The Evolution of Health over the Life Cycle *

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Abstract

Recent studies have identified health dynamics and health shocks as major sources of risk over the life cycle. Health has implications for many economic variables including asset accumulation, labor supply, and income and wealth inequality. Despite the importance of health in economic studies, there is no unified objective measure of health status. In this paper we propose such a measure: *frailty*, defined as the cumulative sum of all adverse health indicators observed for an individual. There is overwhelming evidence in the gerontology literature that this simple measure is a strong predictor of mortality and other health outcomes. We construct a frailty index for individuals in the PSID, HRS and MEPS separately and make the following three observations. One, our constructed frailty index is remarkably consistent across the three datasets in terms of persistence, and dynamics of its distribution. This is in contrast to the most commonly used measure of health, self-reported health status. Two, individuals' health decays at a substantially faster pace over the lifecycle when measured by frailty as opposed to self-reported health status. Three, health status is more persistent when measured by frailty as opposed to self reported health status. We estimate a dynamic process for frailty over the life cycle and show that an important driver of increasing inequality in health with age is dispersion in growth rates of frailty across individuals. Our frailty measure and dynamic process can be used by economists to study the evolution of health status over the life cycle and its implications.

Keywords: *health, inequality, life-cycle, idiosyncratic rick, heterogeneous health profiles*
JEL Classification numbers: I14, D15, C33

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1 Introduction

Recent studies have identified health dynamics and health shocks as major sources of risk over the life cycle. Health has implications for many economic variables including asset accumulation, labor supply, and income and wealth inequality.[1] However, despite the importance of health in economic studies, there is no unified objective measure of health status. Instead, most studies use survey responses on individuals' self-assessed health status (see for example, De Nardi et al. (2010), Kopecky and Koreshkova (2014), among others).[2] This assessment is by definition subjective and, as we argue, it is often not consistent across different surveys (see section 2 and appendix A.1). Moreover, due to the nature of the survey, the 'self reported health status' (SRHS from now on) is always a discrete (category) variable. For example, individuals are asked to describe their health status by reporting a number between 1 to 5, with 1 meaning 'excellent', and 5 meaning 'poor' health (and 'very good', 'good' and 'fair' in between). Therefore, an individual who reports the number 1 is considered healthier than an individual who reports number 2. However, this information does not help us understand how much healthier is a 1 relative to a 2.

In this paper we construct a single, continuous variable called *frailty index* (or $frailty$ for short) that can summarize individual health. Frailty index is simply the accumulated sum of all adverse health events that has occurred to an individual. Our construction is inspired by and based on findings in gerontology literature.[3] The idea behind the construction of frailty index is as follows. As individuals age, they accumulate health problems. These health problems can range from symptoms to clinical signs and laboratory abnormalities to diseases and disabilities. These health problems are referred to as *deficits*. Mitnitski et al. (2001) and Mitnitski et al. (2002) have demonstrated that health status can be represented by combing deficits in an index variable, called frailty index. Mitnitski et al. (2005) and Goggins et al. (2005) find that frailty index is comparable between databases even when list of deficits used to construct the index do not coincide. They also find the frailty index to be a better predictor of mortality and institutionalization than age.

We follow the guidelines described in Searle et al. (2008) to construct frailty index for individuals using three different datasets: The Panel Study of Income Dynamics (PSID), Health and Retirement Study (HRS) and Medical Expenditure Panel Survey (MEPS). All three datasets contain rich set of survey questions on various aspects of individual health conditions. In each case we normalize frailty index to be a variable between 0 and 1. Therefore, a frailty index of 0.2 means that a person has accumulated 20 percent of all possible deficits.

We start by comparing and contrasting frailty index to SRHS. All the three datasets that we use collect responses about self reported health status by asking individuals to describe their own assessment of their health using a number between 1 and 5 which correspond to 'excellent', 'very good' , 'good', 'fair', and 'poor' health. We document three patterns. One,

[1] See De Nardi et al. (2017), Blundell et al. (2017), O'Donnell et al. (2015) and De Nardi et al. (2010) among many others.

[2] An alternative approach is to track a list of variables that contain various health events for individual. See Amengual et al. (2017) .

[3] See Searle et al. (2008); Rockwood and Mitnitski (2007); Rockwood et al. (2007); Mitnitski et al. (2001, 2005); Kulminski et al. (2007a,b); Goggins et al. (2005); Woo et al. (2005), and among others.

at each age, poorer SRHS is associated with higher frailty index on average. However, there is substantial variation in the average frailty index over lifetime, even for a given self reported health status. Two, As individuals get older, the fraction of those with bad (good) health rise (decline) faster according to frailty index relative to SRHS. Three, frailty index is more persistent than SRHS.

Our first and second findings suggest that individuals adjusts their standards of good or bad health as they age. This leads to a bias in SRHS towards categorizing people in better health condition than they actually are. In contrast, frailty index is an objective measure that is based on actual events, diagnosis, etc. Therefore, it is less prone to these biases. Moreover, unlike SRHS, the level of frailty index has a meaning in that it corresponds to the number (or fraction) of deficits accumulated. Another advantage of frailty over SRHS is consistency across different datasets. As we show in appendix A.1 the distribution of SRHS evolves very differently in MEPS than PSID and HRS, whereas the dynamics of frailty distribution across all three datasets is very similar (this is despite the fact that the set of the deficit variables that we use to construct frailty index is not exactly the same across these three databases).

Next, we use the frailty index, to measure the evolution of individual health over the life cycle. Specifically, we estimate the stochastic process that determines both the distribution and dynamics of frailty over the life cycle using PSID (we report results for HRS in the appendix). We show that, after controlling for common observables, the variance of frailty increases with age. We choose a statistical process that has the ability to match this property. To this end, we draw from the earnings literature. The macro literature on estimating earnings processes has favored models in which the residual consists of an AR(1) process plus a transitory shock (see, for example, Storesletten et al. (2004) - STY from now on). Guvenen (2009) revisits models that allow for individuals to have heterogeneous earnings profiles. We follow Guvenen (2009) and explore two versions of the model. In the *restricted* version, we assume that there is a fixed effect in log frailty but individuals do not have different ex-ante frailty profiles. Under this view the increasing variance of log frailty with age is driven by persistent (perhaps even unit root) shocks. The *unrestricted* version allows for heterogeneous frailty profiles. This version would be consistent with the view that individuals face different frailty profiles during the adult life due to differences in their genes and/or the investments made in their health as children.

