are exogenous, therefore over-identifying restrictions are valid (except from females where result from AR(3) test does not pass the autocorrelation test). 4. We control for age and age square in the estimation.

In the full HRS sample, we find that individuals modify their SSE in response to some changes. Variables with a significant effect are assumed to capture what has not been anticipated by individuals. To control for parental mortality, we add an interaction term between recent (newly occurred between waves) death of parents (father and mother

separately) and age of death of parents. This allows us to control for the impact of a death depending on whether it occurs at an old or younger age. Changes that significantly affect survival expectations are new health conditions (cancer, lung disease, stroke, health limitation and psychiatric diseases), changes in the mobility index and doctor visits and the recent death of father. For the male sample, we observe that those who have been newly diagnosed with cancer, lung disease or psychiatric disease, or got divorced have lower SSE. For females, health shocks that affect SSE in the next period include a new stroke and onset of heart disease. A recent death of the father significantly affects the SSE in the next period but the direction of the effect depends on the age of death of father; if the father died at an advanced age then respondents update their SSE upwards, while and if the dad died at a young ages then respondents revise their SSE downwards. We found no significant effect of a recent death of the mother. We therefore do not find any gender symmetry for parental longevity such as daughters paying more attention to longevity of their mother and sons to their fathers.

Heterogeneity by cognition

Results in Table 5.11 and 5.12 assess whether cognitively impaired respondents differ from the cognitively better able in how new information affects their survival expectations. In both the recall and arithmetic ability domain, lower cognition individuals seem to modify their expectations according to fewer events than the cognitively more able. For instance, their expectations only react to onset of health limitation for the low recall score cognition group and to lung diseases and changes in the number of doctor visits for the low arithmetic ability group. This implies that new information about many other health events does not seem to prompt them to update their survival expectations. For higher cognitive ability individuals (according to both scores), more new health events like cancer, lung diseases, stroke and psychiatric diseases are a reason to update their SSE.

Lagged survival expectations have a significant effect on current survival expectations in each of the groups. In this case, we find that the coefficient on lagged SSE for low recall is larger than high recall (although this is not the case for arithmetic ability groups). One possible interpretation is that the low cognition group does not use the new information, but more use the past information.

Table 5.11: Differences in the role of new information for SSE by cognition groups using Total Recall Score.

Dependent Var: $SSE_{diff(t+1)}$	Low cognition		Changing cognition		High cognition	
	Coeff	Std. Err	Coeff	Std. Err	Coeff	Std. Err
$SSE_{diff(t)}$	0.661***	0.208	0.787***	0.121	0.467***	0.166
Recent death of dad	-33.895	23.310	-17.990	20.262	-20.719	13.537
Recent death of mom	-33.168	20.554	-2.370	16.715	5.976	11.835
Mom age	0.044	0.041	0.066**	0.029	0.099**	0.041
Dad age	0.034	0.032	0.005	0.025	0.084**	0.036
Death of dad x dad age	0.433	0.270	0.237	0.236	0.244	0.156
Death of mom x mom age	0.399	0.240	0.041	0.190	-0.041	0.135
Newly widowed	0.371	2.226	0.851	2.135	-0.101	1.888
Newly divorced	-4.125	2.602	5.935**	2.660	-2.914	2.897
Onset of:						
Health limitation	-3.270***	1.187	-1.132	1.184	-1.842*	1.052
High blood press.	1.319	1.666	0.550	1.544	1.148	1.274
Diabetes	0.517	2.100	-3.152	2.277	0.381	1.829
Cancer	-4.206*	2.516	-5.761**	2.364	-5.406***	1.832
Lung disease	-3.362	2.956	-3.122	2.751	-8.573***	2.789
Heart disease	-0.542	2.117	-3.324	1.988	-1.901	1.600
Stroke	-1.055	3.404	-5.937	3.712	-11.068***	3.067
Psychiatric disease	0.184	2.470	-10.723***	2.852	-4.545*	2.645
Arthritis	-1.116	1.575	-0.271	1.389	-0.178	1.102
Δ Doctor Vis.	-0.010	0.023	-0.033**	0.015	-0.024	0.023
Δ ADL Musc. Index.	-0.299	0.330	-0.621*	0.332	0.283	0.281
Δ ADL Mob.	-0.606	0.365	-0.520	0.379	-0.216	0.372
	N= 8064		N= 8514		N= 7606	
Hansen test of overiden.	Pr>chi2= 0.828		Pr>chi2= 0.912		Pr>chi2= 0.024	
Test for Autocorrelation						
AR (1)	Pr>z= 0.000		Pr>z= 0.000		Pr>z= 0.000	
AR(2)	Pr>z= 0.002		Pr>z= 0.000		Pr>z= 0.003	
AR(3)	Pr>z= 0.393		Pr>z=0.413		Pr>z=0.090	

Notes: 1. *, **, *** refers to significance at 10%, 5% and 1% respectively. 2. Standard errors in parenthesis. 3. To determine the validity of instruments, we conduct two tests. AR test (for autocorrelation in the error term) fail to reject at lag 3 at 5% level, therefore we start instrumenting at this lag. To determine if our instruments are exogenous, we calculate Hansen's J statistic. We fail to reject the null hypothesis that instruments are exogenous, therefore over-identifying restrictions are valid (except from high cognition where result from Hansen test does not pass the test). 4. We control for age and age square in the estimation.

