Heterogeneous and Uncertain Health Dynamics and Working Decisions of Older Adults*

Angela Denis†

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Abstract

I study heterogeneity in health dynamics of older adults and its effects on their working decisions. After showing evidence of this heterogeneity, I use subjective survival expectations to infer health beliefs in a Bayesian-learning framework, and I flexibly estimate how working decisions depend on those beliefs. The results show individuals incorrectly believe their health will deteriorate too fast and eliminating that bias would increase labor-force participation by up to 2 percentage points. Providing information on blood glucose and cholesterol levels has only small effects on beliefs and working decisions, and consequently, policies with larger effects on beliefs are needed to delay retirement.

JEL codes: D83, I14, J14, J26.

Keywords: Health dynamics, older adults, retirement, uncertainty, beliefs.

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†Banco de España. E-mail: angela.denis@bde.es

1 Introduction

The population is aging rapidly, putting considerable strain on public budgets. The number of older adults out of the labor force who will need to be supported by each worker is projected to increase by around 40% between 2018 and 2050. Therefore, promoting employment at older ages has garnered large interest.[1] The success of policies promoting the employment of older adults depends on our correct understanding of the determinants of working decisions of this group, for whom health is an important factor.

For older adults, health deteriorates naturally with aging. Yet, little is known about the heterogeneity in health dynamics of older adults and how this heterogeneity affects their working decisions. Using data from the Health and Retirement Study (HRS), this paper addresses this question.

I start by showing evidence that health dynamics are indeed heterogeneous among older adults. That is, while some individuals see their health slowly deteriorating with age, other individuals see their health deteriorating more rapidly. This heterogeneity helps explain why as the population ages the variance of health increases, a pattern observed in the data but mostly ignored by traditional models of health.

As these heterogeneous dynamics happen at older ages, individuals may not know their own dynamics before hand. Furthermore, what matters for their decisions is their beliefs about their own health dynamics. Hence, I next study uncertainty in health dynamics, by assuming individuals are Bayesian learners and update their beliefs as they see their health changing with age. To pin down the parameters of the initial beliefs, namely bias and uncertainty, I leverage data on subjective survival expectations. Future survival depends on future health, and hence, on health dynamics; therefore, beliefs about survival speak to beliefs about health dynamics. In particular, I show that the covariance between changes in health and changes in survival rates is the key moment for identifying uncertainty in beliefs.

Using the Simulated Method of Moments, I find individuals are indeed uncertain, updating their beliefs over time, and they are negatively biased, that is, on average, they believe their health will deteriorate faster than the average rate in the population.

I study the link between health beliefs and working decisions of older adults, focusing on the extensive margin. I estimate reduced-form equations of working decisions, as a function of those beliefs, parametrically by probit and non-parametrically by neural networks. In this last case, to deal with the fact that some of the inputs are unobserved by the econometrician (mainly, the individual-level heterogeneity in initial beliefs), I implement an iterative

[1] See statistics from OECD (2019). In 2015, the OECD adopted an agenda promoting employment at older ages, to protect living standards and public finances (OECD (2015)).

approach in the spirit of EM algorithms (Dempster, Laird, and Rubin (1977)).

I show beliefs matter for working decisions, and that expecting health to deteriorate more rapidly is associated with lower probabilities of working. Furthermore, for individuals in their 50s who are not working, there is a positive interaction between beliefs and health. This interaction suggests that adjustment costs of finding a job are important, as an improvement in health that is expected to be short-lived has lower effects than one that is expected to be long-lived. The flexibility of the data-driven estimation method implies this result is not a consequence of any structure imposed to the model, and it suggests that structural models studying labor decisions of older adults should consider such costs.

I use my flexible estimated model to implement an impulse-response approach, and to simulate the impact of eliminating the initial bias in beliefs in working decisions. The results show that eliminating the initial bias increases participation by 2 percentage points, a large effect that lasts beyond traditional retirement ages.

In the context of heterogeneous and uncertain health dynamics, a health shock has two effects on working decisions: it affects working decisions by changing health status, and it affects working decisions by changing beliefs about health dynamics. I decompose the effect of a health shock into these two channels, and find that nearly all the effect goes through affecting health status. This result comes from the signal-to-noise ratio of health being low, and it implies health by itself is not enough to resolve the uncertainty and correct the bias in beliefs.

To summarize my main findings, I document four facts. First, individuals are uncertain about their own health profiles; second, they have biased initial beliefs; third, health changes are not enough to resolve uncertainty; and fourth, beliefs matter for working decisions. A natural question that follows is: Can we provide additional information to individuals in order to correct their beliefs and affect their working decisions?

In the last part of the paper, I look at this question in the context of an information experiment available in the HRS. Starting in 2006, the HRS collects and analyzes blood samples of their interviewees and informs them about their blood-glucose and cholesterol results. Although the implementation in the HRS was not designed as an information experiment, in order to save costs the blood sample is collected for a random half of the sample each wave, providing us with exogenous variation. A reduced-form analysis in the spirit of difference-in-differences[2] shows small and insignificant effects of this additional information on survival expectations and working decisions. A modified learning model, that includes the biomarker results as additional signals, tells us why: the magnitude of this blood-based signal is too small.

[2] As discussed in section 6, the design needs to control also for changes in the interview mode.

Relation to the literature. This paper is related to three strands of the literature. First, it is related to the literature studying health dynamics, a literature that consistently finds persistence and heterogeneity in health, both among the general population (Halliday (2008), Hernández-Quevedo, Jones, and Rice (2008), Contoyannis, Jones, and Rice (2004)) and among older adults (Heiss, Börsch-Supan, Hurd, and Wise (2009), Heiss (2011), Heiss, Venti, and Wise (2014), Lange and McKee (2011)). However, most of this literature only allows for heterogeneity in health-levels. An exception is Halliday (2008), who also allows for heterogeneity in health changes with age, but finds only weak evidence of this heterogeneity. However, he focuses on a much younger population, whereas I focus on older individuals for whom health changes with age are prevalent.

Second, this paper is related to the literature on empirical learning, specifically, to the literature studying individuals' learning of own unobserved heterogeneity, for example, regarding abilities (Stinebrickner and Stinebrickner (2014), Arcidiacono, Aucejo, Maurel, and Ransom (2016)), productivity (Arcidiacono, Aucejo, Maurel, and Ransom (2016)) and income profiles (Guvenen (2007), Guvenen and Smith (2014)). The paper is most closely related to Guvenen and Smith (2014), who study an income process with heterogeneous levels and heterogeneous growth rates. The main difference is that I use subjective expectations, as opposed to the outcome variable, to identify the parameters of the learning model, and therefore my results are robust to misspecification of the relation between beliefs and outcomes. Additionally, this paper relates to a more recent literature on the provision of information and its effects on beliefs (see, e.g., Delavande and Kohler (2015), Wiswall and Zafar (2014), Bates (2020)).

Finally, the paper is related to the literature on health and other outcomes of older adults. Particularly, the paper is related to the literature studying the effects of health on work and retirement choices (Siddiqui (1997),McClellan (1998), Bound, Schoenbaum, Stinebrickner, and Waidmann (1999), French (2005), Disney, Emmerson, and Wakefield (2006), Zucchelli, Jones, Rice, and Harris (2010), Maurer, Klein, and Vella (2011)) and plans (Dwyer and Mitchell (1999), McGarry (2004)). Although this literature considers future health as uncertain, it assumes a known stochastic process for health. On the contrary, this paper allows for a stochastic health process that is not fully known, introducing the role of health beliefs as an additional determinant of those decisions.

Outline. The paper proceeds as follows. Section 2 describes the data. Section 3 provides evidence of heterogeneity in health dynamics, and Section 4 provides evidence of uncertainty. Section 5 presents the main results for working decisions as a function of beliefs. Section 6 analyzes the information experiment available in the HRS. Finally, Section 7 concludes.

2 Data

I use data from waves 4 to 12 of the Health and Retirement Study (2014) (HRS),[3] a US longitudinal survey representative of individuals 50 years and older. The survey includes questions about health, survival expectations, and labor participation, among others.

In surveys, health is usually measured by self-assessed health, an ordinal variable taking five values from very poor to excellent. This variable has been shown to correlate with several outcomes; however, its limited range makes it not ideal to study health dynamics with age. Instead, I use a battery of health-related measures included in the HRS to construct, via factor analysis, a summary health variable that I use throughout the paper.[4] The measures include self-assessed health, but also, number of chronic conditions and difficulties in activities of daily living, among others. These measures reflect the health concept that is relevant for the working decisions of older adults, related to how individuals perceive their health in relation to their everyday activities. Appendix B presents descriptive statistics on these health-related measures and provides details on the estimation of the summary health variable h_{it}. Larger values of h_{it} represent better health, and an increase of one unit in h_{it} corresponds to having one less chronic condition.

The HRS also asks about subjective survival expectations in variable $plive10_{it}$. Specifically, the question asks *What is the percentage chance you will live to be (80, 85, 90, 95 or 100) or more?* The reference age is a function of the individual's age and the wave of the survey, and it is usually around 10 to 15 years into the future.[5]

I study the extensive margin of labor-participation decisions of older adults, and define a binary variable p_{it} as 1 for working individuals.

The overall sample consists of 156,976 observations from 31,210 individuals 50 years and older, interviewed in person[6] in some wave between waves 4 and 12. Table 1 presents descriptive statistics on the main variables.[7]

[3] The HRS (Health and Retirement Study) is sponsored by the National Institute on Aging (grant number NIA U01AG009740) and is conducted by the University of Michigan. For most of the analysis, I use the RAND HRS Longitudinal File (2014), which is an easy-to-use dataset based on the HRS core data. This file was developed at RAND with funding from the National Institute on Aging and the Social Security Administration.

[4] This approach of using several measures to construct a summary variable is not unique to this paper; see, for example, Heiss, Venti, and Wise (2014), Lange and McKee (2011), and Blundell, Britton, Dias, and French (2017)

[5] The HRS also includes another question on survival expectations to the age of 75, only asked of individuals under 65 years old. I use this variable to check the fit of the beliefs model in Section 4.

[6] I exclude proxy interviews because these interviews do no ask questions about survival expectations.

[7] For different analyses in the paper, I restrict the sample further as needed; see Appendix A for an overview of these samples.

Table 1: Summary statistics of main variables

	Observations	Mean	SD	Min	Max
Age	156,976	67.44	10.35	50	109
Health	148,866	5.19	0.67	2.94	6.15
Survival expectations	125,658	0.47	0.32	0	1
Working decisions	156,582	0.37	0.48	0	1

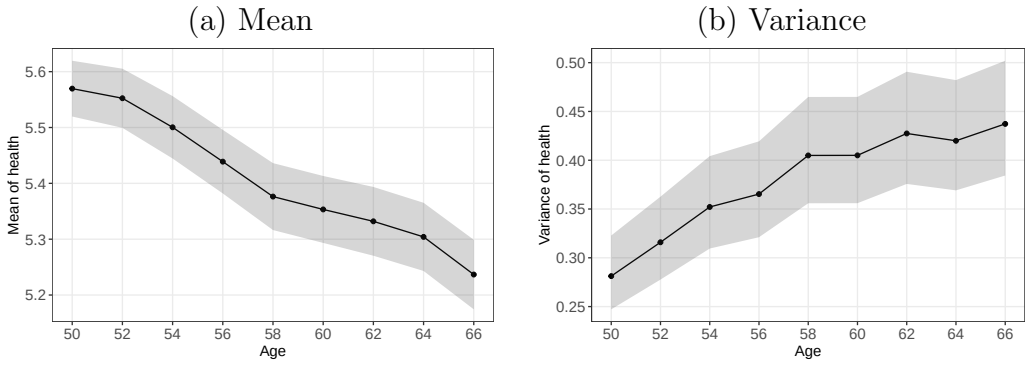
*Notes: plive*10 *is re scaled so it takes values between 0 and 1.*

3 Heterogeneous health dynamics

The first innovation introduced in this paper is to consider individual heterogeneity in the dynamics of health of older adults, in particular, in how health changes with age.

To motivate this heterogeneity, Figure 1 shows the mean and variance of health h_{it} by age. The figure shows that as people age their average health decreases while its variance increases. This pattern of decreasing mean and increasing variance is robust to sample composition and also holds for most of the individual measures. The pattern in these graphs suggests a process with heterogeneous slopes with age.

Figure 1: Mean and variance of health by age



Notes: Results from a balanced sample of 433 individuals observed at 50 years with at least 9 consecutive waves. The bands represent 95% confidence intervals.

Let i denotes an individual and t denotes his age. I focus on individuals 50 years and older and define t as 0 for age 50.[8] I assume the following health process,

$$h_{it} = \rho h_{it-1} + \alpha_i + \delta_i \cdot t + \epsilon_{it}.$$

[8] Given the two years between waves, throughout this paper, I consider age as measured in two-year bins.

The parameter $\rho \in (0,1)$ captures persistence in health, α_i captures heterogeneous levels in health, δ_i captures heterogeneous changes in health with age, and ϵ_{it} represents health shocks. That is, health is a dynamic process that, as people get older, naturally deteriorates in a heterogeneous way across individuals. Both the persistence of health and its heterogeneity in levels are well-recognized elements of health in the literature, both among the general population (see, e.g., Hernández-Quevedo, Jones, and Rice (2008)) and among older individuals (see, e.g., Heiss, Venti, and Wise (2014)). The first novel element in this paper is to allow for heterogeneous slopes of health with age, δ_i. As larger values of h_{it} represent better health and health decreases with age, $\delta_i < 0$. I assume the health process is exogenous and focus on changes in health due to aging.