Each of these specifications has strong implications for the effect of frailty heterogeneity on welfare, insurance, and behavior. Therefore, we explore the ability of each version to replicate key properties of the data. In particular, we observe that in the data the variance of residual frailty rises with age. We also observe that autocovariances of residual frailty decline with lags at each age.[4] We find that the unrestricted specification of the model that allows for ex-ante heterogeneity in frailty profiles matches these features in the data better. Therefore, our preferred model is the unrestricted one.

Based on the unrestricted model we find that more than 60 percent of dispersion in frailty index at age 55 is due to heterogenous profiles and the rest can be attributed to the effect of persistent shocks that individuals experience over their lifetime (this figure rises to more

[4]Similar pattern is documented for variance and covariance of log earnings. See, for example, Guvenen (2009)

than 85 percent for 75 year olds).

This paper is closely related to the quantitative literature that study health dynamics and its implications over the life cycle. For instance, Pijoan-Mas and Rios-Rull (2014) study the impact of the evolution of health and socioeconomic characteristics with age on expected longevities. An interesting finding in their study is that some key socioeconomic variables such as education and wealth have little predictive power for two-year survival rates once health status is known. De Nardi et al. (2017) estimate the health dynamics of high school males, allowing for history-dependence of health dynamics. They then quantitatively evaluate the lifetime consequences of bad health in a structural life-cycle model. Most of these studies use SRHS to measure health status. As we explained previously, SRHS has several drawbacks compared to objective measures, especially for understanding the dynamics of health status over the life cycle.[5] Our paper contributes to this literature by providing an alternative approach to measure health dynamics over the life cycle. We show that our frailty-based approach is arguably better than the existing SRHS-based approach for capturing health dynamics and its implications over the life cycle.

There are a number of papers in the literature that use objective health condition variables to measure health status.[6] For instance, Gilleskie et al. (2017) use body mass to measure health status and studies the impact of health status on wages in a life-cycle model. Amengual et al. (2017) construct a objective discrete measure of health based on the information about Activities of Daily Living (ADL) and Instrumental Activities of Daily Living (IADL) in the HRS data, and they estimate health dynamics of the elderly using a panel Markov switching model.[7] By using objective indicators of health conditions, these studies avoid the disadvantages of subjective self-reported health measures. However, as argued by Blundell et al. (2017), the objective health indicators used in these studies provide an incomplete view of health since they only cover a subset of health conditions. Our paper differs from these studies because, following the gerontology literature, we construct a frailty index using a large number of objective frailty indicators. The frailty index serves as a comprehensive summary of an individual's overall health status.

This paper is also related to the literature on estimating earnings processes and medical expenses processes.[8] Our statistical analysis of the underlying stochastic processes for frailty index draw heavily from this literature, which has favored simpler models so that the estimated processes can be easily incorporated into quantitative life-cycle models. Following this tradition, we model the frailty residual as an AR(1) process plus a transitory shock. In addition, inspired by the findings in Guvenen (2009), we emphasize a version of the statistical model with heterogenous frailty profiles, and find that it is preferred to the one without this dimension of heterogeneity. Our favorable findings for the heterogenous-profile process

[5] Though it has been documented in the literature that SRHS is highly correlated with objective measures and it is a strong predictor of mortality risk (see, for example, Idler and Benyamini (1997), Van Doorsaler and Gerdtham (2002)), the limitations of SRHS, in particular for life-cycle analysis, still remain.

[6] See Bound (1991), Smith (2004), Gilleskie et al. (2017), and Amengual et al. (2017).

[7] It is worth noting that Amengual et al. (2017) argue that their discrete measure has an advantage over a continuous measure as the latter cannot be included in structural models. We would argue that it is in fact a disadvantage as it is less flexible than a continuous measure like ours. One can always discretize a continuous process but not so obvious how to go the other way.

[8] See, for example, Storesletten et al. (2004) and Guvenen (2009) for the estimation of earnings processes, and Hubbard et al. (1995) and French and Jones (2004) for estimating medical expenses processes.

are consistent with the empirical evidence provided in Case et al. (2002), who find that children from households with different income experience different dynamics of health over their adulthood.

It is worth mentioning that Poterba et al. (2017) also construct an objective health measure for HRS respondents using a similar set of variables to ours and principle component analysis. They find that difficulties with ADLs/IADLs and self-reported measures of health have the highest weights. Our constructed frailty measure is similar in the sense that it is a summary statistics that captures the variations in a collection of indicators variables. The advantage of frailty index, aside from its simplicity, is that it is directly corresponds to the notion of deficit accumulations. Moreover, its construction is clear, transparent and comparable across datasets. Finally, there is overwhelming evidence from gerontology literature that this index is strong predictor of various health outcomes such as mortality and entry to nursing homes.

The rest of the paper is organized as follows. In Section 2, we construct frailty index from several main datasets and compare it to SRHS. In Section 3, we estimate the underlying stochastic process for frailty index. In Sections 4 and 5 We estimate the relationships between frailty index and some other key variables in Section 6, and conclude in Section 7.