Table 5.12: Differences in the role of new information for SSE by cognition groups using Arithmetic Ability Score.

Dependent Var: $SSE_{diff(t+1)}$	Low cognition		Changing cognition		High cognition	
	Coeff	Std. Err	Coeff	Std. Err	Coeff	Std. Err
$SSE_{diff(t)}$	0.299***	0.148	0.835***	0.162	0.556***	0.121
Recent death of dad	-98.846***	27.793	7.715	21.561	-8.342	10.751
Recent death of mom	-4.244	19.461	-43.751*	22.595	6.020	10.692
Mom age	0.108***	0.036	0.046	0.034	0.097***	0.031
Dad age	0.049	0.033	0.018	0.042	0.060***	0.024
Death of dad x dad age	1.192***	0.324	-0.049	0.254	0.107	0.124
Death of mom x mom age	0.050	0.228	0.506*	0.259	-0.040	0.122
Newly widowed	1.934	2.373	2.740	2.455	-1.934	1.701
Newly divorced	-1.269	2.782	-0.668	3.170	-3.323	2.417
Onset of:						
Health limitation	-0.649	1.378	-2.197	1.495	-2.995***	0.841
High blood press.	-0.738	2.008	-0.630	1.870	2.570**	1.085
Diabetes	-2.485	2.520	1.266	2.728	-0.774	1.568
Cancer	2.559	2.894	-4.837	3.171	-7.240***	1.631
Lung disease	-9.152**	3.577	1.458	3.305	-7.305***	2.250
Heart disease	0.025	2.445	-2.933	2.403	-1.748	1.268
Stroke	-6.817	4.048	-5.536	3.502	-5.830**	2.748
Psychiatric disease	1.089	2.339	-5.091	3.140	-5.945**	2.441
Arthritis	-0.230	2.000	-1.590	1.682	-0.104	0.924
Δ Doctor Vis.	-0.054**	0.025	0.013	0.016	-0.033	0.021
Δ ADL Musc. Index.	-0.194	0.394	-0.770*	0.401	0.016	0.224
Δ ADL Mob.	-0.269	0.411	-1.025	0.464	-0.506*	0.279
	N= 4758		N= 6809		N= 12617	
Hansen test of overiden.	Pr>chi2= 0.562		Pr>chi2= 0.225		Pr>chi2= 0.035	
Test for Autocorrelation						
AR (1)	Pr>z= 0.000		Pr>z= 0.000		Pr>z= 0.000	
AR(2)	Pr>z= 0.071		Pr>z= 0.000		Pr>z= 0.000	
AR(3)	Pr>z= 0.655		Pr>z=0.039		Pr>z=0.286	

Notes: 1. *, **, *** refers to significance at 10%, 5% and 1% respectively. 2. Standard errors in parenthesis. 3.. To determine the validity of instruments, we conduct two tests. AR test (for autocorrelation in the error term) fail to reject at lag 3 at 5% level, therefore we start instrumenting at this lag. To determine if our instruments are exogenous, we calculate Hansen's J statistic. We fail to reject the null hypothesis that instruments are exogenous, therefore over-identifying restrictions are valid (AR(3) test does not pass for changing cognition and Hansen test for high cognition show over-identifying restrictions are not valid) . 4. We control for age and age square in the estimation.

Table 5.13: Differences in the role of new information for SSE by education groups

Dependent Var: $SSE_{diff(t+1)}$	High school drop out + GED		High school grad.		Some college		College	
	Coeff.	Std. Err	Coeff.	Std. Err	Coeff.	Std. Err	Coeff.	Std. Err
$SSE_{diff(t)}$	0.222	0.155	0.782***	0.174	0.628***	0.151	0.702***	0.155
Recent death of dad	-51.263	32.169	-26.987*	16.294	-37.023*	29.475	2.434	13.655
Recent death of mom	-22.053	24.973	27.505*	16.675	-32.621*	20.238	-9.702	13.305
Mom age	0.096***	0.033	0.060	0.034	0.051	0.043	0.046*	0.034
Dad age	0.055	0.034	0.019	0.033	0.039	0.029	0.027	0.037
Death of dad x dad age	0.586	0.372	0.352*	0.192	0.443*	0.335	0.030	0.159
Death of mom x mom age	0.255	0.288	-0.287	0.191	0.393*	0.235	0.138	0.151
Newly widowed	0.326	2.036	1.109	2.008	-0.094	2.732	-2.952	2.393
Newly divorced	-0.921	2.432	-2.046	3.400	0.107	2.931	0.516	3.403
Onset of:								
Health limitation	-0.241	1.304	-2.477**	1.121	-2.419*	1.240	-4.599***	1.296
High blood press.	-0.922	1.797	0.532	1.548	2.993*	1.619	1.781	1.495
Diabetes	-0.510	2.264	-0.158	2.256	1.029	2.213	-2.532	2.230
Cancer	1.087	2.827	-7.658***	2.422	-4.149*	2.646	-8.104***	2.058
Lung disease	-2.105	2.526	-10.237***	3.164	-3.434	3.510	-6.972*	3.866
Heart disease	-1.030	2.208	-2.927	2.016	-4.309*	2.263	-0.177	1.930
Stroke	-0.312	3.767	-11.155***	3.328	-2.237	3.573	-9.604**	4.611
Psychiatric disease	-5.675**	2.639	-2.821	2.660	-5.691**	2.849	-2.555	2.924
Arthritis	0.108	1.773	-1.329	1.463	1.151	1.475	-1.161	1.299