Health is an important determinant for survival, especially for older adults, with individuals surviving to older ages having better health to begin with than individuals that don't survive that long. Hence, ignoring survival when studying health of older adults leads to bias (see e.g. Heiss, Venti, and Wise (2014)). I assume the survival process is given by

$$S_{it} \quad = \quad \mathbb{1}\{\gamma h_{it-1} + \theta_0 + \theta_1 \cdot t + \theta_2' x_i + \eta_{it} \geq 0\} S_{it-1},$$

where x_i is a vector of time-invariant demographic characteristics that includes gender, race, hispanic ethnicity, and education. η_{it} is a random shock, which I assume independent to the health shock ϵ_{is}. The parameter γ captures the dependence of survival on health.

I estimate a system of equations for health h_{it} and survival S_{it}, assuming health levels α_i and health slopes δ_i are jointly normally distributed, with a mean that varies with demographic characteristics. Hence, the overall model is a random coefficients model, which I estimate by maximum likelihood; see Appendix C for the details.

Table 2 presents the main results. The table shows that health decreases with age and has a relatively low persistence. In terms of the individual heterogeneity, the table shows two main results. First, there is heterogeneity in both the intercepts and the slopes of the health process, with positive and significant variances. Second, these two sources of heterogeneity are uncorrelated, which implies knowing α_i does not provide additional information on δ_i. The coefficient for health in the survival equation is positive and significant, implying that survival is more likely for healthier individuals. The full set of results are reported in Table C1. It shows that individuals with low levels of education have worse health, health deteriorates faster for non-white individuals, and probabilities of survival are higher for women and Hispanic individuals.

By allowing for heterogeneous slopes of health with age, the model does indeed predict an increasing variance with age. To see this, Figure 2 repeats the exercise for a sample of

Table 2: MLE results on health and survival

	Symbol	Coefficient	Pvalue
Persistence	ρ	0.223	0.000
Mean of α_i	μ_α	0.955	0.000
Mean of δ_i	μ_δ	-0.057	0.018
SD of α_i	σ_α	0.235	0.000
SD of δ_i	σ_δ	0.043	0.000
$Corr(\alpha_i, \delta_i)$	ϕ	-0.033	0.714
SD of health shocks	σ_ϵ	0.266	0.000
Survival dependence on health	γ	0.583	0.001
Controls		Yes	
N alive observations		8,901	
N dead observations		112	
N individuals		1,671	
-Log likelihood		3,027.6	

Notes: Main results of estimating the health and survival processes. Details and full set of results are given Appendix C.

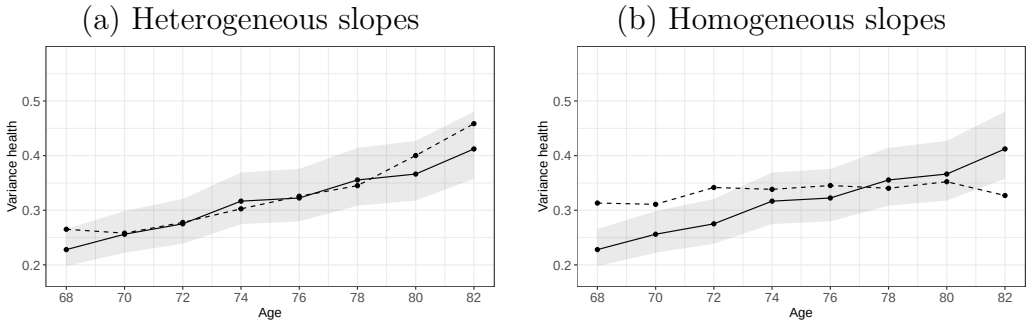
individuals observed from 66 years old and plots the predicted variance of health with age for two cases: allowing for heterogeneous slopes across individuals (left panel) and assuming instead homogeneous slopes across individuals (right panel). The figure shows that by ignoring slope heterogeneity, we predict a rather constant variance of health, contrary to what is observed in the data.[9]

I include several robustness checks in Appendix C.2. First, I estimate a version of the model with heteroskedastic error ϵ_{it}, allowing its variance to depend on age. The results show an increasing variance of health shocks does not explain away the heterogeneity in slopes δ_i. Second, I estimate a version of the model adding the unobserved heterogeneity (α_i, δ_i) to the survival equation. The results show α_i and δ_i are not (jointly) significant; that is, I find no direct effect of heterogeneity in survival, once we control for health. This lack of significance has an important implication for the beliefs model in the next section: it means survival does not contain additional information about δ_i, beyond the information already contained in health.[10] Finally, I estimate a similar model using self-assessed health instead of the summary health variable h_{it}. The results show the presence of heterogeneous slopes

[9] In an additional exercise, I observe that allowing for heterogeneous slopes but ignoring survival leads to an overestimation of the variance of health with age, as the prediction includes the left tail of health distribution that has lower probability of survival.

[10] If this were not the case and survival contained additional information about δ_i, the learning model defined in Section 4 would not be valid, as it does not include survival as a signal.

Figure 2: Variance of health in models with and without slope heterogeneity



Notes: The sample consists of 26,950 correlative observations from 7,301 individuals observed since they were 66 years old. Over the span of the following eight waves, 996 of them died. The figure plots data from 354 individuals with health in all 9 waves. The solid lines plot the health data and the dotted lines plot the predicted values of health in each model.

with age is robust to using only this measure.

4 Uncertain health dynamics and beliefs

The second innovation of this paper is to allow for individuals to be uncertain about their health dynamics. In this section, I study how much individuals know about their own health dynamics, by using data on subjective survival expectations. Given that future survival depends on future health, survival expectations depend on expectations about future health, and therefore, on beliefs about health dynamics.

Survival expectations have been shown to have predictive power for survival (Hurd, McFadden, and Merrill (2001), Hurd and McGarry (1995)) and to be consistently updated with new health information (Hurd and McGarry (2002), Smith, Taylor, and Sloan (2001)). Furthermore, survival expectations are correlated with several outcomes for older individuals.

4.1 Belief about health dynamics

I assume individuals know α_i but not δ_i and try to learn it given their observed health history.[11] Furthermore, I assume individuals are rational Bayesian learners with prior beliefs

[11] Given that health deteriorates in old age, I assume 50-year-old individuals do not know δ_i, which has not affected them before. This assumption is consistent with results from Halliday (2008), who studies health dynamics with discrete heterogeneity, using the Panel Study of Income Dynamics. He studies younger individuals, ages 22 to 60, and finds no heterogeneous slopes with age. Furthermore, I assume individuals know their heterogeneous level α_i because they have observed their health for several decades. This assumption can be generalized; in studying income profiles, Guvenen (2007) proposes a similar process for income with heterogeneous intercepts and slopes, both unknown. He finds the learning process for

about δ_i given by $N(\hat{\delta}_{i0}, \hat{\sigma}_0^2)$,[12] which they update over time as they see their health changing. Then, by Bayes rule, posterior beliefs about δ_i over time are also normally distributed. The posterior mean $\hat{\delta}_{it}$ and variance $\hat{\sigma}_t^2$ have closed-form solutions which are given in Appendix D.1.

Hence, conditional on health history, the key parameters determining beliefs are the parameters governing initial beliefs:

$$\begin{aligned} b &= \mathbb{E}(\hat{\delta}_{i0} - \delta_i), \\ \lambda^2 &= \frac{\hat{\sigma}_0^2}{Var(\delta_i)}. \end{aligned}$$

The parameter b measures the bias in initial beliefs at the population level. If b is positive (negative), individuals are upward (downward) biased, and hence, they believe health deteriorates on average more slowly (faster) than the average rate. The parameter λ measures the degree of initial uncertainty individuals face regarding δ_i, which affects their amount of learning over time. If $\lambda = 0$, there is no uncertainty and therefore no learning. The larger the value of λ, the more uncertain individuals are. The Bayesian learning and normality assumptions allow me to reduce the dimensionality of the problem, giving structure to time-varying beliefs that are unobserved by the econometrician.

4.2 Relation with survival expectations

To pin down these two parameters, b and λ, I use data on subjective survival expectations. The intuition for identification using this data is as follows. Each period, individuals observe their health and update their beliefs regarding their unknown δ_i. This new information allows them to also update their beliefs about their future health, and hence their expectations about future survival. Thus, slope beliefs, unobserved by the econometrician, are closely linked to survival expectations, which are observed. The function depends on the health and survival processes estimated in Section 3, and it is given in Appendix D.2. If individuals have a large negative (positive) bias in beliefs, expecting their health to deteriorate too fast (slow), survival expectations are going to be lower (larger). Hence, we can pin down b using levels of survival expectations. Proposition 4.1 provides a formal identification result for λ using data on expected survival *rates*.

intercepts is much faster than the learning process for slopes.

[12] The assumption of common-prior variance across individuals, i.e. that $\hat{\sigma}_0$ does not depend on i, is usual in the learning literature. See, for example, Guvenen (2007) and Arcidiacono, Aucejo, Maurel, and Ransom (2016). However, the assumption is important for the identification results provided later.

Proposition 4.1 (Identification of λ) *Let the health and survival processes be given by equations (A2) and (A3), and assume individuals are Bayesian learners with prior beliefs about δ_i following $N(\hat{\delta}_{i0}, \hat{\sigma}_0^2)$. Let Ω_{it} be the information set of individual i after observing his health up to period t. Thus, $\alpha_i, \hat{\delta}_{it}, \hat{\sigma}_t^2 \in \Omega_{it}$. Consider the subjective expectations about survival rates between periods $t+2$ and $t+3$, from the point of view of $t+1$ and t, that is, the expected survival rates 1 and 2 periods ahead.*

$$\begin{aligned} b_{it+1}^{(1)} &\equiv \mathbb{P}(S_{it+3} = 1 | S_{it+2} = 1, \Omega_{it+1}), \\ b_{it}^{(2)} &\equiv \mathbb{P}(S_{it+3} = 1 | S_{it+2} = 1, \Omega_{it}). \end{aligned}$$

Define $\Delta B_{it+1} = \Phi^{-1}(b_{it+1}^{(1)}) - w_t \Phi^{-1}(b_{it}^{(2)})$, the weighted difference of these expected survival rates, where Φ is the standard normal CDF and w_t is constant across individuals.[13]

Then, there exist a function $F(\lambda, t, \Theta)$, such that, conditional on h_{it}, $b_{it}^{(1)}$ and $b_{it}^{(2)}$,

$$Cov(\Delta B_{it+1}, \Delta h_{it+1}) = F(\lambda, t, \Theta), \tag{1}$$

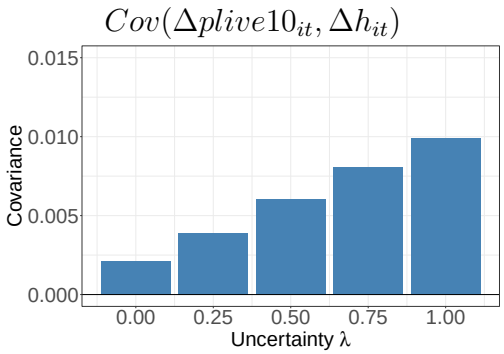
with $F(\lambda, t, \Theta)$ increasing in λ.

The proposition says that, given Θ, we can identify λ with enough longitudinal data on health and subjective expectations about these survival *rates*, as the left hand side of equation (1) would be observed and the right hand side is increasing in the one unknown parameter λ. Furthermore, this equation says that the key moment for identification is the covariance between changes in health and changes in expectations about survival *rates*. The proof is in Appendix D.3. The key equation (equation (A11)) shows individuals update their survival expectations for two reasons. The first reason is that health is a persistent process; thus, any change in health will have future repercussions on health and therefore on survival. If health is not a persistent process, that is $\rho = 0$, this channel disappears. The second reason is that learning implies a change in future predictions of health and therefore of survival. If there is no uncertainty in beliefs, that is $\lambda = 0$, there is no learning and this channel disappears. The more uncertain beliefs are, i.e for larger values of λ, the larger is the change in expected survival given the same change in health.

In practice, we do not have data on subjective expectations about survival *rates*, but we do have data on (unconditional) subjective expectations about survival and the same intuition applies there. Appendix D.4 describes a simulation exercise showing that the covariance between changes in health and changes in survival expectations is the key moment to identify the uncertainty parameter λ, as shown in Figure 3.

[13] The expression for w_t is given in Appendix D.3.

Figure 3: Simulated covariance by uncertainty parameter λ



Notes: Covariance in simulated data following the structure of the available data in the HRS. The x-axis shows the value of the uncertainty parameter λ used in the data-generating process. See Apendix D.4 for details and results on other moments.

4.3 Estimation of beliefs parameters

To estimate bias b and uncertainty λ, I use the Simulated Method of Moments (SMM).[14] I use six moments, three in levels and three in differences, corresponding to the mean of $plive10_{it}$, its variance, and its covariance with h_{it}. As subjective survival expectations are measured with error,[15] I allow for non-classical i.i.d. measurement error $\nu_{it} \sim N(\mu_{merror}, \sigma^2_{merror})$, such that the observed survival expectations are given by $\widetilde{plive10}_{it} = \max\{\min\{plive10_{it} + \nu_{it}, 1\}, 0\}$.[16]

Table 3 presents the estimation results. It shows individuals face a sizable amount of uncertainty and a large amount of negative initial bias; that is, individuals believe their health will deteriorate with age at a faster rate than what is actually true on average. In line with previous literature, subjective survival expectations are subject to large amounts of measurement error. Following Manski and Molinari (2010), I also estimate a version including rounding and find similar results. Overall, these results are consistent with previous evidence that finds that, on average, older adults up to 65 years old underestimate their chances of survival (Elder (2013), Ludwig and Zimper (2013)). Those papers also find adults 80 years and older overestimate their survival chances. My results explain the higher declared

[14] I assume $(\alpha_i, \delta_i, \hat{\delta}_{i0})$ are jointly normally distributed, with $Cov(\alpha_i, \hat{\delta}_{i0}) = Cov(\alpha_i, \delta_i)$. This assumption implies the information about δ_i contained in α_i is already incorporated in initial beliefs $\hat{\delta}_{i0}$. In Section 3 we estimated this covariance to be zero.