2 Frailty Index

As individuals age they develop an increasing number of health problems, functional impairments, and abnormalities. Some of these conditions are rather mild (e.g., reduced vision) while some are serious (e.g., cancer). However, as the number of these conditions rises, the person's body becomes more frail and vulnerable to adverse outcomes. This is the idea behind *the frailty index* proposed by Mitnitski et al. (2001). The index is constructed by summing the number of health conditions a person has accumulated at each age and taking the ratio of this sum to the number of potential conditions considered. As an example, a person that has 10 conditions out of a list of 40 is assigned a frailty index of 0.25. Despite its simplicity, the frailty index is strongly correlated with the risk of death, institutionalization and worsening health status (see Searle et al. (2008); Rockwood and Mitnitski (2007); Rockwood et al. (2007); Mitnitski et al. (2001, 2005); Kulminski et al. (2007a,b); Goggins et al. (2005); Woo et al. (2005)).[9]

We follow guidelines described in Searle et al. (2008) to construct frailty indices for samples of individuals in three different datasets: the Panel Study of Income Dynamics (PSID), Health and Retirement Study (HRS) and Medical Expenditure Panel Survey (MEPS). All three datasets contain a rich set of survey questions on various aspects of individual health conditions. We include the following sets of variables in our calculations:[10]

- Restricted activity, difficulty in Activities of Daily Living (ADL) and Instrumental ADL (IADL): such as difficulty eating, dressing, walking across room, etc.
- Cognitive impairment: such as immediate word recall, backwards, counting, etc.

[9] Remarkably, these findings are consistent across data-sets and the potential list of conditions considered.
[10] See the Appendix for a complete list of variables used for each dataset.

- Medical diagnosis/measurement: such as high blood pressure, diabetes, heart disease, cancer, high BMI, etc.

We call each of these conditions/impairments/diagnosis a *deficit*. The lists of deficit variables available in each dataset do not exactly match. For example, in the HRS, we have 37 deficit variables. In the PSID we are limited to only 30.[11] However, as Searle et al. (2008) point out, it is well documented by multiple studies that the exact number and composition of variables included in the calculation of the frailty index does not matter. The resulting rate of change in the frailty index and its relationship with health outcomes, such as mortality, is the same (especially when at least 30 variables are included, see Kulminski et al. (2007a)).[12]Detailed information on the sample size, data coverage and list of deficit variables for each dataset can be found in the Appendix.

2.1 The Frailty Index and Self-reported Health Status

The frailty index is by construction an objective measure of health that is easily comparable across different surveys (in the same way that medical expenditure or labor earnings is comparable). Similar to self-reported health status (henceforth, SRHS), the frailty index is a ranking of individuals by health status (higher frailty means poorer health). Moreover, in contrast to SRHS, the magnitude of the difference in the frailty index between two individuals is also informative about how much healthier one is relative to the other.[13] Another desirable feature of the frailty index is that it is essentially a continuous variable. This feature is particularly useful in statistical analysis or economic modeling.

These qualitative features are not the only advantages of the frailty index over SRHS. The frailty index also gives a more accurate picture of how an individual's health evolves with age. We illustrate this point by comparing SRHS and frailty using the PSID data. Similar patterns emerge if we use the HRS data (only available for older individuals) and the MEPS data (only one frailty observation per individual). The comparison in HRS and MEPS are reported in Appendix.

We start by looking at how the frailty index evolves over an individual's lifetime and how it relates to SRHS. Figure 1 reports the average frailty index by 5-year age groups and SRHS category ('excellent' ,'very good' ,'good' ,'fair' ,'poor'). The figure shows that, in every age group, better SRHS is associated with a lower frailty index. This is expected. As we argued above, the frailty index is a sum of accumulated heath deficits. A person who has higher accumulation of deficits is more prone to bad health outcomes and therefore is more likely to have a poorer assessment of his/her own health. However, even within the same SRHS category there is substantial variation in frailty with age. For example, a 30-34 year old with

[11]Most, but not all, of these 30 variables are also included in HRS.

[12]Searle et al. (2008) write: "To be clear, it does not matter if study A considered 40 deficits from set X of deficits and study B considered 60 deficits from set Y of deficits; the estimates from each (e.g. the rate of deficit accumulation, the relationship between deficit accumulation and mortality, or the limit to deficit accumulation) appear to be similar. This finding suggests that frailty is a real phenomenon, which is a property of a biologically complex system."

[13]This is confirmed by numerous studies that have shown its power in predicting mortality and other health outcomes. For example, see Searle et al. (2008); Mitnitski et al. (2001, 2005) and among others for more details.

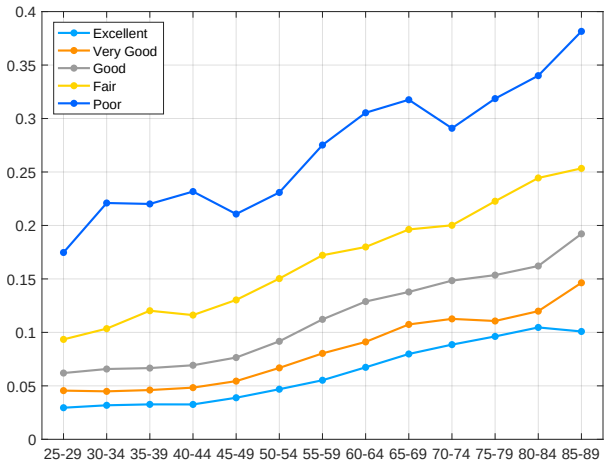


Figure 1: Average frailty index by self reported health status and age. Source: authors' calculation using PSID.

'fair' SRHS has a much lower frailty index on average than a 70-74 year old with the same SRHS. Using SRHS leads to the conclusion that these two individuals have the same health status, even though by our objective measure, the 70-74 year old has accumulated far more health deficits and is in poorer health. To summarize:

> **Fact 1:** At each age, poorer self-reported health status is associated with a higher frailty index on average. However, within self-reported health status categories, there is substantial variation in the average frailty index by age.

Figures 7a and 7b in the Appendix show that similar patterns hold in the HRS and MEPS.