Table 5.13: Continued

Δ Doctor Vis.	-0.020	0.021	-0.004	0.023	-0.038	0.027	-0.006	0.020
Δ ADL Musc. Index.	-0.850**	0.365	-0.012	0.306	0.268	0.376	0.301	0.362
Δ ADL Mob.	-0.094	0.357	-0.672**	0.350	-0.774	0.475	-0.483	0.496
	N= 5012		N= 8317		N= 5314		N=5541	
Hansen test of overiden.	Pr>chi2= 0.064		Pr>chi2= 0.158		Pr>chi2= 0.775		Pr>chi2= 0.079	
Test for Autocorrelation								
AR (1)	Pr>z= 0.000		Pr>z= 0.000		Pr>z= 0.000		Pr>z= 0.000	
AR(2)	Pr>z= 0.064		Pr>z= 0.003		Pr>z= 0.000		Pr>z= 0.000	
AR(3)	Pr>z= 0.020		Pr>z=0.509		Pr>z=0.291		Pr>z=0.295	

Notes: 1. *, **, *** refers to significance at 10%, 5% and 1% respectively. 2. Standard errors in parenthesis. 3. To determine the validity of instruments, we conduct two tests. AR test (for autocorrelation in the error term) fail to reject at lag 3 at 5% level, therefore we start instrumenting at this lag. To determine if our instruments are exogenous, we calculate Hansen's J statistic. We fail to reject the null hypothesis that instruments are exogenous, therefore over-identifying restrictions are valid (AR(3) test does not pass for the lower education). 4. We control for age and age square in the estimation.

Heterogeneity by education

Separate results for education groups in Table 5.13 suggest that the updating process is different for the four education groups. The SSE is strongly driven by the onset of new health events like a health limitation, cancer, stroke and lung diseases among the higher education groups but this is not the case for the lowest education group (high school drop out and GED). The only two significant new health events for this group are the onset of lung disease and changes in the mobility index. More interestingly, we do not observe a significant effect of the lagged SSE on the next period's SSE for this group. This suggests that lower educated do not take their previous period's survival probabilities into account while forming their current expectations and that there is more noise in their responses.

5.5 Conclusions

Using eight waves of the HRS, we confirm the findings of earlier studies based on a shorter panel that, on average, individuals' reported survival expectations predict their actual longevity (Hurd and McGarry, 2002, Perozek 2008). But we find that this predictive ability varies with age, education and cognition. For the much older AHEAD cohort, survival expectations do not predict actual mortality. We find a stronger association of subjective survival expectations with mortality for the group of individuals who have higher cognitive functioning and higher education. Subjective survival expectations for low educated and low cognition groups are not predictive of mortality. Education appears to be more important in building accurate survival expectations than cognitive functioning.

This substantial heterogeneity in the predictive ability of subjective survival expectations across cognition and education groups may be either due to mistakes that are made while forming expectations or, once formed, low ability to express them as a probability. It is important to discriminate between these two explanations, as forming inaccurate survival expectations can have serious consequences for decisions concerning later life. To this end, we investigate further the ability of different groups of individuals to form and update their survival expectations. In particular, we look at the extent to which individuals update expectations with new information, since this directly tells us about the ability to revise survival expectations with new events. Using dynamic panel data models which allow for state dependence of survival expectations, we find that, apart from ageing, individual expectations about survival are also influenced by recent death of the parents, onset of new diseases such as cancer, stroke and psychiatric diseases. We also observe differences between males and females in forming their expectations. For instance, males modify their longevity expectations after a divorce, whereas females do not.

Our results indicate that individuals in the lower cognition/education groups modify their expectations in response to a considerably smaller number of new events than individuals in higher cognition/education groups. High school drop-outs and individuals with low cognitive functioning generally do not appear to revise their survival expectations downward when they contract a new disease or health condition, while the higher educated and cognitively more able do. These results also partly explain why education and

cognitive ability is related to the predictive power of subjective survival expectations for mortality as those in higher cognition and education groups are better able to revise their expectations after new health events and integrate old and new information when updating their expectations.