[15] See, for example, Manski and Molinari (2010) and Kleinjans and Van Soest (2014).

[16] Note that the measurement error shifts observed survival expectations by μ_{merror} on average. Similarly, the bias in initial beliefs b also shifts observed survival expectations. However, these two biases have different effects over time: the average shift due to measurement error is constant with age, given the i.i.d. assumption, whereas the average shift due to initial bias in beliefs is decreasing with age as individuals update their beliefs over time. Thus, we can separately identify both effects.

Table 3: SMM results on prior beliefs

	Symbol	Coefficient	Lower bound	Upper bound
Uncertainty	λ	0.338	0.336	0.340
Bias	b	-0.061	-0.061	-0.060
Mean of measurement error	μ_{merror}	0.121	0.118	0.123
SD of measurement error	σ_{merror}	0.177	0.176	0.177

Notes: Prior beliefs about slopes are unobserved $N(\delta_i + b, \lambda^2\sigma_\delta^2)$, depending on the bias b and uncertainty λ parameters, whereas subjective survival expectations $plive10_{it}$ are observed but measured with error. The estimation uses a subsample of 2,000 individuals with eight periods, chosen randomly for computational reasons. Moments are simulated using 20 draws of measurement error and 20 draws of unobserved heterogeneity. The bounds correspond to a 95% confidence interval, constructed using standard errors clustered at the individual level.

probabilities of this group by measurement error.

Table 4 presents the fit of the results. The top panel shows the fit of the targeted moments using $plive10_{it}$, whereas the bottom panel shows the fit of similar non-targeted moments using survival expectations to age 75. The table shows that the estimation performs reasonably well both for targeted and non-targeted moments.

The learning model and the results in this section rely on two simplifying assumptions: that health is exogenous and that health is the only source of information regarding δ_i. The first assumption is common in models of labor supply of older adults and it is considered a reasonable simplification for this group, as studies that allow for health care effects on health find only small effects (French and Jones (2017)). This exogeneity assumption rules out the possibility of individuals changing their behavior to affect their health. If that were possible, survival expectations would include individuals' plans of changing their future behavior in order to change their future health and their survival chances. As those planned behaviors are more likely to compensate for bad future outcomes, the results in this paper would be lower bounds on the magnitude of the underlying bias and uncertainty under an endogenous health process. The second assumption is common in learning models, and it rules out endogenous acquisition of health-related information, for example, by some individuals going to their doctors to better predict their future health. This assumption is partly addressed in Section 6 where I look at an additional and exogenous source of information that may shift beliefs.

Table 4: Moments' fit

(a) Targeted moments			
	Data moment	SE	Simulated moment
$\mathbb{E}(plive10)$	0.531	(0.00011)	0.538
$\mathbb{E}(plive10^2)$	0.371	(0.00012)	0.357
$\mathbb{E}(plive10 \cdot h)$	2.890	(0.00065)	2.957
$\mathbb{E}(\Delta plive10)$	-0.013	(0.00002)	-0.014
$\mathbb{E}((\Delta plive10)^2)$	0.070	(0.00003)	0.066
$\mathbb{E}(\Delta plive10\Delta h)$	0.007	(0.00002)	0.007
(b) Non-targeted moments			
	Data moment	SE	Simulated moment
$\mathbb{E}(plive75)$	0.702	(0.00017)	0.806
$\mathbb{E}(plive75^2)$	0.556	(0.00021)	0.687
$\mathbb{E}(plive75 \cdot h)$	3.886	(0.00101)	4.469
$\mathbb{E}(\Delta plive75)$	-0.001	(0.00010)	0.018
$\mathbb{E}((\Delta plive75)^2)$	0.054	(0.00008)	0.042
$\mathbb{E}(\Delta plive75\Delta h)$	0.006	(0.00005)	0.003

Notes: Panel (a) uses the same sample used for estimation. Panel (b) uses a subsample of $1,247$ *individuals up to 65 years old who are asked* $plive75_{it}$ *(the percentage chance you will live to be 75). Standard errors are clustered at the individual level.*

5 Working decisions and beliefs about health

In this section I answer the main question of this paper, that is, how beliefs about health dynamics matter for labor-participation decisions of older adults. I am interested in understanding if individuals expecting their health to deteriorate more rapidly make different working decisions than similar individuals -with similar health- but who expect their health to deteriorate more slowly. The overall goal is to understand how bias in these beliefs affect working decisions, and therefore, if eliminating that bias could significantly change those decisions.

In a dynamic model of working decisions of older adults, there are several potential channels through which future health, and therefore health beliefs, may matter for working decisions. First, health may affect the marginal utility of consumption and the disutility of work. Second, it may enter the budget constraint via health-related costs and via wages through effects in productivity. Third, as mentioned before, health affects the probability of survival. These channels could have opposite effects on working decisions. For example, individuals may decide to work longer in order to save more, in cases when they expect to be in good health and therefore anticipate to live long, or in cases when they expect to be in bad health and therefore anticipate higher health-related costs. Hence, the overall effect of health beliefs on working decisions of older adults is an empirical question.

I focus on the extensive margin and study the working decision rule, $p_{it} = 1$. This rule is a function of the information available to individuals when they make their decisions, that is, their information sets Ω_{it-1}.[17] These sets include variables that are observed and unobserved by the econometrician. I consider an environment with heterogeneous and uncertain health dynamics, and focus on a class of dynamic models where the observed variables are given by age t, lagged working decision p_{it-1}, lagged health h_{it-1}, and other controls z_{it}, and the unobserved variables are given by health levels α_i, slope beliefs $\hat{\delta}_{it-1}, \hat{\sigma}^2_{t-1}$, and i.i.d. taste shocks ν_{it}.[18]

Let Φ denote the expected decision rule,

$$\mathbb{E}_\nu(p_{it}|t, p_{it-1}, h_{it-1}, \alpha_i, \hat{\delta}_{it-1}, \hat{\sigma}^2_{t-1}, z_{it}) \quad = \quad \Phi(t, p_{it-1}, h_{it}, \alpha_i, \hat{\delta}_{it}, \hat{\sigma}^2_t, z_{it}), \tag{2}$$

with $\frac{\partial \Phi}{\partial \hat{\delta}_{it}}$ the main object of interest.[19] In estimating this quantity, the main difficulty is the

[17] I assume labor-participation decisions p_{it} are made before health shocks are realized and health h_{it} is observed.

[18] This restriction rules out other forms of heterogeneity, for example, heterogeneity in preferences which we are not able to identify without further assumptions.

[19] The assumptions of the learning model imply the posterior variance $\hat{\sigma}^2_t$ is constant across individuals of the same age t. Given that age is also a relevant determinant of working decisions, I don't have enough variation

presence of time-varying unobserved variables, $\hat{\delta}_{it}$ and $\hat{\sigma}_t^2$. To overcome this difficulty, the key observation is that, under the assumptions of the model, these time-varying unobserved variables can be written as functions of time-invariant unobserved variables (α_i,$\hat{\delta}_{i0}$) and the observed health path ($h_{i1}, \dots h_{iT_i}$). Furthermore, the vector (α_i,$\hat{\delta}_{i0}$) has a known distribution, whose parameters are identified using survival expectations data. Hence, in estimation I integrate out this heterogeneity. The availability of longitudinal data, including data on survival expectations, is key to estimate the effect of health beliefs on working decisions.

5.1 Effects of health beliefs

I study how working decisions p_{it} depend on health and health beliefs by estimating reduced-form equations (2) parametrically, by probit, and non-parametrically, by neural networks. Appendix E.2 provides details of the probit implementation, and Appendix E.3 provides details of the neural network implementation, including the EM algorithm used to deal with the unobserved heterogeneity. In this section, I restrict the analysis to a sample of individuals who are attached to the labor market, defined as individuals with at least 20 years of working experience. Appendix E.1 includes descriptive statistics on the observed control variables z_{it}.

The parametric results are shown in Table 5 column (1). The table shows that beliefs do matter for working decisions of older adults, with a positive and significant coefficient for $\hat{\delta}_{it-1}$. This positive sign implies expecting better health, that is, expecting health to deteriorate more slowly with age, is associated with larger probabilities of working. The table also shows that survival expectations $plive10_{it-1}$ are significant predictors of the probability of working, but that significance holds only while slope beliefs are not accounted for (see columns (2) and (3)). These results are consistent with survival expectations reflecting individuals' beliefs about slope heterogeneity. Thus, once those beliefs are considered, survival expectations do not provide additional information.

Regarding the other controls, as expected, the probability of working decreases with age and increases with better lagged health. Also, the probability of working is larger for individuals who were working the previous period, confirming the dynamic aspect of the working decisions.

I then turn to a more flexible non-parametric estimation that can account for non-linear and heterogeneous effects. The results are shown in Figure 4. The figure shows the average marginal effects of health h_{it-1} on working decision p_{it} by health h_{it-1} and health beliefs $\hat{\delta}_{it-1}$. It shows that health has positive marginal effects which are larger for individuals in

to disentangle these two effects separately; any results would be based on functional-form assumptions alone. Therefore, I focus instead on interpreting the effects of the posterior mean.

Table 5: Parametric results on the probability of working

		(1)		(2)		(3)	
		Coefficient	SE	Coefficient	SE	Coefficient	SE
Age	$t-1$	-0.20***	(0.016)	-0.08***	(0.003)	-0.19***	(0.016)
Lagged work	p_{it-1}	2.03***	(0.018)	2.03***	(0.019)	2.03***	(0.019)
Lagged health	h_{it-1}	0.17***	(0.024)	0.26***	(0.033)	0.18***	(0.046)
Health intercepts	α_i	0.24***	(0.036)	0.07	(0.046)	0.24***	(0.075)
Beliefs mean	$\hat{\delta}_{it-1}$	1.93***	(0.249)			1.90***	(0.499)
Beliefs variance	$\hat{\sigma}^2_{t-1}/\sigma^2_\delta$	-13.85***	(2.048)			-13.33***	(2.102)
Survival expectations	$plive10_{it-1}$			0.11***	(0.031)	0.01	(0.043)
Controls	other vars Ω_{it-1}	Yes		Yes		Yes	
N individuals		14,969		14,718		14,718	
N observations		58,040		55,592		55,592	

Notes: Standard errors are clustered at the individual level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.*

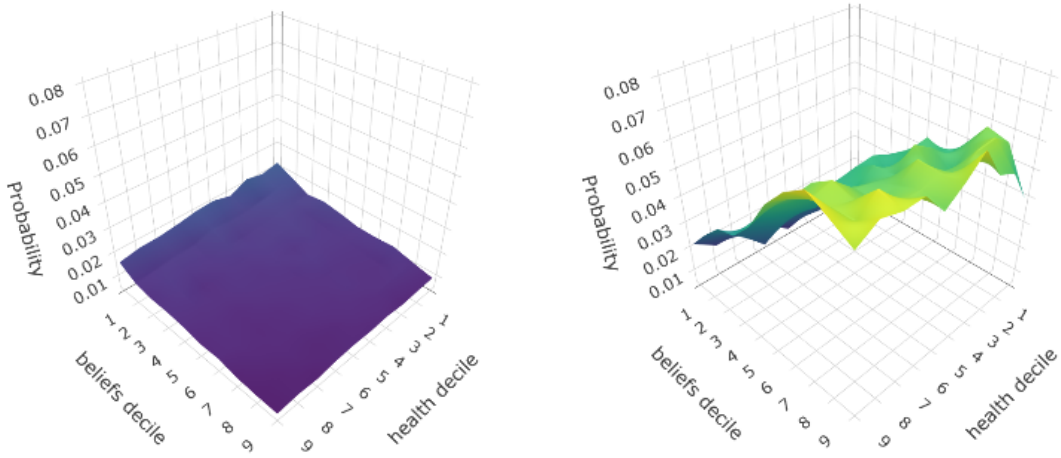
their 50s who were not previously working (panel b). Furthermore, for this group, there is an interaction between beliefs and health in determining future participation decisions, as health has larger marginal effects for individuals with better beliefs, that is, for individuals who believe their health will deteriorate more slowly. This pattern is not observed for individuals age 66 to 75 years old. Figure E1 in the appendix show similar results for the marginal effects of beliefs.

One potential explanation for the pattern observed in Figure 4 is the presence of adjustment costs of going back to work. These adjustment costs could be due, for example, to lower wages caused by the loss of tenure, difficulties in finding jobs or in adapting to new work environments. If going back to work is costly, then the decision to go back to work depends on whether the expected benefits are larger than those costs. In turn, those expected benefits depend on how long individuals expect to remain working. For individuals who expect their health to deteriorate slowly, an improvement in health today will last for several periods, so they anticipate remaining in work. For individuals who expect their health to deteriorate rapidly, an improvement in health today will quickly dissipate, so they anticipate not wanting to work for long. Hence, an improved health today has a different implication depending on health beliefs.