Next, we compare the evolution of the frailty distribution with the evolution of the SRHS distribution. To facilitate this comparison we partition individuals in each age group into five frailty categories ('excellent', 'very good', 'good', 'fair', and 'poor'). The cutoff values of frailty that determine which category is assigned are age-independent and set such that the distribution of individuals across frailty categories and SHRS categories is the same for the 25-29 year-old age group.[14] For example, the fraction of 25-29 year-olds with SHRS of 'excellent' is 28%. Assuming a monotonic relationship between SHRS and frailty, we set the cutoff value for 'excellent' frailty such that 28% of 25-29 year-olds are also in the 'excellent' frailty category. The resulting cutoff value of frailty is 0.074. At each age, individuals with a frailty index value less than 0.074 are assigned to the frailty category 'excellent'. Next, we find the cutoff value for the 68th percentile (68% of 25-29 year-olds have a SRHS of 'excellent' or 'very good'). In each age group, anyone whose frailty is below this cutoff that is not in the 'excellent' category, is assigned to the frailty category 'very good'. The other two cutoffs are chosen accordingly at the 93rd and 99th percentiles and determine the assignment of individuals to the 'good', 'fair' and 'poor' frailty categories.

The shaded areas in Figure 2 show how the distribution of SRHS evolves with age. Each shaded area is the fraction of individuals in each SRHS category by age. As expected, the fraction of individuals with 'excellent' or 'very good' SRHS falls with age (going from 70% for

[14] Small differences between the age 25-29 distributions are due to discreteness.

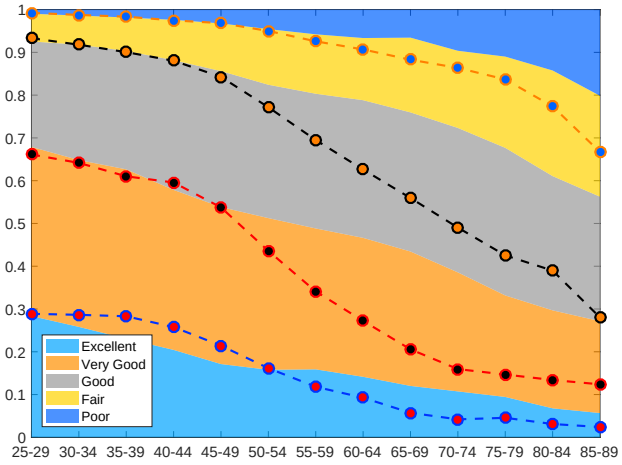


Figure 2: Distribution of health status by age. The colored areas show the fraction of individuals by SRHS at each age. The dashed line shows the fraction of health status according to frailty index. Source: authors' calculation using PSID.

age group 25-29 to 30% for age group 85-89). At the same time, the fraction of individuals with 'fair' or 'poor' SRHS increases with age (going from less than 10% for age group 25-29 to more than 40% for age group 85-89). There is not much change in the share of the middle group (those with SRHS of 'good').

We track how many individuals at each age fall within these fixed frailty index cutoffs. This is demonstrated in Figure 2 using the dashed lines. As we see the overall pattern is very similar to that of SRHS. The important difference, however, is that the decline in 'excellent'/'very good' shares and rise in 'fair'/'poor' shares happens more rapidly with age relative to that of SRHS. For example, up to the 45 to 49 age group both measures give very similar distribution of health status. More than half of individuals in 45 to 49 age group have 'excellent' or 'very good' health according to both SRHS. The same fraction also has frailty index that is below the cut off for 'very good' health group. Also, close to 14% of individuals have 'fair' or 'poor' health according to both measures. However, there is a departure for the older age groups. By age 70 to 74, only 20% of individuals have frailty index low enough to fall below the 'excellent' or 'very good' cut offs. But 38% of them have SRHS of 'excellent' or 'very good'. A the same time 48% of individuals have frailty index higher that the cut off for 'fair' or 'poor' health, while only 27% of them SRHS of 'fair' or 'poor'. We interpret these patterns as evidence that SRHS underestimates the measures decline in health status.

–To summarize:

Fact 2: As individuals get older, the fraction of those with bad (good) health rise (decline) faster according to frailty index relative to SRHS.

Figures 8a and 8b in the Appendix A.1 presents similar patterns in HRS and MEPS. However, the differences in rate of change in distributions is more pronounced, both in HRS and MEPS.

Finally, we compare the persistence in health status according to SRHS with that of frailty index. To do this we calculate conditional probability of transitioning between health status categories for SRHS and frailty index. For frailty index we use categories that we construct according to the procedure outlined above.

Table 1 shows transition probabilities between different health status. Left panel is the transition probability between different SRHS categories. The right panel is the transition probabilities between different health group constructed using frailty index. Notice that the diagonal values are all higher for frailty index relative to SRHS. This is an evidence that frailty index is more persistent than SRHS. As an example, someone with an 'excellent' frailty index has 69% chance of maintaining this status while a person with 'excellent' SRHS has only 56% chance. The gap in persistence is similar in the opposite end of health spectrum (for unhealthiest people). However, in the middle range the difference is not as pronounced. To summarize:

Fact 3: Frailty index is more persistent than SRHS.

Transition Probabilities (%)											
Self Reported Health Status						Health Status by Frailty Index					
	'excellent'	'very good'	'good'	'fair'	'poor'		'excellent'	'very good'	'good'	'fair'	'poor'
'excellent'	56.4	32.3	9.3	1.6	0.5	'excellent'	69.4	22.9	6.5	0.9	0.3
'very good'	14.3	56.6	25.0	3.4	0.7	'very good'	14.0	55.7	26.4	3.4	0.6
'good'	4.3	24.6	54.8	14.0	2.4	'good'	2.9	18.3	60.5	15.9	2.3
'fair'	1.6	6.7	28.7	48.6	14.4	'fair'	0.6	3.2	25.7	53.1	17.4
'poor'	1.0	1.7	8.8	29.4	59.2	'poor'	0.3	0.7	3.8	21.7	73.6

Table 1: Transition probabilities for health status. Left panel: Self Reported Health Status. Right panel: health status by frailty index. Source: authors' calculation using PSID.