Our analysis shows that subjective survival expectations contain valid and important information over and above self-assessed health and more objective health indicators. This subjective information about longevity seems however more relevant for some individuals such as higher educated and cognitively able, than for others. Mistakes made in forming survival expectations have important consequences for future decisions such as retirement and savings. For instance, the recent policy reforms on retirement and pensions imply that future pensions will be less generous in terms of pension levels and/or eligibility ages. Therefore, mistaken survival expectations are important in the context of these reforms that place more responsibility on individual choice of pension or savings plans. For example, our results show that low education and cognition reduce the likelihood of revising survival expectations downward when objective risks such as cancer or stroke emerge. This suggests that low education/cognition individuals may be less likely to change behaviour and seek appropriate medical treatment in response to conditions that pose a serious threat to their longevity.

The existing procedures to recover individuals' preferences from observed choices are typically based on the rational expectations assumption. It is difficult to accept that everyone is able to form rational expectations or that everyone acts as if they have rational beliefs. In reality, individuals' subjective longevity expectations therefore might be biased, and in addition may not be fully captured by the subjective data only. Assuming rational expectations can lead to model misspecification and erroneous conclusions about individual preferences, which may have important implications for the analysis of economic decision making processes and public policy outcomes. Therefore, there is still a lot of scope for future research on subjective probabilities considering the heterogeneity of respondents and on the formation of these expectations.

Appendix

Table 5.14: Descriptive statistics of the HRS sample used in the analysis.

Variable	Mean	St. Dev
SSE_{diff}	-5.400	28.281
Previous SAH excellent	0.192	0.394
Previous SAH very good	0.338	0.473
Previous SAH good	0.294	0.456
Previous SAH fair	0.131	0.337
Previous SAH poor	0.045	0.207
Computed SAH change better_3	0.003	0.057
Computed SAH change better_2	0.026	0.158
Computed SAH change better_1	0.170	0.376
Computed SAH change same	0.026	0.158
Computed SAH change worse_1	0.539	0.498
Computed SAH change worse_2	0.213	0.410
Computed SAH change worse_3	0.041	0.199
High blood pressure	0.428	0.495
Diabetes	0.127	0.333
Cancer	0.082	0.275
Lung Diseases	0.077	0.266
Arthritis	0.484	0.500
Heart Diseases	0.151	0.364
Stroke	0.030	0.170
Psychiatric Diseases	0.085	0.279
Onset of High blood pressure	0.048	0.214
Onset of Diabetes	0.023	0.151
Onset of Cancer	0.020	0.138
Onset of Lung Diseases	0.013	0.113
Onset of Arthritis	0.054	0.225
Onset of Heart Diseases	0.027	0.162
Onset of Stroke	0.009	0.093
Onset of Psychiatric Diseases	0.015	0.121
CESD depression score	0.916	1.608
ADL Mobility Index	0.704	1.151
ADL Muscle Index	1.028	1.239
IADL Index	0.122	0.417
Health limitation	0.198	0.399
Number of doctor visits	7.300	13.097
Hospitalization	0.165	0.371
Male	0.447	0.497
Age	62.883	5.038
Lnwealth	11.904	1.725
Lnincome	10.585	0.980
Married	0.750	0.433
Education years	12.765	2.922
Unemployed	0.094	0.292
Employed	0.386	0.487
Retired	0.498	0.500
Disabled	0.022	0.145
White	0.852	0.355
Daddied	0.923	0.267
Momdied	0.744	0.436
Momless75	0.357	0.479
Mom7685	0.381	0.486
Dadless75	0.554	0.497
Dad7685	0.288	0.453

Chapter 6

Conclusions

The research that I report in the four chapters that make up the main body of this thesis aims to contribute to the area of the Economics of Health. The first two chapters are concerned with the relationship between health and labor market dynamics. The last two deal with health measurement and differences in health perceptions. In this final chapter I summarize my main findings and present my conclusions from the four studies.

In the first two chapters, I analyse the effects of health, financial incentives and their interaction on the propensity to leave the labour market. In chapter 1, I study the transitions of older individuals using the Spanish sample of ECHP (European Community Household Panel). The analysis is guided by the Option Value Model (Stock and Wise, 1990), which provides a useful framework for the discussion of the influence of health shocks on the effects of popular measures of financial incentives - such as pension wealth and peak value - on the likelihood of remaining at work. In this chapter, I postulate a hypothesis about such influence, which I term the *subordination hypothesis*, stating that the observed effects of these financial incentives on the propensity to remain at work depend on the health status of the individual. This is because health shocks are bound to affect the determination of these individual incentives through changes in key parameters such as the marginal disutility of labour, life expectancy or the discount rate in a way that cannot be observed by the researcher.