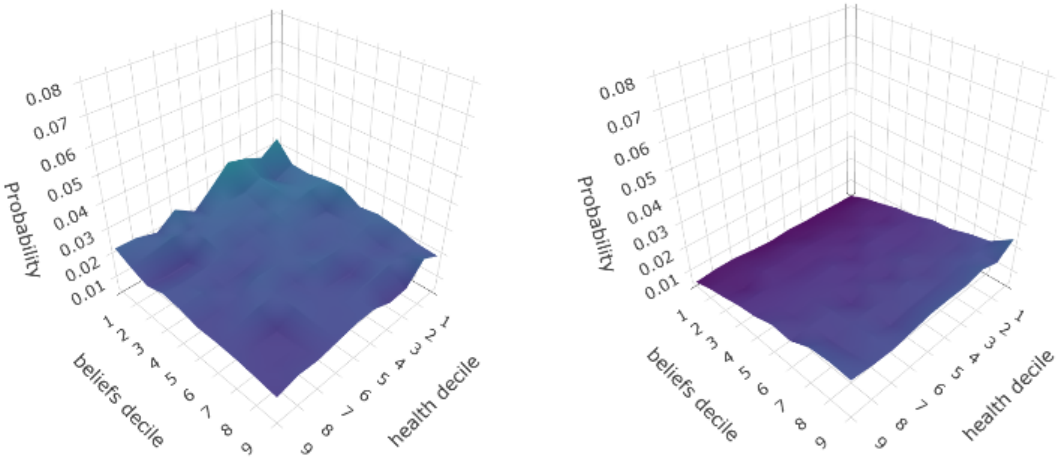
The data-driven approach used for the non-parametric estimation has the advantage of letting the data suggest mechanisms that may be overlooked otherwise, like the adjustment-cost mechanism described before. Overlooking important mechanisms is a source of misspecification in structural models. Hence, the approach in this paper complements such models, by providing a flexible way to identify patterns in the data that suggest mechanisms to incorporate in those models.

Figure 4: Average marginal effect of health h_{it-1} on the probability of working p_{it}

(a) Individuals in their 50s, $p_{it-1} = 1$ (b) Individuals in their 50s, $p_{it-1} = 0$



(c) Individuals 66 to 75 years, $p_{it-1} = 1$ (d) Individuals 66 to 75 years, $p_{it-1} = 0$



Notes: Non-parametric results. In each plot, the x- and y-axis correspond to deciles of health h_{it-1} and mean slope beliefs $\hat{\delta}_{it-1}$ for the corresponding subsample of the plot. The z-axis corresponds to the work response (probability).

5.2 The dual role of health shocks

In the context of uncertain health dynamics, a health shock ϵ_{it-1} has two roles in working decisions. On the one hand, a health shock ϵ_{it-1} affects health h_{it-1}. On the other hand, an uncertain individual cannot perfectly distinguish between ϵ_{it-1} and δ_i within h_{it-1}. Hence, the effect of a shock ϵ_{it-1} on h_{it-1} is partly interpreted as new information regarding δ_i, affecting beliefs $\hat{\delta}_{it-1}$. The total effect of a health shock is a weighted sum of the effects

through these two channels. Using Bayes' rule, we can write,

$$\frac{d\mathbb{P}(p_{it}=1|\Omega_{it-1})}{d\epsilon_{it-1}} = \underbrace{\frac{\partial \mathbb{P}(p_{it}=1|\Omega_{it-1})}{\partial h_{it-1}}}_{\text{health-state channel}} + \underbrace{\frac{\partial \mathbb{P}(p_{it}=1|\Omega_{it-1})}{\partial \hat{\delta}_{it-1}} \overbrace{\frac{(t-1)\hat{\sigma}^2_{t-1}}{\sigma^2_\epsilon}}^{\text{factor}}}_{\text{information channel}}, \tag{3}$$

where the factor term corresponds to the change in the posterior mean $\hat{\delta}_{it-1}$ given a marginal change in ϵ_{it-1}, and it is related to the signal-to-noise ratio of health as a signal.

Empirically, I find that on average less than 1% of the effect of health shocks ϵ_{it-1} in working decisions comes through the information channel, because health shocks have only small effects on beliefs $\hat{\delta}_{it-1}$.[20] This last observation implies that health itself is not enough to quickly reduce the initial bias in beliefs, and hence this bias could have sizable effects on working decisions over time, a question I address next.

5.3 Eliminating the initial bias

In this section, I study how eliminating bias in initial beliefs, that is, setting $\mathbb{E}(\hat{\delta}_{i0}) = \mathbb{E}(\delta_i)$, would affect labor participation of older adults. To do this, I use an impulse-response-function approach, changing only initial beliefs. Over time, this change in initial beliefs translates into changes in posterior beliefs, labor-participation decisions, and decisions regarding assets and health insurance.[21] Note this exercise assumes no other variable changes in response to the change in initial beliefs or to the subsequent changes in participation, assets, or health insurance. Therefore, the exercise relies on these variables capturing the main choices.[22]

Figure 5 shows the results. It shows that the effect on the probability of working has an inverted-U shape. In the early 50s, the effect is small given that individuals are still mostly working. But as people start to retire, their new beliefs imply larger probabilities of working that do not vanish completely over time and remain above 2 percentage points for individuals in their early seventies. Note that, in the baseline scenario, the average probability of working is 34% at age 66 and 17% at age 78; hence, the increment in the figure is not trivial.[23] Furthermore, because this effect results from eliminating a misconception

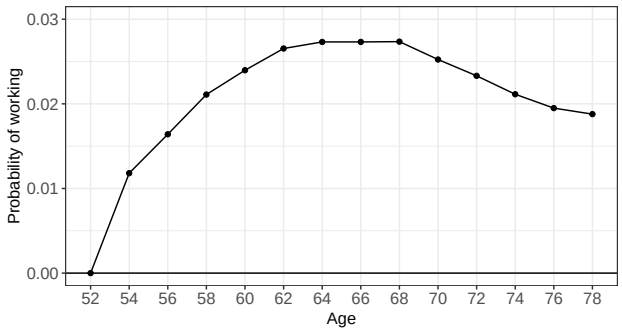
[20] The Online Appendix shows that this overall result also holds when looking at specific ages and past participation decisions.

[21] The effects on these last two variables were also predicted using a neural-network approach.

[22] The results presented in this section use the incomplete prior of the unobserved heterogeneity which already accounts for the information in the health and survival expectations variables. Incorporating the additional information has only a minor effect.

[23] As a reference, using a structural model, French and Jones (2011) find that raising Medicare age from

Figure 5: Impulse-response function to a shift in prior beliefs eliminating overall bias b



Note: Impulse-response function using the subsample of individuals used in estimation that are observed at 52 years old, corresponding to 1,184 individuals.

at the population level, it is an easier policy target that could be addressed by information campaigns, without the need to provide individual-specific information.

Overall, these results show that health beliefs matter for the working decisions of older adults, and that health itself is not a precise enough signal to correct the bias in beliefs. Hence, in Section 6 I look at an information shock that could potentially affect beliefs, and through them, affect working decisions.

6 An information experiment: Blood-based biomarkers as signals of health

In this section, I study the effects of an information shock on expectations and decisions: information on blood-based biomarkers. I exploit that in 2006 the HRS introduced the collection of blood samples for measuring biomarkers. In particular, three biomarkers are measured and individuals are informed of their results: HDL cholesterol, total cholesterol, and blood glucose hbA1c.[24]

To control costs associated with the collection of biomarkers, the HRS randomly split the sample into two halves, and in each wave, it collects these biomarkers in only one of those halves. Hence, this collection scheme provides us with an information experiment, that is, it provide us with exogenous variation in who receives this information. Note, however, that

65 to 67 leads individuals to work an additional 0.074 years over ages 60 to 69, whereas eliminating two years' worth of Social Security benefits increases time spent in the workforce by 0.076 years.

[24] Two other biomarkers are measured: C-reactive protein (CRP), a general marker of systemic inflammation, and Cystatin C, an indicator of kidney functioning. However, individuals are not informed of their results on these two biomarkers; hence, they do not provide additional information.

the goal of the HRS was not to set an experiment, and therefore this experiment is not ideal. An ideal experiment would include a control arm of individuals who get their blood taken and their biomarkers measured but who are not informed of their results. Still, the HRS collection scheme of biomarkers does provide us with exogenous variation that I use in this section.

6.1 Reduced-form approach

I start by estimating the overall effect of receiving this information on individuals' survival expectations and working decisions, using a difference-in-difference type of argument.

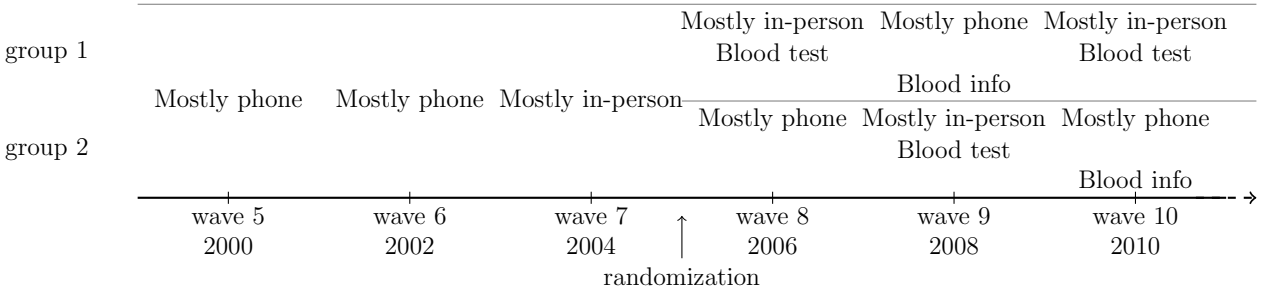
There is one additional difference between the two groups: interview mode. Traditionally, the HRS has been a phone interview,[25] while an in-person interview is required to collect the blood samples. The interview mode could have an effect on individuals' answers, in particular, on questions regarding opinions and expectations. However, the timing of the information provision allows me to separately identify the interview-mode effect from the information effect, because that information is only provided to individuals after the fieldwork.[26] Hence, individuals do not have the information in the wave when the blood is collected, but in the following wave.

Figure 6 presents the timing of the biomarker collection and the information experiment, and it helps us visualize the identification strategy. First, a difference-in-differences analysis using waves 7 and 8 returns the interview-mode effect. Second, a difference-in-differences analysis using waves 7 and 9 returns the interview-mode effect (with the opposite sign) plus the information effect of receiving the additional signal. Hence, we can identify the information effect by adding these two terms.

[25] There are exceptions to this general rule, with in-person interviews conducted for first interviews of new cohorts, people who request in-person interviews, and individuals residing in nursing homes. A shift to in-person interviews in 2004 also occurred in an attempt by the HRS to increase individuals' consent to link their survey responses with administrative data. These differences in interview mode are unimportant for the analysis as long as they are applied in the same way across the two groups.

[26] The results are provided around a month after the survey has ended (see Edwards (2018) for details).

Figure 6: Timing of the biomarker collection and information experiment



Therefore, I estimate the following equation:

$$y_{iw} = \beta_0 + \beta_1 d_{g_i} + \beta_{2w} d_w + \beta_{3w} d_{g_i} \cdot d_w + \epsilon_{iw}, \tag{4}$$

where i denotes an individual and w denotes a wave. I consider two dependent variables separately, survival expectations $plive10_{iw}$ and a binary of working p_{iw}. I estimate these equations using a balanced sample of individuals observed from waves 5 to 9.[27] d_{g_i} is a dummy for the group of individuals set for blood collection in wave 8 (group 1 in Figure 6, with group 2 as the reference category), and d_w are dummies for waves 6 to 9 (wave 5 is the reference category). Hence, the interview-mode effect is given by β_{3w_8}, and the information effect is given by $\beta_{3w_8} + \beta_{3w_9}$, where the interview-mode effects in each group cancel each other out. While we can not test for parallel trends, parallel pre-trends hold if $\beta_{3w_6} = \beta_{3w_7} = 0$. Randomization in the selection of the two groups implies $\beta_1 = 0$.

Table 6 presents the estimation results of equation (4) for both $plive10_{iw}$ and p_{iw} in columns 1 and 4 respectively. For both outcomes, the groups are similar and pre-trends are parallel. When looking at the results for survival expectation, $plive10_{iw}$, the table shows a positive and significant interview-mode effect of 1.77 percentage points and a similar but insignificant information effect of 1.36 percentage points. Though insignificant, this positive sign is aligned with what we already know about beliefs: on average, individuals' beliefs about health and survival are downward biased. Therefore, providing more information shifts those expectations up. When looking at the results for working decisions, p_{iw}, we find no significant effect of interview mode[28] or information. Overall, these results suggest the signal is not large enough to have a significant effect on expectations and decisions.

The table also shows the results by education level. For adults with a college degree,

[27] I use only up to wave 9 because from wave 10 onward the groups are no longer comparable, given that they have been provided information with different timing.

[28] The lack of an interview-mode effect on working decisions is expected, given the more objective nature of working outcomes versus survival expectations.