Table 18 in Appendix A.1 shows the pattern is similar in the HRS data. The transition probabilities are not calculated for MEPS due to data limitation. [15]

3 Estimation of Frailty Process

We are interested in the evolution and dynamics of health status over life cycle. Given the advantages of the frailty index over SRHS, we use the frailty index as a measure of health status. Our goal in this section is to estimate a stochastic process of frailty over the life cycle. To this end, we consider two variations of a statistical model that is flexible enough to capture important properties of frailty dynamics. In particular, the model allows for innovations to frailty to be persistent. This is consistent with the findings in Section 2. The model also allows the variance of log frailty to increase with age which. As we will show below, this is a feature of the data. Both these features of frailty are also observed in earnings dynamics. and Thus our choice of statistical model is inspired by the literature on estimating earnings processes. We first lay out the model and describe the estimation procedure. Then we describe the empirical moments used to estimate the model and present the estimation results.

[15] MEPS contains only one observation of the health conditions used in calculation of frailty index for each individual. Therefore, tracking individual frailty index over time is not possible.

3.1 Statistical Model

Our statistical model is very similar to the one used by Guvenen (2009) to estimate the earning process. In particular, we assume that the log of the frailty index f_{it} for individual i at age t is the sum of a deterministic component whose effect is common to all individuals and a residual that is individual-specific:

$$\ln f_{it} = X'_{it}\beta + R_{it}, \tag{1}$$

where X_{it} is a set of all covariates including age, age-squared, gender, marital status and education.[16] The set of covariates also includes a full set of cohort dummies. The residual consists of two components and is given by

$$R_{it} = \alpha_i + \gamma_i t + z_{it} + u_{it}. \tag{2}$$

The first component, $\alpha_i + \gamma_i t$, allows for individual-specific effects on the levels and growth rate of frailty. This is an important component of the model as it allows to capture heterogeneity in initial frailty level and growth rate of frailty over the life cycle. We assume that (α_i, γ_i) is randomly distributed across individuals with mean zero, variances σ^2_α and σ^2_γ, and covariance $\sigma_{\alpha\gamma}$. The key parameter of interest is σ^2_γ which determines the degree of heterogeneity in growth rate of frailty across individuals. As we discuss below, this parameter is one of the key determinants of growth of variance of log frailty over the life cycle.

The second component is the sum of an AR(1) process and a transitory shock u_{it}. Thus

$$z_{it} = \rho z_{it-1} + \varepsilon_{it}, \tag{3}$$

where $z_{i,0} = 0$.[17][18] The shocks ε_{it} and u_{it} are assumed to be independent of each other and over time, and independent of α_i and γ_i. We assume that ε_{it} has mean zero and variance σ^2_ε and u_{it} has mean zero and variance σ^2_u. The second component captures the dynamics in frailty as individuals go through various random health events over their life cycle. The key parameters here are persistence ρ and variance of permanent shocks σ^2_ε. These parameters also contribute to the growth of variance of log frailty over the life cycle. More importantly they determine the degree of risk in health status that individuals face over their life cycle.

Under this specification of the dynamic process, the cross-sectional variances and covariances of the residual at age t are given by

$$var(R_{it}) = \sigma^2_\alpha + 2\sigma_{\alpha\gamma}t + \sigma^2_\gamma t^2 + var(z_{it}) + \sigma^2_u, \tag{4}$$

$$cov(R_{it}, R_{it+k}) = \sigma^2_\alpha + \sigma_{\alpha\gamma}(2t + k) + \sigma^2_\gamma t(t + k) + \rho^k var(z_{it}), \tag{5}$$

[16] We renormalize frailty so that it ranges from 1 to 2 instead of 0 to 1 before taking logs.

[17] Note that t represents age and not time which means we are assuming that the stochastic component of frailty can vary with age but is time-invariant. The variance of log frailty increases with both age and time in both the PSID and HRS samples. However, the increase with age is much more dramatic so we went with an age-dependent but time-invariant stochastic component.

[18] There are a number of variants of this setup used in the literature to estimate earnings processes. The macro literature has focused on versions that avoid turning the white noise into a moving average component as processes with moving average components are not as easy to embed in quantitative macro models.

Variable	Coefficient	Std. Err.
Age	0.00087	(0.00031)
Age^2	0.00002	(3.0e-6)
Years of School	-0.00482	(0.00012)
Male	0.0042	(0.00058)
Married	-0.02718	(0.00064)
Const.	0.05078	0.01936
Cohort dummies included		
	N=69,092, R^2=0.2060	

Table 2: OLS regression results for log frailty using PSID sample for ages 25–95.

where

$$var(z_{it}) = \rho^2 var(z_{it-1}) + \sigma_\varepsilon^2 = \sigma_\varepsilon^2 \sum_{j=0}^{t-1} \rho^{2j}.$$

Equations (4) and (5) show how the theoretical age profile of variance and covariances for the residual term R_{it} depend on model parameters. In particular note that a higher value of σ_γ^2 makes the age profile of variance more convex in age (equation (4)). The same is true also for persistence parameter ρ. If ρ is larger than 1, then the term $\sigma_\varepsilon^2 \sum_{j=0}^{t-1} \rho^{2j}$ is convex in age, t. Moreover, covariance terms at each age are increasing in lag length, meaning that $cov(R_{it}, R_{it+k+1}) > cov(R_{it}, R_{it+k})$ for all k. The opposite is true if ρ is smaller than one. As we discuss below these features are important in identifying parameters σ_γ^2 and ρ.

We estimate the model in two stages. We first estimate β using OLS and compute the stochastic component R_{it} as residuals In the second stage, we estimate the parameters of the stochastic component (equation (2))using a minimum distance estimator that minimizes the distance between the variances and covariances implied by the model, equations (4) and (5), and their empirical counterparts. This is the standard GMM estimator proposed by Chamberlain (1984) .

Table 2 provides the results of the OLS estimation of the deterministic component of the process using our main PSID sample. Frailty is increasing with age and decreasing in years of schooling. Being male increases frailty by 0.42 percent while being married reduces frailty by 2.75 percent.

The sizable negative effect of marriage on frailty is consistent with other findings in the literature on the impacts of marriage on health.[19] Education also has sizable negative effect on frailty. Every additional year of school reduces frailty by 0.48 percent.