The reasoning sustaining the *subordination hypothesis* suggests that the net effect of a health shock, *ceteris paribus*, on pension wealth and peak value is indeterminate and therefore we cannot expect *a priori* the effects of pension wealth or peak value (on the probability of remaining at work) to change in a particular direction after a health shock. However, I argue that when shocks modify the chances to remain at work through the two

incentive measures in the same direction, it is *a priori* more likely that they increase the odds of exiting work, because pension wealth increases (and therefore the chances of remaining at work decrease) and peak value decreases (and therefore the chances of remaining at work also decrease). These insights have important policy implications, for they suggest that changes in pension rules – such as modifications in pension wealth or peak value - designed to encourage workers to remain in the labour market might be less effective among workers in poor health.

I test the *subordination hypothesis* by including interactions between health shocks and empirical measures of pension wealth and peak value in a discrete time duration model for age at retirement. My estimates from the Spanish sample of the ECHP provide some support for the hypothesis. While the elasticity of retirement to pension wealth changes modestly with health shocks, the latter appear to dampen the effect of peak value on the probability to remain at work. These findings are in line with those of Banks et al. (2007) who use the detailed information on pension incentives available in the English Longitudinal Study of Ageing (ELSA) to estimate their effects on retirement decisions. Indeed, they find that the (negative) impact of peak value on the probability of retirement is significant only for those without health problems.

The findings reported in Chapter 1 therefore highlight an important point: marginal changes to the financial incentives in pension systems will achieve little in terms of keeping older workers at work if these workers are unresponsive to financial incentives because of health problems. Therefore, such policies need to be considered in conjunction with appropriate health policies. Likewise, improving working conditions at young ages is likely to contribute to keeping workers in the labor force longer in good health.

The research reported in Chapter 2 deals with related issues. First, I provide further evidence on the *subordination hypothesis* using rich administrative data from a Dutch health care sector pension fund, which I link to the Dutch hospitalization database. Secondly, I postulate an *affordability hypothesis* stating that, after sudden adverse health events, individuals with higher pension wealth are better able to “afford” to withdraw from the labor market. The use of rich and large datasets allows me to investigate these two hypotheses in the domains of early retirement and disability insurance exits. Also, in contrast to much previous research on labour market dynamics of older workers, which has

focused mainly on men, I investigate these hypotheses separately for males and females. As ever more women enter the labor force, it is important to investigate their potentially different behavior regarding their retirement decisions.

Even after controlling for variables such as tenure in the sector, part-time work, number of children, household composition and marital status, I find that women are more responsive to pension incentives than men. This is in line with the results for the Italian labour market reported by Belloni and Alessi (2009). These results suggest that policies that modify the financial incentives embodied in pension rules are likely to have a greater impact on women. I attribute this greater degree of responsiveness among women to their greater tendency to work part-time and their lower wages. Nonetheless, the mechanisms underlying the differential sensitivity to financial incentives between males and females require further investigation. An interesting issue for future research in this sense would be the effects of the partner's financial incentives on women's retirement decisions.

For both men and women I find that new adverse health events affect the propensity to exit work via the disability route. This result is in line with Erdogan-Ciftci et al. (2011) and is not surprising since disability insurance claims are first and foremost based on the existence of a health problem. On the other hand, I find that adverse new health events affect transitions to early retirement only for males. Interestingly, financial incentives turn out to be very important in explaining transitions to early retirement, but they also explain transitions to disability. In particular, I find that pension wealth is negatively associated with the hazard of entering disability. This is in line with previous evidence showing that early retirement and disability are potential substitute pathways into retirement in the Netherlands (Kerkhofs et al, 1999; de Vos et al, 2011).

Additionally, in Chapter 2, I present evidence suggesting that a new health event increases the probability of transiting into early retirement *only* for workers with high pension wealth. Moreover, this difference is not compensated by a correspondingly larger effect on the hazard of entering disability among workers with low pension wealth. Therefore, my results provide support for the *affordability hypothesis* stated earlier. Indeed, while the occurrence of a new health event increases the probability that an older worker exits the labor market, the magnitude of the effect and the pathway chosen may depend on the resources the individual can enjoy after leaving the labour market. In contrast to my results

in Chapter 1 for the Spanish labour market, I find little empirical support for the *subordination hypothesis* in the Dutch labour market.

The analysis in Chapters 1 and 2 has focused on transitions out of salaried employment into retirement and/or disability. However, other potential responses to health events and/or financial incentives are adjustments in the number of hours worked or transitions into self-employment. There is a lot of policy-oriented literature on phased or gradual retirement (Hutchens, 2007) and indeed this option is also gaining importance in the Netherlands. An interesting issue for future research is therefore how financial incentives and health affect part-time work and or self-employment.