Table 6: Information and interview-mode effects of biomarker experiment

	Survival expectation ($plive10_{iw}$)			Working decision (p_{iw})		
	All	Below college	College	All	Below college	College
	(1)	(2)	(3)	(4)	(5)	(6)
Group 1 (β_1)	-0.0047	-0.0024	-0.0138	0.0034	0.0055	-0.0077
	(0.007)	(0.008)	(0.012)	(0.011)	(0.012)	(0.022)
Wave 6 (β_{2w_6})	-0.0142	-0.0121	-0.0209	-0.0723	-0.0680	-0.0861
	(0.004)	(0.005)	(0.008)	(0.006)	(0.007)	(0.012)
Wave 7 (β_{2w_7})	-0.0150	-0.0144	-0.0172	-0.1156	-0.1156	-0.1155
	(0.005)	(0.005)	(0.008)	(0.007)	(0.008)	(0.013)
Wave 8 (β_{2w_8})	-0.0641	-0.0612	-0.0737	-0.1645	-0.1577	-0.1865
	(0.005)	(0.006)	(0.009)	(0.007)	(0.008)	(0.015)
Wave 9 (β_{2w_9})	-0.0357	-0.0322	-0.0470	-0.2040	-0.2004	-0.2153
	(0.005)	(0.006)	(0.010)	(0.008)	(0.009)	(0.015)
Group 1, wave 6 (β_{3w_6})	0.0028	-0.0006	0.0137	0.0061	0.0026	0.0175
	(0.006)	(0.007)	(0.011)	(0.008)	(0.009)	(0.016)
Group 1, wave 7 (β_{3w_7})	-0.0027	-0.0024	-0.0033	0.0099	0.0099	0.0100
	(0.006)	(0.007)	(0.011)	(0.009)	(0.011)	(0.018)
Group 1, wave 8 (β_{3w_8})	0.0177	0.0129	0.0331	0.0098	0.0031	0.0315
	(0.007)	(0.008)	(0.012)	(0.010)	(0.012)	(0.020)
Group 1, wave 9 (β_{3w_9})	-0.0042	-0.0112	0.0182	0.0099	0.0119	0.0043
	(0.007)	(0.008)	(0.013)	(0.010)	(0.012)	(0.021)
Constant	0.5397	0.5242	0.5896	0.4877	0.4504	0.6071
	(0.005)	(0.005)	(0.009)	(0.008)	(0.009)	(0.016)
Observations	41,930	31,815	10,115	41,923	31,810	10,113
R-squared	0.004	0.004	0.005	0.021	0.021	0.022
Interview mode effect (a)	0.0177	0.0129	0.0331	0.0098	0.0031	0.0315
P-value	0.0078	0.1038	0.0057	0.3244	0.7910	0.1137
Information effect (a)+(b)	0.0136	0.0016	0.0512	0.0197	0.0149	0.0359
P-value	0.2604	0.9082	0.0199	0.3040	0.5004	0.3521
F test pre-trends	0.4195	0.0577	1.4434	0.5838	0.5031	0.5946
P-value	0.6574	0.9439	0.2364	0.5578	0.6047	0.5519

Notes: Results of estimating equation (4). The sample consists of $N = 8,386$ individuals with non-proxy interviews who are at least 50 years old in wave 8 and who give a valid answer to $plive10_{iw}$ every wave between waves 5 and 9. Seven of these observations do not have information on p_{iw}. Standard errors are clustered at the household level.

both the interview-mode and information effects are larger and significant when looking at survival expectations. These effects are also somewhat larger for their working decisions though still not significant. For adults with less than a college degree, no significant effects are found for any of the two outcomes. These differences by education level suggest that the ability to process the information matters, with more educated adults internalizing the information better.[29]

Appendix F.1 further decomposes group 1 into adults who have a bad biomarker result versus those who do not. As we cannot make the same distinction in group 2,[30] we lack the relevant control groups to identify information effects by the type of signal received (without selection issues). However, this analysis is interesting because it shows that, compared to those who do not receive a bad result, those who receive a bad result already have lower survival expectations and working probabilities by wave 7, two years before the biomarkers are measured. This result suggests that the later group already knew at least some of the information signaled by the biomarkers.

6.2 Model-based approach

In this section, I use the learning model to re-assess the reduced-form results. I predict survival expectations for groups 1 and 2 over waves 8 and 9, including the biomarker results as additional signals when available. That is, in wave 8, when neither group has been informed of their biomarker results, I predict these survival expectations as I did in Section 4, assuming a learning model with health as the only signal. In wave 9, I do the same for predicting survival expectations of group 2. For group 1, who already received their biomarkers results, I predict survival expectations assuming a learning model with biomarkers as additional signals. The goal is to compare these model-base results with the reduced-form results.

For these biomarkers to be valid signals in the learning model, a necessary condition is that they must be correlated with δ_i, the unknown value that is learned over time. To predict survival expectations when biomarkers are available, we need a measure of these correlations as well as their precision as signals.

To estimate those additional parameters, I use the information experiment in a spirit similar to Todd and Wolpin (2006). Specifically, I estimate those parameters by Simulated Method of Moments using *future* data on group 2: their survival expectations in wave 10

[29] A similar analysis for the number of doctor visits finds no interview-mode nor information effects for either group. Still, more educated individuals may be better able to incorporate the new information with the help of their physicians, even if the number of doctor visits remains the same.

[30] One possibility would be to use the biomarker results in wave 9 to attempt the same distinction for group 2. However, using subsequent waves, we can see that biomarker results change from wave to wave, invalidating this strategy.

Table 7: Predicted survival expectations in a model with health and blood glucose as signals

	Number of observations	Predicted survival expectations		
		wave 8	wave 9	wave 9 - wave 8
Control (group 2)	4,852	.458	.454	-.003
Treated (group 1)	5,357	.448	.449	.001
Treated with bad blood glucose	552	.391	.385	-.005
Treated with good blood glucose	3,649	.460	.463	.003
Treated no blood glucose	1,156	.438	.437	-.002

Notes: The sample consists of $N = 10,209$ individuals with non-proxy interviews who are at least 50 years old in wave 8 and who provide a valid answer to $plive10_{iw}$ in waves 8 and 9. Survival expectations are predicted from a model with one signal for the control group (health) and two signals for the treated group (health and blood-glucose results). These two signals are assumed to be independent conditional on individual heterogeneity. The parameters determining the strength of blood glucose as a signal of δ_i come from an estimation using future values of the control group (waves 9 and 10).

and their biomarker results collected in wave 9 (and informed to them between waves 9 and 10). Then, I use these estimates to predict survival expectations of group 1 in wave 9, based on their health and biomarker results. The randomness in the selection of the groups implies the parameters recovered by looking at group 2 must also represent the parameters governing the biomarker signals for group 1.

Table 7 presents the results based on the learning model. According to this table, by having the biomarkers as additional signals, group 1 increases their survival expectations between waves 8 and 9 by 0.4 percentages points relative to the control group. This change in survival expectations is positive but negligible, consistent with the reduced-form results in Table 6. Thus, though a valid signal for health dynamics, biomarker results are not enough to shift beliefs substantially and significantly affect decisions.

7 Conclusion

This paper documents individual-level heterogeneity in health dynamics among older adults and studies how individuals' beliefs about their own health dynamics affect their working decisions.

I start by showing evidence that health dynamics are indeed heterogeneous, in particular, in their slope of health with age, with health deteriorating faster for some older adults than for others. This heterogeneity helps explain why as the population ages the variance of health increases, a fact ignored by traditional models of health.

Motivated by this evidence, I turn to the question of how much individuals know about their own health dynamics. I assume individuals are Bayesian learners and update their beliefs as they see their health changing with age. As future survival depends on future health and, therefore, on health dynamics, I leverage data on subjective survival expectations to estimate the learning parameters. I find that indeed individuals are uncertain about how fast their health will deteriorate with age, and that they are also negatively biased, that is, on average, they believe their health will deteriorate faster than the average rate in the population.

For older adults, health is an important determinant of many decisions. When health dynamics are uncertain, beliefs about future health may also be important, in particular for decisions with dynamics consequences. In this paper, I study working decisions of older adults, focusing on the extensive margin. I find beliefs matter for these decisions, and that expecting health to deteriorate more rapidly is associated with lower probabilities of working. Furthermore, if we could eliminate the bias in beliefs, participation would increase by up to 2 percentage points, an effect that would last beyond traditional retirement ages.

Additionally, flexible estimation using neural networks shows there is an interaction between health and beliefs about health in the working decisions of older adults. For individuals in their 50s who are not working, improving health has a larger effect on the probability of working when beliefs about health are also better, that is, when the improvement in health is expected to last longer, suggesting the presence of adjustment costs of returning to work.

Overall, the previous results suggests there is room for policies to affect labor-participation decisions by shifting individuals' beliefs about their health. In the last part of the paper, I look at one such policy: the provision of information on blood-glucose and cholesterol levels. I take advantage of the randomization of the collection and provision of such information within the HRS to analize the data. The results show this additional information has negligible effects on survival expectations and working decisions. This negligible result is due to a small effect of the information on beliefs.

In future work, it will be of interest to study other policies which could have an effect on beliefs and decisions. For example, policies that provide aggregate information (aiming at correcting the bias in beliefs at the population level) or more individualized information.[31] In the case of the HRS, policies could include providing information about biomarkers on kidney function and systemic inflammation, as well as genetic information, all already collected in the survey but not shared with individuals.

[31] Information policies have been studied in other settings, for example, Delavande and Kohler (2015) and Bates (2020). Information policies have also been studied in the context of surveys, for example, Armona, Fuster, and Zafar (2018) and Wiswall and Zafar (2014).

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APPENDIX

A Overview of sub-samples

In this section, I provide an overview of the sub-samples used for the main analyses in the paper, starting from the main sample of individuals 50 years and older with a personal interview between waves 4 and 12.

Table A1: Sub-samples

Section 3 Heterogeneity	Table 2	Individuals observed at 50 years old and their consecutive waves
	Figure 1	Individuals observed at 50 years old with over 9 consecutive waves
	Figure 2	Individuals observed at 66 years old with over 9 consecutive waves
Section 4 Uncertainty	Table 3	Subset of 2,000 individuals with 8 consecutive waves
	Table 4 (a)	Subset of 2,000 individuals with 8 consecutive waves
	Table 4 (b)	Individuals up to 65 years old who are also asked *plive*75
Section 5 Working decisions	Table 5	Individuals with at least 20 years of working experience
	Figure 4	Individuals with at least 20 years of working experience
	Figure 5	Individuals with at least 20 years of working experience, observed at 52 years old
Section 6 Information experiment	Table 6	Individuals interviewed in waves 5 to 9 with a valid answer in *plive*10
	Table 7	Individuals interviewed in waves 8 and 9 with a valid answer in *plive*10

B Health measurement

In this section, I describe the health measures and the method used to construct the summary health variable h_{it}. Table B1 presents descriptive statistics on these health-related measures.

Table B1: Descriptive statistics on health measures

Measure	Observations	Mean	SD	Min	Max
Number of chronic conditions	156,968	1.83	1.34	0	7
Self-assessed health	156,862	2.86	1.11	1	5
Body mass index (kg/m^2)	154,602	27.89	5.81	7	83
Eyesight in general	156,768	2.85	1.01	1	6
Eyesight at a distance	156,833	2.57	1.01	1	6
Eyesight up close	156,822	2.75	1.04	1	6
Hearing	156,869	2.63	1.09	1	5
Pain	156,550	0.63	0.97	0	3
Difficulties in ADLs regarding mobility	156,748	1.09	1.45	0	5
Difficulties in ADLs of large muscles	156,737	1.28	1.33	0	4
Difficulties in other ADLs	151,923	0.40	0.66	0	2
Summary health variable h_{it}	148,866	5.19	0.67	2.94	6.15

Notes: Descriptive statistics on health measures and summary health variable. The sample includes 156,976 observations and comprises 31,210 individuals interviewed in person, in wave 4 or later, that are 50 years old or older. Chronic conditions include high blood pressure, heart attack, diabetes, stroke, lung disease, arthritis, and cancer. The categories for self-assessed health, eyesight and hearing variables are 1. excellent, 2. very good, 3. good, 4. fair, 5. poor, with an extra category 6. legally blind for eyesight variables. The categories for the level of pain are 0. no pain, 1. mail pain, 2. moderate, 3. severe. ADL stands for activities of daily living. ADLs regarding mobility include walk 1 block, several blocks, across room, climb one flight of stairs, several flight of stairs. ADLs involving large muscles include push or pull large object, sit for two hours, get up from chair, stoop kneel or crouch. Other ADLs include carry 10 lbs and reach arms.

In constructing the summary health variable, I consider the following model. Let M_{ijt} be the j^{th} observed measure of unobserved health h_{it}, for $j = 1, \dots 11$. I assume a linear factor model structure,

$$M_{ijt} = \mu_j + \lambda_j h_{it} + \epsilon^h_{ijt}, \tag{A1}$$

where ϵ^h_{ijt} is a measurement error, and intercepts μ_j and loadings λ_j are invariant in age t. To fix the location and scale of h_{it}, I set the intercept and loading of the number of chronic conditions to 0 and 1 respectively. I estimate equation (A1) by confirmatory factor analysis

(CFA)[1], assuming classical measurement errors. Estimates of latent health h_{it} are obtained by minimizing the generalized sum of squares deviations of the factor from their true values.