Figure 3 presents the empirical counterparts of equations (4) and (5). As is commonly done in the literature on earning dynamics, we use empirical moments that have been adjusted for cohort effects.[20] The left panel shows the cross-sectional variances of the log frailty residuals, R_{it}, by age. The panel shows both the raw variances and the cohort-adjusted ones.

[19] A summary of the literature is provided by Wood et al. (2009). Guner et al. (2017) find that the positive effect of marriage on health is primarily due to selection but that protective effects of marriage also play a role especially at older ages.

[20] See Deaton and Paxson (1994), Guvenen (2009) and Storesletten et al. (2004).

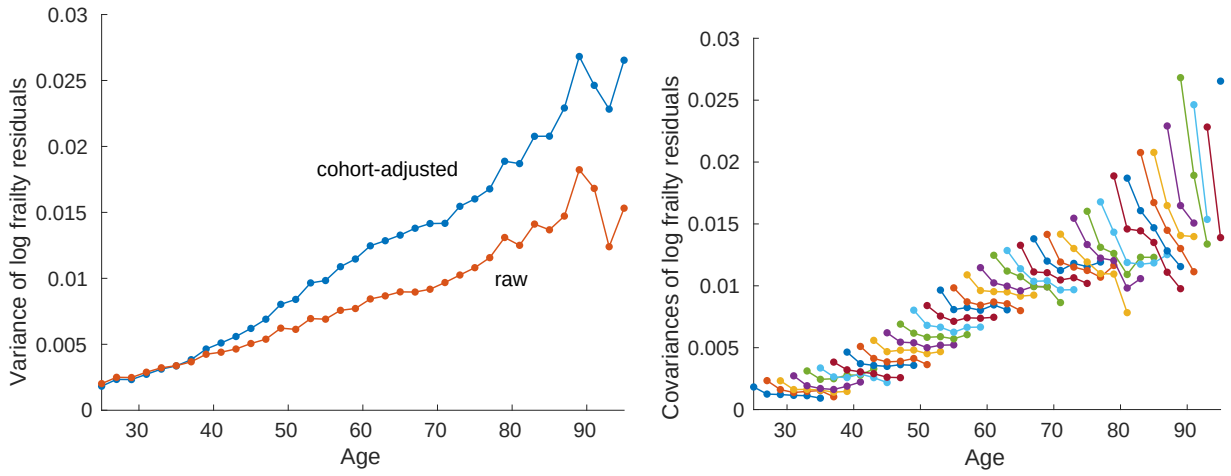


Figure 3: Raw and cohort-adjusted variances (left) and cohort-adjusted covariances (right) of the residuals, R_{it}, by age in PSID.

To construct the variances we group individuals into 2-year, non-overlapping, age groups (25–26 year-olds, 27–28 year-olds, and so on). The raw variance profile is the means of the squared residuals of each age group. To obtain the cohort-adjusted variance profile, we regress the raw variances on a full set of age and cohort dummies to obtain cohort-adjusted squared residuals. To maintain the same level of inequality after cohort effects are removed, the cohort-adjusted variances are rescaled such that the adjusted variance at age 35 is the same as the raw variance at age 35. The variance profile demonstrate two key features. One, as individuals age the cross section variance of the log frailty residual increases. Two, the rate at which the cross section variance increases with age is slightly higher for older individuals. In other words, age profile of the variance is slightly convex in age.

The right panel of Figure 3 shows the entire empirical covariance matrix after adjusting for cohort effects. The first point in each line is the variance of that age group's log frailty residual R_{it}, the next point is the covariance between R_{it} and R_{it-1} and so on. To get the cohort-adjusted covariances we regress age and individual-specific moments on cohort and age dummies separately for each age group. We then compute cohort-adjusted individual-specific moments using the residuals and age effects rescaled in the same manner as we rescaled the variances. The cohort-adjusted covariances are the means of the moments for each age group and lag.[21] Autocovariances also demonstrate a general increasing pattern with age. However, more importantly, autocovariances in each have a negative slope with respect to lag length with flatter profile for younger ages and steeper profile for older ages.

We suspect that the rapid declines in the autocovariances at older ages is driven by sample attrition due to death being correlated with frailty. Highly frail individuals are more likely to die and, as a result, are less likely to contribute to higher-order autocovariances. Since this selectivity bias becomes more severe as the lag length increases it puts downward pressure on the autocovariance structure. While the mortality rates, and hence attrition rates, of working-age individuals are fairly low, retirees are more likely to both be highly frail and to

[21] Additional details on the construction of the cohort-adjusted variance-covariance matrix can be found in the Appendix.

die. Hence the effect of this selectivity bias on the autocovariance structure of retirees is of particular concern. To address this concern, we drop the covariance moments of individuals 65 and older from the estimation. Thus our empirical moments consist of the variances and entire autocovariance structure for working-age individuals and only the variances for retirees. We explore robustness of our estimation to this choice of target moment in Section 6.

The assumptions we make about the structure of the stochastic component has implications for the ability of the model moments to match well the moments from the data. We estimate two versions of the statistical model presented above. Our preferred version puts no *a priori* restriction parameter. We call this version *Unrestricted*. We estimated a *restricted* model in which we shut down variation in the growth rate of frailty across individuals by setting $\sigma^2_\gamma = 0$. Under this version of the model, variation in frailty due to the individual fixed effect α_i remains. In theory, both versions of the model have the ability to match the patterns in the data documented in Figure 3.