In the last two chapters, I analyse three types of measures of health perceptions commonly used in the modeling of labour supply. In Chapter 3, I compare the validity of two alternative ways of measuring recent health changes by examining their ability to predict subsequent survival. In particular, I consider the validity of *prospective health changes* (i.e., changes in longitudinally observed self-assessed health), versus a *retrospective measure of health changes* (i.e., responses to a question asking individuals directly to compare their health to a reference point in the past). I find that for a high proportion of reports (39.8%), prospective health changes in self-assessed health do not concur with the retrospective measure.

In terms of explanatory power for mortality, I find that *prospective changes* carry more information than *retrospective* ones. My initial results suggest that both measures of health changes predict mortality in a model that controls only for self-assessed health and socioeconomic characteristics. However, within a richer model that controls additionally for prior presence of chronic conditions and the onset of new conditions, I find that *prospective health changes* still predict mortality whereas *retrospective changes* no longer do so. I therefore conclude that, while the collection of retrospective health information in cross-sections is a cheaper and simpler alternative to the prospective collection of longitudinal health data, the former provides a measure that has less predictive power as far as mortality is concerned.

As previously shown by Jylhä (2009) and Huisman and Deeg (2010), self-assessed health is influenced by health-relevant information, but also by psychological filters like

perception, interpretation and memory. Therefore, the formation of health perceptions may involve a number of cognitive processes, such as the recall of relevant experience and the evaluation of relevant information, which may be depend on cognitive ability. I address this conjecture in Chapter 4. In particular, I investigate the formation of a forward-looking health perception, subjective survival expectations, and test its validity for different groups defined in terms of cognitive ability and educational level. This measure can be interpreted as a perception of expected lifespan, which is bound to be determined by factors ranging from individual background to, crucially, education and cognition.

As reported in Chapter 4, I find a stronger association of subjective survival expectations with mortality among individuals who have a higher level of cognitive functioning and higher education. Namely, subjective survival expectations do not predict mortality for the least educated and least cognitively able. This result holds especially when cognition is measured in terms of arithmetic ability but also in terms (short-term and immediate) memory (word recall). Even stronger disparities in predictive ability exist across education groups, and the strongest predictive power is found among college graduates. I attribute this result to the existence of greater measurement error for the less educated and the cognitively impaired. While the definition of measurement error with respect to subjective expectations data is far from clear (Elder, 2010), difficulties in expressing expectations in probabilities presumably generate noise, which will downwardly bias the predictive power of survival expectations with respect to actual survival. If measurement error is greater for the less educated and the cognitively impaired, then the estimated correlation between subjective and actual survival will be weaker for these groups. This has implications for empirical work, but it does not necessarily have substantive implications in terms of individuals' decisions concerning later life such as retirement and savings, unless it emanates from actually mistaken, rather than just wrongly reported, longevity expectations. Concerning these two alternative mechanisms, I find that individuals in the lower cognition/education groups modify their expectations in response to a considerably smaller number of new events than individuals in higher cognition/education groups. The less educated and cognitively able are less likely to revise survival expectations downward in response to new information on objective risks, such as the onset of cancer or the occurrence of a stroke. This suggests that less educated/cognitively able individuals may be less likely to change behavior and seek appropriate medical treatment in response to conditions that pose a serious threat to their longevity. As a result, these systematic biases

in the formation of survival expectations suggest low education and cognition groups may be making sub-optimal decisions with respect to their finances and health.

Using subjective expectations elicited from the agents in economic models of individual behavior is intuitive and economically appealing. However, as I argue in Chapter 4, this approach faces several difficulties. First, subjective expectations data are not widely available. Secondly, and as evidenced in this thesis, even when subjective expectations data are available, measurement errors present a challenge to their widespread use. This suggests another interesting topic for future research: how to incorporate effectively information on individual expectations about health and longevity in models of labour supply?

Nederlandse Samenvatting

De financiële en economische crisis waarmee de 21e eeuw haar tweede decennium ingaat, creëert acute problemen voor overheidsuitgaven in vele landen, voornamelijk voor de landen waar de demografische opbouw van de bevolking al gekenschetst werd als een ‘tijdbom’ voor de toekomstige pensioenuitgaven. Meer dan ooit lijkt het nu noodzakelijk om zowel de arbeidsproductiviteit alsook de pensioenleeftijd te verhogen voor een duurzame vermindering van de overheidsuitgaven. Dit zijn echter geen nieuwe problemen. Het arbeidsaanbod kromp aanzienlijk tussen de late jaren 70 en de late jaren 90 als gevolg van genereuze financiële regelingen met betrekking tot prepensioen en arbeidsongeschiktheid. Als antwoord hierop zijn er in vele landen de afgelopen twintig jaar grote wijzigingen in de sociale voorzieningen geïmplementeerd, zoals het verhogen van de pensioengerechtigde leeftijd, het koppelen van de pensioenleeftijd aan de levensverwachting, het afschaffen van prepensioen regelingen, en het aanscherpen van de ontvankelijkheidscriteria voor uitkeringen met betrekking tot werkloosheid of arbeidsongeschiktheid (OECD, 2009).