Table B2 presents the results. The table shows all the coefficients are significant and have the expected signs. It also shows the percentage of the variance of each measure M_{ijt} that is explained by health h_{it}, with variables regarding difficulties in ADLs having the largest R-squared, consistent with their common use in the assignment of many health-related benefits.[2]

Table B2: CFA results for health measurement

Measure of health	Coefficients		R-squared
	Intercept	Loading	
Number of chronic conditions$^{(a)}$	0	1	0.29
Self-assessed health	8.111	-1.017	0.44
Body mass index	37.491	-1.845	0.05
Eyesight in general	5.667	-0.546	0.15
Eyesight at a distance	5.135	-0.497	0.13
Eyesight up close	5.418	-0.517	0.13
Hearing	4.807	-0.421	0.08
Pain	4.827	-0.811	0.36
Difficulties in ADLs regarding mobility	9.396	-1.606	0.64
Difficulties in ADLs of large muscles	8.928	-1.476	0.63
Difficulties in other ADLs	3.790	-0.654	0.50

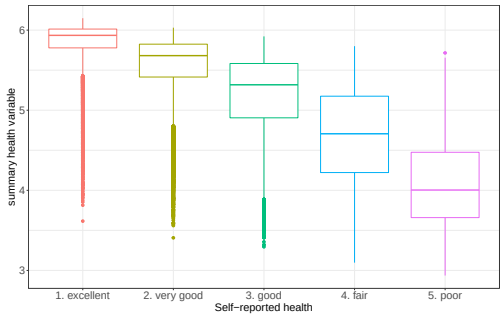
Note: The sample consists of 148,666 observations from Table B1 that have information on all health measures. (a) The first measure corresponds to 7 minus the number of chronic conditions, so larger values represent better health. For this measure, the intercept and loading are fixed to 0 and 1, respectively. All other coefficients are significant at 1%.

Figure B1 presents a box plot for h_{it} per value of self-assessed health. The figure shows both measures are highly correlated, but h_{it} captures more variation than what is captured by the self-assessed measure, specially among those with worse health. As discussed in Appendix C, the heterogeneity in health dynamics is robust to the use of self-assessed health instead of the summary variable h_{it}.

[1] The values of h_{it} predicted by CFA are highly correlated with the values predicted by principal component analysis.

[2] For example, difficulties in ADLs are considered by Medicare in the provision of long-term care services.

Figure B1: Summary health variable h_{it} by category of self-assessed health



Notes: Sample of 148,866 observations from Table B2.

C Estimation of health dynamics

In this section, I provide details of the estimation of the health dynamic process, the full set of results, and some additional robustness checks.

C.1 Baseline results

I assume the health and survival processes are given by,

$$h_{it} = \rho h_{it-1} + \alpha_i + \delta_i \cdot t + \tau \cdot t^2 + \epsilon_{it} \tag{A2}$$

$$S_{it} = \mathbb{1}\{\gamma h_{it-1} + \theta_0 + \theta_1 \cdot t + \theta_2' x_i + \eta_{it} \geq 0\} S_{it-1}, \tag{A3}$$

where $\epsilon_{it} \sim N(0, \sigma_\epsilon^2)$ and $\eta_{it} \sim N(0,1)$ are independent over time and independent of each other at all leads and lags. x_i is a vector of demographic characteristics including gender, race, hispanic ethnicity, and education. Furthermore,

$$\begin{pmatrix} \alpha_i \\ \delta_i \end{pmatrix} \Big| x_i, h_{i0} \sim N\left(\begin{pmatrix} \mu_\alpha + \nu_\alpha' x_i + \omega_\alpha h_{i0} \\ \mu_\delta + \nu_\delta' x_i + \omega_\delta h_{i0} \end{pmatrix}, \begin{bmatrix} \sigma_\alpha^2 & \phi\sigma_\alpha\sigma_\delta \\ \phi\sigma_\alpha\sigma_\delta & \sigma_\delta^2 \end{bmatrix} \right). \tag{A4}$$

Let Θ be the vector of parameters of this random-coefficients model. The MLE estimator $\hat{\Theta}$ solves

$$\max_{\Theta} \sum_{i=1}^{N} log\left(\int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \prod_{t=1}^{T_i} \mathbb{P}\left(h_{it}, S_{it} | h_{it-1}, S_{it-1} = 1, x_i, \alpha, \delta\right) \cdot \phi(\alpha, \delta | x_i, h_{i0}) d\alpha d\delta \right).$$

Table C1 presents the MLE results for three different specifications. The middle panel shows the results for the baseline specification, that includes survival and allows for heterogeneous slopes. As mentioned in the main text, these results show there is heterogeneity in

levels and slopes, both of them uncorrelated. There is also no correlation between health at age 50, h_{i0}, and δ_i, but h_{i0} is correlated with α_i. Furthermore, next-period survival is strongly dependent on health. In terms of other observable characteristics, individuals with low levels of education have worse health, health deteriorates faster for non-white individuals, and the probabilities of survival are higher for women and Hispanic individuals. For completeness, the left panel shows the results allowing for unobserved slope heterogeneity but ignoring survival, while the right panel shows the results including survival but ignoring unobserved slope heterogeneity.[3] Columns (5) and (6) show that if we ignore slope heterogeneity, we overestimate the persistence of health.

C.2 Robustness checks

In this appendix, I study three robustness checks to the baseline model of health dynamics, whose results are shown in Table C2. First, I estimate the model allowing for heteroskedastic errors in the health equation, with a variance that varies linearly with age. The results (in the left panel) show that this heteroskedasticity does not explain away the heterogeneity in health slopes δ_i. Second, I estimate the model allowing for the heterogeneous levels and slopes to directly affect survival. The results (in the middle panel) show there is no such (joint) effect, hence, survival does not provide additional information on δ_i. Finally, I use self-assessed health h_{it}^{SAH} instead of the summary variable h_{it}, and estimate an ordinal model. The results (in the right panel) show the presence of slope heterogeneity is robust to using this measure alone.

D Uncertain dynamics and learning model details

In this section of the appendix, I provide details regarding the beliefs about δ_i and its relation with subjective survival expectations $plive10_{it}$.

D.1 Bayesian updating equations

According to Bayes' rule, individuals' beliefs about their health slopes δ_i (henceforth, slope beliefs) are normally distributed, $N(\hat{\delta}_{it}, \hat{\sigma}_t^2)$, with mean and variance defined recursively

[3] The model on the right panel allows for heterogeneity in slopes by observed characteristics, but it does not allow unobserved heterogeneity in slopes.

Table C1: MLE results on health and survival under different assumptions

	Heterogeneous slopes without survival eq		Heterogeneous slopes with survival eq		Homogeneous slopes with survival eq	
	Coefficient (1)	Pvalue (2)	Coefficient (3)	Pvalue (4)	Coefficient (5)	Pvalue (6)
ρ	0.225	0.000	0.223	0.000	0.366	0.000
τ	0.001	0.087	0.001	0.119	0.001	0.108
μ_α	0.968	0.000	0.955	0.000	0.781	0.000
$\nu_{\alpha female}$	-0.029	0.132	-0.029	0.131	-0.024	0.163
$\nu_{\alpha white}$	0.026	0.338	0.027	0.335	0.018	0.458
$\nu_{\alpha hispanic}$	0.004	0.909	0.005	0.889	-0.001	0.973
$\nu_{\alpha less\_HS}$	-0.134	0.000	-0.134	0.000	-0.120	0.000
ω_α	0.599	0.000	0.603	0.000	0.492	0.000
μ_δ	-0.060	0.012	-0.057	0.018	-0.051	0.000
$\nu_{\delta female}$	0.006	0.146	0.006	0.136	0.005	0.198
$\nu_{\delta white}$	0.015	0.007	0.015	0.008	0.013	0.011
$\nu_{\delta hispanic}$	0.010	0.196	0.010	0.199	0.006	0.390
$\nu_{\delta less\_HS}$	-0.003	0.677	-0.003	0.624	0.001	0.896
ω_δ	0.000	0.956	0.000	0.962		
σ_α	0.235	0.000	0.235	0.000	0.212	0.000
σ_δ	0.042	0.000	0.043	0.000		
ϕ	-0.030	0.741	-0.033	0.714		
σ_ϵ	0.266	0.000	0.266	0.000	0.285	0.000
γ			0.583	0.001	0.640	0.000
ι_1			-0.277	0.334	-0.422	0.125
ι_2			0.044	0.986		
ι_3			0.029	0.306	0.036	0.287
ι_4			0.241	0.601		
θ_0			0.529	0.326	0.514	0.336
θ_1			-0.178	0.136	-0.193	0.092
$\theta_{2female}$			0.259	0.002	0.255	0.002
θ_{2white}			0.019	0.847	0.029	0.758
$\theta_{2hispanic}$			0.317	0.079	0.311	0.078
θ_{2less_HS}			-0.106	0.305	-0.114	0.267
N alive observations	8,901		8,901		8,901	
N dead observations	0		112		112	
N individuals	1,671		1,671		1,671	
-Log likelihood	2,498.6		3,027.6		3,067.6	

Notes: The sample consists of 1,671 individuals observed at age 50 and their following consecutive observations until death or loss of follow up, for a total of 8,901 observations. The demographic variables include an indicator for female, for white race, for hispanic ethnicity, and for education less than high school. Standard errors are clustered at the individual level.

by

$$\frac{\hat{\delta}_{it}}{\hat{\sigma}_t^2} = \frac{\hat{\delta}_{it-1}}{\hat{\sigma}_{t-1}^2} + \frac{(h_{it} - \rho h_{it-1} - \alpha_i - \tau t^2)t}{\sigma_\epsilon^2} \quad \text{(A5)}$$

$$\frac{1}{\hat{\sigma}_t^2} = \frac{1}{\hat{\sigma}_{t-1}^2} + \frac{t^2}{\sigma_\epsilon^2}. \quad \text{(A6)}$$

Table C2: MLE robustness checks

Dependent variable:	Health h_{it}		Health h_{it}		Self-assessed health h_{it}^{SAH}	
	Coefficient (1)	Pvalue (2)	Coefficient (3)	Pvalue (4)	Coefficient (5)	Pvalue (6)
ρ	0.225	0.000	0.223	0.000	0.230	0.000
τ	0.001	0.088	0.001	0.119	0.012	0.000
μ_α	0.961	0.000	0.955	0.000	-1.185	0.000
$\nu_{\alpha female}$	-0.030	0.122	-0.029	0.131	-0.005	0.951
$\nu_{\alpha white}$	0.027	0.330	0.027	0.335	0.242	0.009
$\nu_{\alpha hispanic}$	0.003	0.928	0.005	0.889	-0.266	0.047
$\nu_{\alpha less\_HS}$	-0.134	0.000	-0.134	0.000	-0.603	0.000
ω_α	0.601	0.000	0.603	0.000	1.151	0.000
μ_δ	-0.059	0.015	-0.057	0.018	-0.054	0.182
$\nu_{\delta female}$	0.006	0.139	0.006	0.136	0.029	0.089
$\nu_{\delta white}$	0.015	0.008	0.015	0.008	-0.009	0.647
$\nu_{\delta hispanic}$	0.010	0.193	0.010	0.199	0.060	0.040
$\nu_{\delta less\_HS}$	-0.003	0.661	-0.003	0.624	0.019	0.406
ω_δ	0.000	0.986	0.000	0.962	-0.043	0.000
σ_α	0.234	0.000	0.235	0.000	0.970	0.000
σ_δ	0.042	0.000	0.043	0.000	0.137	0.000
ϕ	-0.025	0.776	-0.033	0.714	-0.257	0.004
σ_ϵ	0.266	0.000	0.266	0.000	1	-
$\sigma_{t\epsilon}$	0.000	1.000				
γ	0.494	0.000	0.583	0.001	0.402	0.000
θ_0	-0.103	0.707	0.529	0.326	1.371	0.000
θ_1	-0.083	0.000	-0.178	0.136	-0.101	0.000
$\theta_{2female}$	0.244	0.005	0.259	0.002	0.164	0.043
θ_{2white}	0.025	0.793	0.019	0.847	0.034	0.711
$\theta_{2hispanic}$	0.248	0.263	0.317	0.079	0.404	0.018
θ_{2less_HS}	-0.096	0.345	-0.106	0.305	-0.076	0.457
ι_1			-0.277	0.334		
ι_2			0.044	0.986		
ι_3			0.029	0.306		
ι_4			0.241	0.601		
O_2					1.712	0.000
$O_3 - O_2$					1.711	0.000
$O_4 - O_3$					2.063	0.000

Notes: Same sample as in Table C1. In columns (1) and (2), $Var(\epsilon_{it}) = \sigma_\epsilon^2 + t \cdot \sigma_{t\epsilon}^2$. In columns (3) and (4) the survival equation (A3) is replaced by $S_{it} = \mathbb{1}\{\gamma h_{it-1} + \iota_1\alpha_i + \iota_2\delta_i + \iota_3 \cdot t \cdot \alpha_i + \iota_4 \cdot t \cdot \delta_i + \theta_0 + \theta_1 \cdot t + \theta_2' x_i + \eta_{it} \geq 0\} S_{it-1}$. In columns (5) and (6) the health equation (A2) is replaced by $\tilde{h}_{it}^{SAH} = \rho h_{it-1}^{SAH} + \alpha_i + \delta_i \cdot t + \tau \cdot t^2 + \epsilon_{it}$, where $\sigma_\epsilon^2 = 1$, $\tilde{h}_{it}^{SAH}$ is a latent variable, h_{it}^{SAH} is the observed ordinal value with larger values represent better health, and O_2, O_3, O_4 are the threshold values. Standard errors are clustered at the individual level.

Equation (A5) shows the posterior mean is a weighted average of the prior mean $\hat{\delta}_{it-1}$ and the signal derived from health h_{it}, with weights that depend on precision. The more certain an individual is to begin with (lower $\hat{\sigma}_{t-1}^2$), the more weight he gives to what he

already knows, namely, the prior. The more precise health is as a signal (lower σ_ϵ^2), the more weight is given to its information. Equation (A6) shows precision increases over time, and increases more when the signal is more precise, that is, when health is less noisy (lower σ_ϵ^2) and when individuals are older.