ρ	σ^2_α	σ^2_γ	$\sigma_{\alpha\gamma}$	σ^2_u	σ^2_ε
A. Unrestricted					
0.7717	0.0006	4.5e-6	2.7e-5	0.0005	0.0005
(0.0376)	(0.0001)	(2.7e-07)	(6.6e-6)	(0.0001)	(0.0001)
B. Restricted					
1.0098	0.0007	–	–	0.0008	0.0002
(0.0008)	(0.0001)	–	–	(2.3e-5)	(8.0e-6)

Table 3: Results – PSID samples: 5 cov, 25–65, variance only 65-95

Table 3 presents the results from the GMM estimation of the restricted and unrestricted versions of the model using the variances and entire autocovariance structure for working-age individuals and only the variances for retirees as target moments. Under the restricted specification, ρ is estimated to be larger than 1 and the hypothesis that $\rho = 1$ is rejected at standard significance levels. This value of ρ is driven by the slightly convex shape of the empirical variance profile. However, notice from equation (5), that under the restricted specification and with ρ larger than 1, the autocovariances are increasing with the lag order which is opposite the pattern in the data. There is a tension in the restricted model between the variance profile and the autocovariance structure. The gradually decaying autocovariances suggest that ρ lies between 0 and 1. However, the slightly convex variance profile forces ρ to be larger than 1.[22]

It is important to note that under unrestricted specification variance of both individual specific component are positive and significantly different from zero. This means that there is heterogeneity in initial level of frailty (captured by σ^2_α) are well as heterogeneity in growth rate of frailty (captured by σ^2_γ).

The advantage of the unrestricted specification is that it has the ability to simultaneously match both the slightly convex variance pattern and the decaying auto-covariance pattern.

[22] As it is pointed out by Guvenen (2009) if the data is generated by a true unrestricted process, then estimating a restricted process can introduce significant upward bias into estimation of persistence parameter. The large estimated value for ρ might be due to this bias.

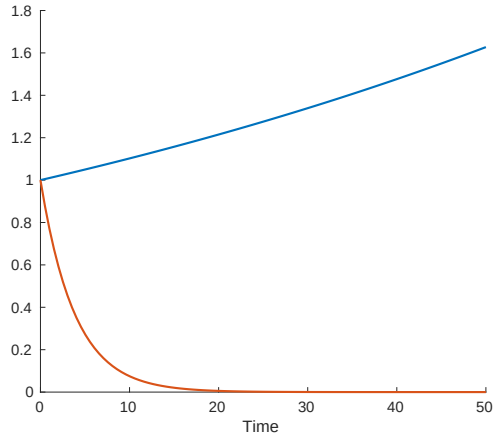


Figure 4: Relative effects going forward of a time-0 AR(1) shock under the restricted versus the unrestricted specification.

This is because under the unrestricted specification, variation in the growth rates of frailty can also induce a convex variance pattern, as can be noted in equation (4). Therefore, with ρ less than 1, the unrestricted specification can match both the slightly convex variance pattern and the decaying auto-covariance pattern. Note that, consistent with this intuition, allowing for heterogenous profiles substantially reduces the estimated value ofρ. The value of ρ estimated in the unrestricted model falls well below 1 and we can reject that shocks are permanent random walk.

The disadvantage of the unrestricted model is that the slope of the covariance pattern becomes more and more steep with age in the data but the model implies the opposite pattern. However, the increasing steepness with age may be due to sample attrition being correlated with frailty and the fact that attrition is increasing with age due to death. Overall we take the unrestricted model as our preferred model as it better captures features in the data. Therefore, the estimates in the first row of Table 3 are our preferred estimates.

We must however stress that each model has important economic implications. The unrestricted model attributes more of the cross sectional variations in frailty to heterogeneity among individuals and less to persistent shocks. On the other hand the restricted model attributes all variations in frailty to shocks. Therefore, these models have different implications for value of risk and insurance vis a vis redistribution across health/frailty types. To make this point more concrete, note that the difference between these two estimates of ρ has enormous implications for the long-run effects of current frailty innovations. Figure 4 plots the relative effect of an innovation today at future ages. When $\rho = 1.0098$ the impact of a frailty shock is increasing with age. After 10 years its effect is 10% larger than it was initially and the impact of the shock doubles after approximately 71 years. In contrast, when $\rho = 0.7717$, the effect of the shock falls to less than 30% by 5 years out and is less than 10% by 10 years out. These figures imply that there is a much higher premium for preventing a frailty shock under restricted specification.

Given our estimation of the unrestricted model we can quantify the contribution of heterogenous profiles and stochastic AR(1) component to the overall inequality in frailty. To

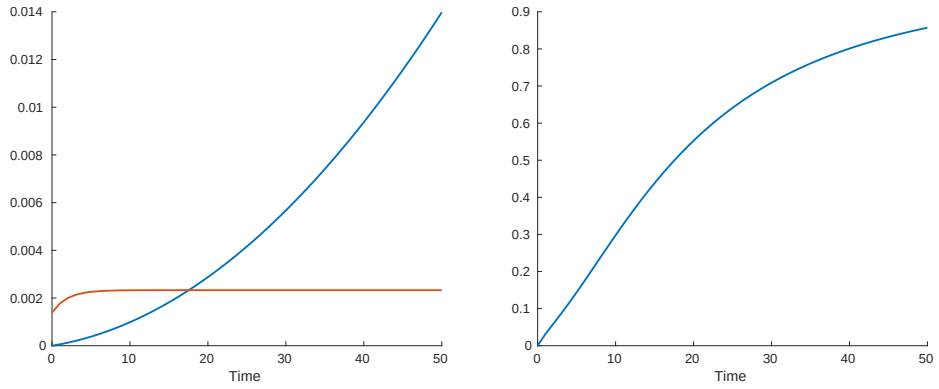


Figure 5: Relative contribution of AR(1) versus Heterogenous profile components

do this we use equation (4) which we rewrite below

$$var(R_{it}) = \left(\sigma_\alpha^2 + \sigma_u^2\right) + \left(2\sigma_{\alpha\gamma}t + \sigma_\gamma^2 t^2\right) + \left(\sigma_\varepsilon^2 \frac{1-\rho^{2t}}{1-\rho^2}\right).$$

The first term is the intercept and is determined by initial heterogeneity in frailty. The second term in the contribution of heterogeneous profiles. The third term is the contribution of stochastic AR(1) term. Left panel in Figure 5 shows the heterogenous profile component (blue line) and AR(1) component (red line). As we argues before, since the estimated persistence parameter ρ is less than 1, the AR(1) component is concave. This component is dominant at younger age. However its contribution to the variation in frailty is capped in less than ten years. On the other hand heterogenous profile component is convex with age and is the main driver of the variance in log frailty for most of the life cycle. Right panel of Figure 5 shows the relative contribution of heterogenous profile component. By age 45 (after 20 years) about 50 percent of variance in log frailty is due to profile heterogeneity. This contribution rises to 87 percent by age 75.