Economen hebben in het verleden veel aandacht besteed aan de effecten van sociale voorzieningen op de dynamiek in de arbeidsmarkt in het algemeen en de pensioenbeslissing in het bijzonder. Het bekende werk van Gruber en Wise (2004) laat zien dat financiële stimulansen een cruciale rol spelen in de beslissing betreffende arbeidsmarktparticipatie. Recente hervormingen om vervroegde uittrekking tegen te gaan in landen als Nederland (Euwals et al. 2011) en Spanje (García-Gómez et al. 2011) hebben dan ook succes geboekt.

Empirisch bewijs, zoals de hierboven geciteerde studies, is essentiële input voor beleidsmakers. Immers, het verhogen van de pensioengerechtigde leeftijd of het introduceren van financiële stimulansen om vervroegde uittrekking tegen te gaan is geen garantie dat mensen ook daadwerkelijk langer door zullen werken. Het lijkt mogelijk om langer door te werken, maar alleen als de stijging in levensverwachting ook gepaard gaat

met een verbetering van de gezondheid. Echter, ondanks een verbetering in de meeste gezondheidsindicatoren over de laatste decennia, blijven grote ongelijkheden in gezondheid bestaan en in sommige gevallen worden deze zelfs groter. In het bijzonder hebben arme en kansarme mensen met lichamelijk zware beroepen een grotere kans op arbeidsongeschiktheid en ziekte (Bajekal en Goldblatt, 2006). Aangezien gezondheid, naast financiële stimulansen, een belangrijke rol speelt in het arbeidsaanbod, kunnen hervormingen van de sociale voorzieningen wellicht een ander effect hebben voor mensen met verschillende niveaus van gezondheid. Mensen in goede gezondheid kunnen wellicht doorwerken tot een hogere leeftijd, terwijl mensen in slechte gezondheid hier niet toe in staat zijn. Bovendien kan het zijn dat werknemers met een lager loon en vermogen gedwongen zijn om langer door te werken ondanks een slechte gezondheid, terwijl rijke werknemers het zich kunnen permitteren om eerder te stoppen met werk.

Deze overwegingen leiden tot de volgende onderzoeksvragen: (1) is de effectiviteit van financiële stimulansen in de sociale voorzieningen afhankelijk van de gezondheid van individuen? En, gerelateerd, (2) is de beslissing om de arbeidsmarkt te verlaten na een schok in gezondheid afhankelijk van iemands financieel vermogen?

In dit proefschrift presenteer ik vier empirische studies. De eerste twee studies concentreren op het effect van 'gezondheidsschokken' (bijv. een hartaanval, een hernia, etc.), financiële stimulansen en de interactie tussen die twee op de beslissing tot uittreding van de arbeidsmarkt ofwel door pensionering ofwel door arbeidsongeschiktheid. Deze studies trachten een antwoord te geven op de onderzoeksvragen geponeerd in de vorige paragraaf. De laatste twee hoofdstukken van dit proefschrift beschrijven twee maten van gezondheidsperceptie die vaak worden gebruikt in het modelleren van economische beslissingen, in het bijzonder beslissingen omtrent arbeidsaanbod. Deze hoofdstukken onderzoeken de validiteit en de formatie van prospectieve en retrospectieve gezondheidspercepties.

Hoofdstuk 1 gaat over de relatie tussen gezondheidsschokken en financiële stimulansen vanuit de sociale voorzieningen betreffende pensioen en arbeidsongeschiktheid. Ik gebruik de theoretische inzichten van het 'Option Value' model (Stock en Wise, 1990) om de rol van gezondheid te onderzoeken in de beslissing om uit te treden van de arbeidsmarkt. Op basis van dit model stel ik de hypothese op dat de effecten van financiële stimulansen

ondergeschikt zijn aan de gezondheid van het individu. Vervolgens onderzoek ik empirisch, gebruik makende van de Spaanse steekproef van het European Community Household Panel, of individuen met verschillende gezondheidsniveaus anders reageren op financiële stimulansen als het gaat om beslissingen met betrekking tot pensionering. Ik vind empirisch bewijs voor deze zogenaamde ‘subordination’ hypothese.