Alternatively, we can write these equations as

$$\hat{\delta}_{it} = \hat{\delta}_{it-1} + K_t \cdot \hat{\zeta}_{it} \tag{A7}$$

$$\hat{\sigma}_t^2 = (1 - K_t \cdot t)\hat{\sigma}_{t-1}^2, \tag{A8}$$

where $K_t = \frac{\hat{\sigma}_{t-1}^2 \cdot t}{\hat{\sigma}_{t-1}^2 \cdot t^2 + \sigma_\epsilon^2} = \frac{t\hat{\sigma}_t^2}{\sigma_\epsilon^2}$, $K_t \leq 1$, and $\hat{\zeta}_{it}$ is individual i's perceived innovation in health at period t, $\hat{\zeta}_{it} = h_{it} - \mathbb{E}(h_{it}|\Omega_{it-1}) = h_{it} - \rho h_{it-1} - \alpha_i - \hat{\delta}_{it-1} \cdot t - \tau t^2$.

D.2 Formula for $plive10_{it}$

Let Ω_{it} be the information set of individual i after observing his health up to period t. From equation (A2), we can write for $l > t$

$$h_{il} = \underbrace{\rho^{l-t} h_{it} + \alpha_i \sum_{k=0}^{l-t-1} \rho^k + \tau \sum_{k=0}^{l-t-1} (l-k)^2 \rho^k}_{\text{known under } \Omega_{it}} + \underbrace{\delta_i \sum_{k=0}^{l-t-1} (l-k)\rho^k + \sum_{k=0}^{l-t-1} \rho^k \epsilon_{i(l-k)}}_{\text{unknown under } \Omega_{it}}. \tag{A9}$$

Let s denote the reference age asked in $plive10_{it}$. Then, by equations (A3) and (A9),

$$plive10_{it} \equiv \mathbb{P}(S_{is} = 1|\Omega_{it}) = \prod_{l=t}^{s-1} \mathbb{P}(S_{il+1} = 1|S_{il} = 1, \Omega_{it}) = \prod_{l=t}^{s-1} \Phi\left(\frac{M_{itl}}{W_{tl}^{1/2}}\right), \tag{A10}$$

where

$$M_{itl} = \gamma\left(\rho^{l-t} h_{it} + \alpha_i \sum_{k=0}^{l-t-1} \rho^k + \hat{\delta}_{it} \sum_{k=0}^{l-t-1} (l-k)\rho^k + \tau \sum_{k=0}^{l-t-1} (l-k)^2 \rho^k\right) + \theta_0 + \theta_1(l+1) + \theta_2' x_i$$

$$W_{tl} = \gamma^2 \hat{\sigma}_t^2 \left(\sum_{k=0}^{l-t-1} (l-k)\rho^k\right)^2 + \gamma^2 \sigma_\epsilon^2 \sum_{k=0}^{l-t-1} \rho^{2k} + 1$$

Note that M_{itl} and W_{tl} are functions of h_{it}, α_i, $\hat{\delta}_{it}$, $\hat{\sigma}_t^2$, x_i and the parameters of the model Θ. Hence, $plive10_{it} = plive10_{it}(\alpha_i, h_{it}, \hat{\delta}_{it}, \hat{\sigma}_t^2, x_i; \Theta)$.

D.3 Proof of proposition 4.1

Applying the same derivation as in equation (A10) and defining $c_t^1 = \frac{W_{t+1t+2}^{1/2}}{\gamma}$ and $c_t^2 = \frac{W_{tt+2}^{1/2}}{\gamma}$, which are constant across individuals, we can write

$$\begin{aligned} c_t^1\Phi^{-1}(b_{it+1}^{(1)}) - c_t^2\Phi^{-1}(b_{it}^{(2)}) &= \underbrace{\rho\big(h_{it+1} - \rho h_{it} - \alpha_i - \hat{\delta}_{it}(t+1) - \tau(t+1)^2\big)}_{\text{due to persistence}} \\ &\quad + \underbrace{(\hat{\delta}_{it+1} - \hat{\delta}_{it})(t+2).}_{\text{due to learning}} \end{aligned} \tag{A11}$$

Using equations (A7) and (A8),

$$\begin{aligned} c_t^1\Phi^{-1}(b_{it+1}^{(1)}) - c_t^2\Phi^{-1}(b_{it}^{(2)}) &= \Big(\rho + K_{t+1}(t+2)\Big)\hat{\zeta}_{it+1} \\ &= \Big(\rho + K_{t+1}(t+2)\Big)\Big(h_{it+1} - \frac{1}{\gamma}\big(W_{tt+1}^{1/2}\cdot\Phi^{-1}(b_{it}^{(1)}) + \theta_0 + \theta_1(t+1) + \theta_2' x_i\big)\Big). \end{aligned}$$

Rearranging the terms, conditional on h_{it}, $b_{it}^{(1)}$ and $b_{it}^{(2)}$ (belonging to Ω_{it}),

$$Cov(\Delta B_{it+1}, \Delta h_{it+1}) = G(\lambda, t, \Theta)\cdot Var(\Delta h_{it+1}) \equiv F(\lambda, t, \Theta),$$

where $w_t = \frac{c_t^2}{c_t^1}$, $G = \frac{\rho+(t+2)K_{t+1}}{c_t^1}$, $\frac{\partial F_1}{\partial \lambda} \geq 0$ (strictly greater than zero when $\lambda > 0$) and $Var(\Delta h_{it+1})$ does not depend on λ. ■

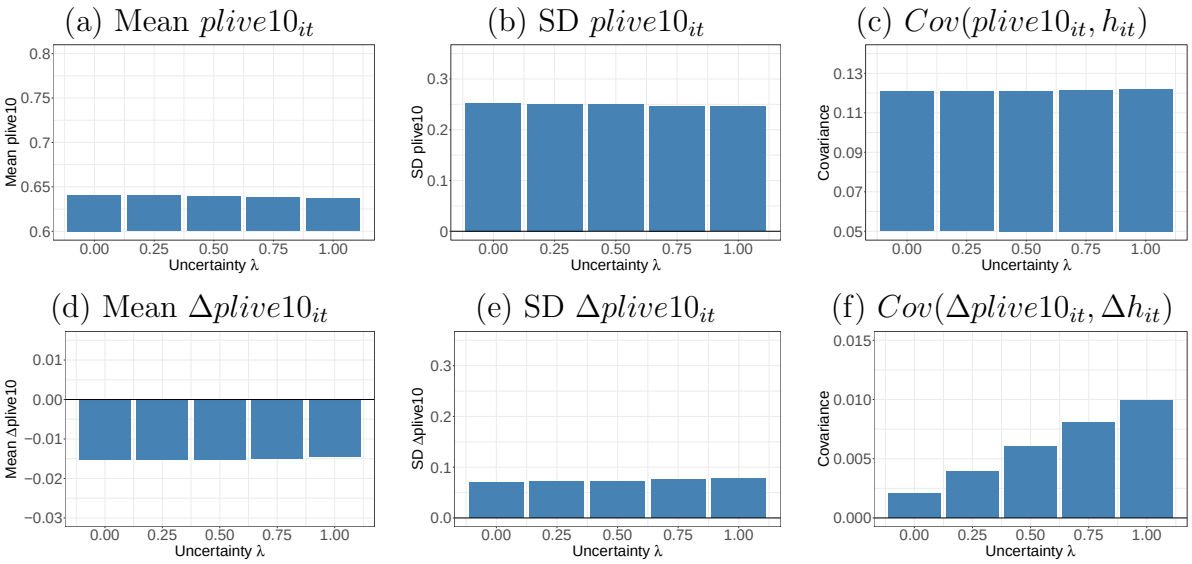
D.4 Simulation results

In this section, I show via simulation that the intuition of proposition 4.1 extends to the available data on survival expectations. In the exercise, I first simulate individual-level heterogeneity (α_i, δ_i) and health h_{it}, according to equations (A2), (A3) and (A4). Then, for different values of the uncertainty parameter λ, I simulate initial beliefs $(\hat{\delta}_{i0}, \hat{\sigma}_0^2)$ assuming $b = 0$.[4] I update those beliefs over time and construct $(\hat{\delta}_{it}, \hat{\sigma}_t^2)$ using the simulated h_{it} and the Bayesian updating equations (A5) and (A6). Finally, I use these simulated beliefs, to construct survival expectations $plive10_{it}$ according to equation (A10). Figure D1 presents the results. In the figure, each graph depicts the uncertainty parameter λ in the x-axis, and a simulated moment in the y-axis. The six graphs correspond to the six moments used for estimation. The top row considers moments in levels, and the bottom row considers moments in differences. The figure clearly shows that, as with data on expectations on survival

[4] I also assume $Cov(\alpha_i, \hat{\delta}_{i0}) = Cov(\alpha_i, \delta_i)$, and set it to zero according to the results of Section 3.

rates, the covariance between changes in health and changes in expectations is increasing in the uncertainty parameter λ, and therefore, a key moment for identification.

Figure D1: Simulated moments of $plive10_{it}$ by uncertainty λ in data-generating process



Notes: Moments in simulated data following the structure of the available data in the HRS. In each figure, the x-axis shows the value of the uncertainty parameter λ used in the data-generating process. In all cases, the bias parameter b is set to zero.

E Working decisions

E.1 Descriptive statistics on additional controls

Table E1 present descriptive statistics on the additional controls of working decisions, z_{it}.

E.2 Probit details

Let ω_{it-1} be the vector of variables in Ω_{it-1} relevant for the working decision p_{it}: age t, past working decision p_{it-1}, past health h_{it-1}, health beliefs $\hat{\delta}_{it-1}$ and $\hat{\sigma}^2_{t-1}$, individual heterogeneity in health α_i, and other controls z_{it-1} described in Table E1. Let t_0 be the age at which individual i is first observed in the data.[5] Then, the likelihood of the vector

[5] Note t_0 and T are individual specific, though I omit that index for ease of notation.

Table E1: Descriptive statistics on additional controls of working decisions

Variable	Mean	SD	Min	Max
Panel (a)				
Age	66.26	7.49	52	80
Work	0.38	0.49	0	1
Female	0.52	0.5	0	1
Education: less than high school	0.20	0.40	0	1
Education: some college	0.55	0.50	0	1
White	0.84	0.37	0	1
Hispanic	0.06	0.24	0	1
Marital Status: married	0.70	0.46	0	1
Marital Status: separated or divorced	0.12	0.33	0	1
Marital Status: widow	0.14	0.35	0	1
Number of household members	2.15	1.03	1	12
Total number of years worked	39.79	9.17	20	68
Spouse works	0.28	0.45	0	1
Spouse has health insurance	0.17	0.38	0	1
Income from pension	6.08	50.49	0	10000
Income from Social Security	6.65	5.95	0	58.3
Wealth	366.51	730.98	-1585.01	10000
Health insurance: employer covering retirement	0.14	0.35	0	1
Health insurance: employer not covering retirement	0.07	0.25	0	1
Health insurance: employer (already 65)	0.17	0.37	0	1
Health insurance: government	0.47	0.5	0	1
Health insurance: other	0.11	0.31	0	1
Panel (b)				
Income from work	30.51	39.83	0	1190.68
Tenure	14.31	12.4	0	66.1
Self-employed	0.22	0.42	0	1
Occupation: managerial	0.16	0.36	0	1
Occupation: professional	0.21	0.4	0	1
Occupation: sales	0.12	0.32	0	1
Occupation: clerical	0.16	0.37	0	1
Occupation: services	0.14	0.35	0	1
Occupation: farming, mechanics, construction, operators	0.22	0.41	0	1
Occupation: FF.AA.	0.00	0.02	0	1
Job requires physical effort	0.17	0.38	0	1
Job requires lifting heavy loads	0.07	0.25	0	1
Job requires stooping or kneeling	0.13	0.34	0	1
Job requires good eyesight	0.68	0.47	0	1
Job involves lots of stress	0.16	0.37	0	1

Note: Descriptive statistics on variables used in estimating working decisions in section 5. The sample consists of observations from 12,623 individuals who have participated in the labor market for at least 20 years, excluding missing values in any of these variables. Panel (a) comprises 48,607 observations, and panel (b) comprises 18,415 observations from working periods. Income and wealth variables are measured in thousands of 2002 dollars. Wealth variables are capped at $10 million dollars.

$(p_{it_0}, \dots p_{iT})$ conditional on $\omega_{it_0}, \dots \omega_{iT}$ is given by:

$$L_i^c = \tilde{L}_{it_0}^c \prod_{t=t_0+1}^{T} \left[\Phi(\beta' \omega_{it-1})^{p_{it}} \cdot \left(1 - \Phi(\beta' \omega_{it-1})\right)^{1-p_{it}} \right],$$

where $\tilde{L}^c_{it_0}$ is the probability of the initial condition p_{it_0}, which I model as a probit function of $(t_0, h_{it_0}, \hat{\delta}_{it_0}, \hat{\sigma}_{t_0}, \alpha_i, z_{it_0})$.