In hoofdstuk 2 van dit proefschrift toets ik de ‘subordination’ hypothese in de context van de Nederlandse arbeidsmarkt. Daarnaast onderzoek ik ook de tweede onderzoeksvraag, welke ik definieer als de ‘affordability’ hypothese, of mensen met hogere pensioenopbouw het zich kunnen veroorloven (‘can afford’) om eerder uit te treden na een gezondheidsschok. De data is dermate rijk – administratieve gegevens van het pensioenfonds voor mensen in de gezondheidszorg – dat ik deze vraagstelling apart voor mannen en vrouwen kan onderzoeken. Verder bezit deze data, in tegenstelling tot de data gebruikt in hoofdstuk 1, objectief gemeten pensioenopbouw en gezondheidsschokken op basis van ziekenhuisgegevens. Deze rijkdom aan data verschaft een groot vertrouwen in robuuste schattingen van de empirische effecten. Voor zowel mannen als vrouwen leidt een gezondheidsschok tot een verhoogde kans op arbeidsongeschiktheid. Als het gaat om het effect van gezondheid op pensionering geldt dit alleen voor mannen. Ook vind ik dat financiële stimulansen niet alleen een rol spelen bij vervroegde pensionering, maar ook bij arbeidsongeschiktheid. Verder vind ik dat gezondheidsschokken de kans om met vervroegd pensioen te gaan *alleen* verhogen voor werknemers met een hoge pensioenopbouw. Bovendien is dit verschil ten opzichte van werknemers met een lage pensioenopbouw niet volledig geëlimineerd door een hoger effect op arbeidsongeschiktheid voor hen. Deze resultaten zijn in lijn met de ‘affordability’ hypothese. Echter, in contrast met de resultaten op basis van de Spaanse data in hoofdstuk 1, vind ik geen empirisch bewijs voor de ‘subordination’ hypothese in de Nederlandse context.

In hoofdstuk 3 onderzoek ik de validiteit van prospectieve veranderingen in gezondheid, i.e. veranderingen gebaseerd op longitudinale informatie over gezondheidsstatus, versus retrospectieve veranderingen in gezondheid, i.e. veranderingen op basis van de antwoorden van individuen op de vraag om hun gezondheid nu te vergelijken met een referentie punt in het verleden. In het bijzonder onderzoek ik de voorspellende waarde van deze maatstaven voor sterfte. Ik concentreer me in dit hoofdstuk op de geobserveerde en gerapporteerde van

veranderingen in zelfervaren gezondheid – vaak de enige gezondheidsmaatstaf in de meeste enquêtes. Ik focus op veranderingen (schokken) in gezondheid, aangezien deze waarschijnlijk belangrijkere indicatoren zijn voor toekomstige uitkomsten dan niveaus van gezondheid. Ik gebruik een unieke longitudinale dataset, namelijk de Nederlandse GLOBE cohort studie, die zowel maatstaven voor veranderingen in gezondheid alsook sterfte omvat. De resultaten wijzen uit dat voor een groot gedeelte van de ondervraagden de prospectieve gezondheidsveranderingen niet overeenkomen met de retrospectieve. Verder laat dit hoofdstuk zien dat veranderingen in gezondheid additionele informatie bevatten in vergelijking met niveaus van gezondheid. De schattingen laten zien dat beide maten voor gezondheidsveranderingen voorspellende kracht hebben voor sterfte, zelfs als wordt gecontroleerd voor het niveau van gezondheid en sociaaleconomische eigenschappen van de persoon. Echter, als er gecontroleerd wordt voor zelfervaren gezondheid en eerdere en nieuwe chronische aandoeningen, zijn prospectieve gezondheidservaringen nog steeds voorspellende voor sterfte, maar retrospectieve veranderingen niet.

In hoofdstuk 4 concentreer ik me op subjectieve overlevingskansen, een gezondheidsmaatstaf die meer toekomstige gezondheid meet dan de gezondheidsveranderingen van hoofdstuk 3. De mate waarin iemand informatie kan verwerken en begrip kan kweken voor zijn verwachte levensduur is mogelijk een belangrijke determinant van toekomstige uitkomsten zoals pensionering en besparingen. Het is dan ook zeer interessant voor zowel economen als beleidsmakers om de formatie en validiteit van deze overlevingskansen te begrijpen. Ik onderzoek de formatie en validiteit van subjectieve overlevingskansen op basis van de Health and Retirement Study (HRS) in de Verenigde Staten. De vraag is of de accuraatheid van de subjectieve overlevingskansen varieert met leeftijd, opleiding en cognitieve vaardigheden. In lijn met eerdere literatuur vind ik dat verwachtingen met betrekking tot levensverwachting een goede voorspellende waarde hebben voor echte overleving. Echter, deze associatie is sterker voor de groep hoger opgeleiden en mensen met grotere cognitieve vaardigheden. Subjectieve levensverwachting is zelfs helemaal niet voorspellend voor echte levensverwachting voor lager opgeleiden en mensen met weinig cognitieve vaardigheden. Ik interpreteer deze resultaten als bewijs voor een grotere ruis ('measurement error') in de subjectieve levensverwachting voor lager opgeleiden en mensen met weinig cognitieve vaardigheden. Deze heterogeniteit in ruis leidt me er toe om te onderzoeken of er ook verschillen zijn in de manier waarop nieuwe informatie de subjectieve verwachtingen beïnvloedt. De

resultaten laten zien dat mensen met een lage opleiding of lage cognitieve vaardigheden de subjectieve verwachtingen in minder gevallen aanpassen – ze zijn minder snel geneigd om hun verwachte levensverwachting aan te passen in antwoord op nieuwe informatie over objectieve risicofactoren zoals de ontdekking van kanker of een herseninfarct.

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