This likelihood depends on variables that are unobserved by the econometrician, namely, slopes beliefs $\{\hat{\delta}_{it}, \hat{\sigma}^2_t\}^T_{t=t_0}$ and heterogeneity in health levels α_i. By equations (A5) and (A6), these time-varying unobserved variables can be written as a function of time-varying observed health $(h_{it_0} \dots h_{iT})$ and time-invariant unobserved variables, namely, beliefs at t_0 $(\hat{\delta}_{it_0}, \hat{\sigma}^2_{t_0})$ and α_i. Hence, I write instead the likelihood of $(p_{it_0}, \dots p_{iT})$, conditional on variables observed by the econometrician, $(t_0, T, h_{it_0} \dots h_{iT}, plive10_{it_0}, \dots plive10_{iT}, z_{it_0} \dots z_{iT})$, by integrating out this time-invariant unobserved heterogeneity,[6]

$$L_i \quad = \quad \int L^c_i \cdot f(\alpha_i, \hat{\delta}_{it_0} | t_0, T, h_{it_0}, \dots h_{iT}, plive10_{it_0} \dots plive10_{iT}, z_{it_0}, \dots z_{iT})$$

I added in the conditioning set the variables regarding survival expectations, which according to equation (A10) provide information on individuals slopes beliefs. The distribution within the integral has no closed form solution, given that surviving up to t_0 adds additional restrictions on the underlying individual heterogeneity. Hence, in practice, I approximate this integral using draws from this distribution gotten by Markov chain Monte Carlo (MCMC).

E.3 Neural network details

Neural networks provide flexible tools for estimation (Goodfellow, Bengio, and Courville (2016)). They are universal approximators, because they are capable of approximating any measurable function to any desired degree of accuracy (Hornik, Stinchcombe, and White (1989)). In the case of a binary outcome, and under some particular specifications, a neural network resembles a maximum likelihood estimation with logistic errors, where the probability of success is a complex non-linear index of the inputs. As mentioned by Farrell, Liang, and Misra (2021), we can think of neural networks as a type of non-parametric or sieve estimation whereby the basis functions are learned from the data, hence allowing for greater flexibility.

As in the probit case, I also need to account for the fact that some of the input variables are unobserved by the econometrician. These unobserved variables are slope beliefs $(\hat{\delta}_{it-1}, \hat{\sigma}^2_t)$ and heterogeneous health levels α_i. Though they are time-varying variables, they can be written as functions of time-invariant unobserved variables $(\hat{\delta}_{i0}, \alpha_i)$ and the observed health path $(h_{i1}, \dots h_{iT_i})$ of each individual.[7] Thus, following a standard likelihood approach,

[6] This expression uses that $\hat{\sigma}^2_t$ is constant across individuals of the same age, and it assumes no other form of unobserved heterogeneity (an assumption that could be relaxed by assuming a distribution for it).

[7] This relationship depends also on the parameters of the health process $(\rho, \sigma^2_\epsilon)$ and the parameters of beliefs

the objective is to maximize the log of the likelihood integrating out this time-invariant unobserved heterogeneity. To do so, I follow the insight of EM-type algorithms (Dempster, Laird, and Rubin (1977)).

Let θ be the parameters governing an outcome variable, in this case, working decisions. When underlying heterogeneity exists, we estimate θ by maximizing a likelihood that integrates out that heterogeneity. In this context, EM-type algorithms provide us with two key insights. First, the parameter θ that maximizes the integrated log-likelihood also maximizes an alternative specification using the posterior distribution given the outcome variable. Formally, let η_i denote the vector of unobserved heterogeneity, $f(\eta_i)$ its prior distribution, and $f(\eta_i|p_i;\theta)$ its posterior distribution given the outcomes p_i. The first insight of EM-type algorithms is to note that

$$arg\max_{\theta} log \int \mathbb{P}(p_i|\eta_i;\theta) f(\eta_i) d\eta_i \quad = \quad arg\max_{\theta} \int log(\mathbb{P}(p_i|\eta_i;\theta)) f(\eta_i|p_i;\theta) d\eta_i. \quad \text{(A12)}$$

The expression on the right-hand side is simpler to use. However, because this posterior distribution depends on θ, it is unknown. Thus, the second insight of EM-type algorithms is to solve the problem for θ iteratively: in iteration k, the E step uses θ_{k-1} to update the posterior distribution of the heterogeneity, and the M step estimates θ_k by maximizing the right-hand side of equation (A12), using that posterior.

I use this same iterative logic as a convenient implementation for maximizing the integrated likelihood under a neural-network approach. In this case, the E step is done by Markov chain Monte Carlo (MCMC) and provides draws from the posterior distribution of $(\alpha_i, \hat{\delta}_{i0})$ given working decisions p_i.[8] Those draws, along with individuals' health histories, are used to simulate the inputs $(\hat{\delta}_{it}, \hat{\sigma}^2_t, \alpha_i)$ and to expand the data. Then, the M step estimates θ by using a neural network on the expanded data.[9] I start this iterative process at an M step using an incomplete posterior: the distribution of $(\alpha_i, \hat{\delta}_{i0})$ conditional on the health history $(h_{i1}, \ldots h_{iT_i})$ and the history of survival expectations $(plive10_{i1}, \ldots plive10_{iT_i})$. This distribution is incomplete because it does not condition on the working decisions, but it does already include the heterogeneity information contained in the health and expectations variables.

(b and λ), but it does not depend on the parameters defining the relation between working decisions and state variables.

[8] MCMC uses the likelihood of p_i given $(\alpha_i, \hat{\delta}_{i0})$ from the previous-iteration M step and the prior distribution of $(\alpha_i, \hat{\delta}_{i0})$.

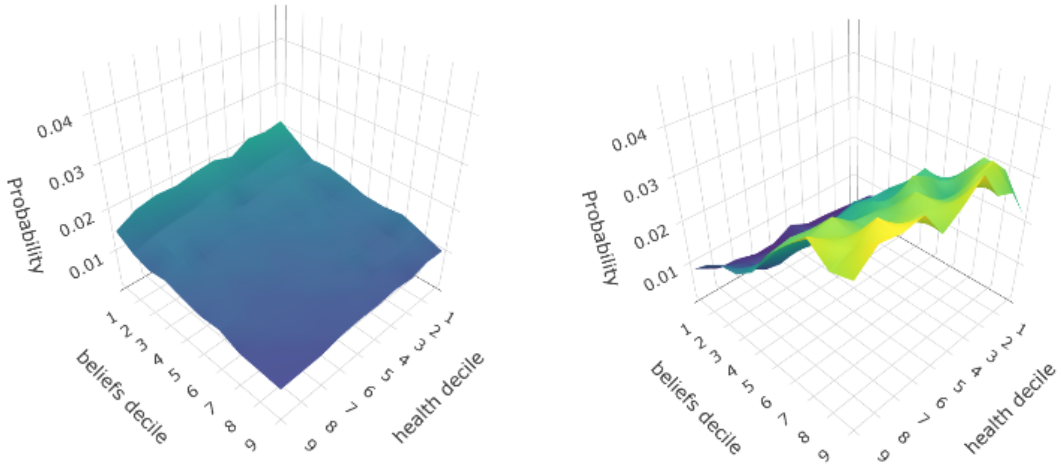
[9] The standard EM algorithm is known to converge, as the likelihood increases in each step of the sequence. This property does not hold in this case, given the lack of uniqueness of the solution. Therefore, the approach is not aimed at getting at the unique solution, but as a convenient implementation.

E.4 Additional results

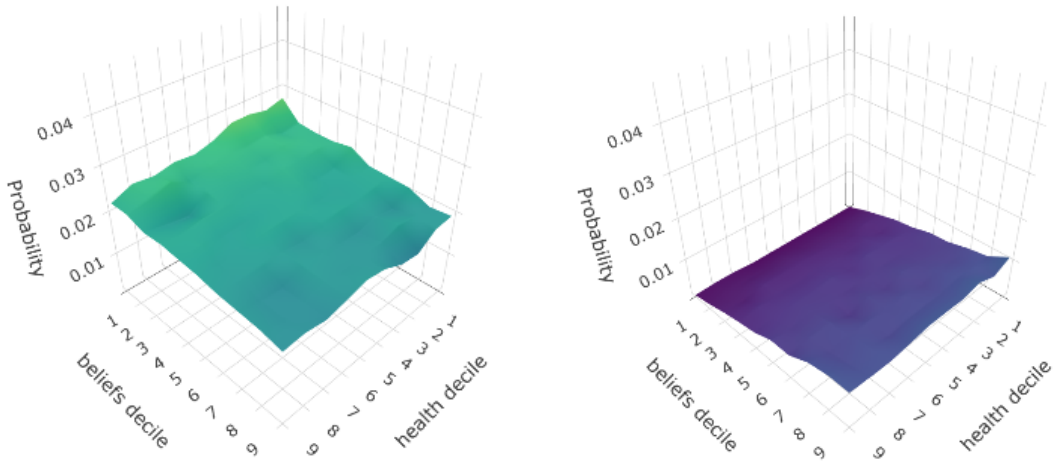
Figure E1 shows the average marginal effects of health beliefs $\hat{\delta}_{it-1}$ on working decision p_{it} estimated by a neural network.

Figure E1: Average marginal effect of health beliefs $\hat{\delta}_{it-1}$ on the probability of working p_{it}

(a) Individuals in their 50s, $p_{it-1} = 1$ (b) Individuals in their 50s, $p_{it-1} = 0$



(c) Individuals 66 to 75 years, $p_{it-1} = 1$ (d) Individuals 66 to 75 years, $p_{it-1} = 0$



Notes: Non-parametric results. In each plot, the x- and y-axis correspond to deciles of health h_{it-1} and mean slope beliefs $\hat{\delta}_{it-1}$ for the corresponding subsample of the plot. The z-axis corresponds to the work response (probability).

F Biomarkers as signals of health

F.1 Distinguishing bad vs good biomarkers results

I use the biomarker results of wave 8 to further distinguish group 1 in two subgroups: individuals whose biomarker results are within normal levels (good results) and those whose results are outside normal levels (bad results). Hence, I estimate the following equation:

$$y_{iw} = \beta_0 + \beta_1 d_{g_i} + \beta_2 d_{b_i} + \beta_{3w} d_w + \beta_{4w} d_{g_i} \cdot d_w + \beta_5 d_{g_i} \cdot d_w \cdot d_{b_i} + \epsilon_{iw}, \quad \text{(A13)}$$

where as before, d_{g_i} is a dummy for group 1 (those who get their blood collected in wave 8), and d_w are dummies for waves. The new variable d_{b_i} is a dummy for the subgroup of individuals in group 1 that gets a bad result in any of the 3 tests. That is, total cholesterol equal or above 240 mg/dL, HDL cholesterol below 40 mg/dL, or blood glucose hbA1c equal or above 6.4%. Note that in this equation, the interpretation of the coefficients is not the same as in equation (4). For example, β_1 is now comparing the individuals in group 1 who get good results versus all individuals in group 2, whether or not their (unobserved) test results are good or bad. Thus, β_1 is not a fair comparison. Consequently, the interest in this equation lies not on the comparison between groups 1 and 2, but on comparing the differences between group 1 individuals that receive good versus bad results.

Table F1 presents the results of estimating equation (A13). The results suggest the information contained on bad test results is at least partially already known by individuals, as they have lower survival expectations even before receiving this information, and their labor participation is also decreasing ahead of time.

Table F1: Biomarkers experiment distinguishing bad vs good test results

	Survival expectation ($plive10_{iw}$)	Working decision (p_{iw})
Group 1 (d_{g_i})	-0.0039	-0.0066
	(0.007)	(0.012)
Group 1, bad results (d_{b_i})	-0.0037	0.0427
	(0.011)	(0.018)
Wave 6 (d_{w_6})	-0.0142	-0.0723
	(0.004)	(0.006)
Wave 7 (d_{w_7})	-0.0150	-0.1156
	(0.005)	(0.007)
Wave 8 (d_{w_8})	-0.0641	-0.1645
	(0.005)	(0.007)
Wave 9 (d_{w_9})	-0.0357	-0.2040
	(0.005)	(0.008)
Group 1, wave 6 ($d_{g_i} \cdot d_{w_6}$)	0.0058	0.0075
	(0.007)	(0.009)
Group 1, wave 7 ($d_{g_i} \cdot d_{w_7}$)	0.0015	0.0189
	(0.007)	(0.010)
Group 1, wave 8 ($d_{g_i} \cdot d_{w_8}$)	0.0223	0.0214
	(0.007)	(0.011)
Group 1, wave 9 ($d_{g_i} \cdot d_{w_9}$)	-0.0005	0.0164
	(0.007)	(0.011)
Group 1, bad results, wave 6 ($d_{g_i} \cdot d_{b_i} \cdot d_{w_6}$)	-0.0125	-0.0059
	(0.010)	(0.013)
Group 1, bad results, wave 7 ($d_{g_i} \cdot d_{b_i} \cdot d_{w_7}$)	-0.0175	-0.0381
	(0.010)	(0.016)
Group 1, bad results, wave 8 ($d_{g_i} \cdot d_{b_i} \cdot d_{w_8}$)	-0.0194	-0.0488
	(0.011)	(0.017)
Group 1, bad results, wave 9 ($d_{g_i} \cdot d_{b_i} \cdot d_{w_9}$)	-0.0156	-0.0278
	(0.011)	(0.017)
Constant	0.5397	0.4877
	(0.005)	(0.008)
Observations	41,930	41,923
R-squared	0.005	0.021
% of group 1 with bad results	23.6	23.6

Notes: Estimation results from equation (A13). The sample consists of $N = 8,386$ individuals with non-proxy interviews who are at least 50 years old in wave 8, and who give a valid answer to $plive10_{iw}$ every wave between waves 5 and 9. Of these, 7 observations do not have information on p_{iw}. Standard errors are clustered at the household level.