3.2 Estimation Results from Subsamples

In this subsection, we show the estimation results in subsamples by gender and education levels.[23] As in the case with the baseline sample, our empirical moments consistent of the variances for individuals aged 25–95 but covariances only for ages 25–65.

Table 4 presents the estimation results under the restricted and unrestricted specifications for men and women. Interesting, the estimated processes do not differ much by gender. Under both specifications, innovations to women's frailty process are more variable but slightly less persistent than innovations to men's. However, the differences are small and only significant under the restricted specification.

On the other hand, as shown in Table 5, the estimation results are different by education. In particular, under our preferred specification (i.e., the unrestricted model), the value of ρ is 0.79 for college graduates while the same number is 0.71 for high school graduates. In

[23] All results are reported using PSID data. Similar estimation results for the HRS data are available upon request from the authors.

addition, the college graduates face smaller persistent shock and smaller transitory shock relative to the high school graduates. However, the heterogeneity in individual fixed effects (both level and growth) are very similar across education groups overall.

Tables 6 and 7 report the estimation results by education for each gender respectively. College educated men have more persistent shocks to frailty relative to those with only high school degree. The pattern is reversed when we look at female by education group, where females with high school education have more persistence frailty shocks. However, as we saw in Table 4 these two different patterns across education groups cancel each other. Overall, there is no difference in persistence among male and female.

	ρ	σ^2_α	σ^2_γ	$\sigma_{\alpha\gamma}$	σ^2_u	σ^2_ε
A. Unrestricted						
All	0.7717	0.0006	4.5e-6	2.7e-5	0.0005	0.0005
	(0.0376)	(0.0001)	(2.7e-07)	(6.6e-6)	(0.0001)	(0.0001)
Men	0.7542	0.0003	4.19e-06	1.14e-05	0.0004	0.0004
	(0.0491)	(0.0001)	(3.04e-07)	(5.88e-06)	(0.0001)	(0.0001)
Women	0.7387	0.0008	4.25e-06	4.57e-05	0.0005	0.0006
	(0.0548)	(0.0001)	(3.6e-07)	(8.94e-06)	(0.0001)	(0.0001)
B. Restricted						
All	1.0098	0.0007	–	–	0.0008	0.0002
	(0.0008)	(0.0001)	–	–	(2.3e-5)	(8.0e-6)
Men	1.0145	0.0003	–	–	0.0007	0.0001
	(0.0013)	(0.0001)	–	–	(2.57e-05)	(7.13e-06)
Women	1.0084	0.0010	–	–	0.0009	0.0002
	(0.0010)	(0.0001)	–	–	(3.31e-05)	(1.15e-05)

Table 4: PSID samples: 5 cov, 25–65, variance only 65-95, by gender

	ρ	σ^2_α	σ^2_γ	$\sigma_{\alpha\gamma}$	σ^2_u	σ^2_ε
A. Restricted						
All	0.7717	0.0006	4.5e-6	2.7e-5	0.0005	0.0005
	(0.0376)	(0.0001)	(2.7e-07)	(6.6e-6)	(0.0001)	(0.0001)
Highschool	0.7101	0.0005	5.01e-06	4.49e-05	0.0004	0.0007
	(0.0650)	(0.0001)	(3.61e-07)	(9.02e-06)	(0.0002)	(0.0002)
College	0.7910	0.0004	4.55e-06	1.12e-05	0.0005	0.0003
	(0.0476)	(0.0001)	(3.12e-07)	(6.78e-06)	(0.0001)	(0.0001)
B. Restricted						
All	1.0098	0.0007	–	–	0.0008	0.0002
	(0.0008)	(0.0001)	–	–	(2.3e-5)	(8.0e-6)
Highschool	1.0098	0.0007	–	–	0.0009	0.0002
	(0.0010)	(0.0001)	–	–	(3.81e-05)	(1.18e-05)
College	1.0123	0.0003	–	–	0.0007	0.0001
	(0.0011)	(0.0001)	–	–	(2.57e-05)	(7.53e-06)

Table 5: PSID samples: 5 cov, 25–65, variance only 65-95, by education

	ρ	σ^2_α	σ^2_γ	$\sigma_{\alpha\gamma}$	σ^2_u	σ^2_ε
A. Unrestricted						
All	0.7542	0.0003	4.19e-06	1.14e-05	0.0004	0.0004
	(0.0491)	(0.0001)	(3.04e-07)	(5.88e-06)	(0.0001)	(0.0001)
Highschool	0.7529	0.1.93e-04	5.14e-06	2.14e-05	0.0005	3.53e-04
	(0.0880)	(1.45e-04)	(4.08e-07)	(8.34e-06)	(1.30e-04)	(1.31e-04)
College	0.8869	2.86e-04	3.53e-06	-4.53e-06	4.03e-04	1.62e–04
	(0.0471)	(8.79e-05)	(3.37e-07)	(7.90e-06)	(4.01e-05)	(3.91e-05)
B. Restricted						
All	1.0145	0.0003	–	–	0.0007	0.0001
	(0.0013)	(0.0001)	–	–	(2.57e-05)	(7.13e-06)
Highschool	1.0149	2.31e-04	–	–	0.0007	1.34e-04
	(0.0013)	(1.05e-04)	–	–	(4.10e-05)	(9.73e-06)
College	1.0181	2.58e-04	–	–	4.92e-04	6.43e-05
	(0.0016)	(5.73e-05)	–	–	(2.19e-05)	(5.24e-06)

Table 6: PSID samples: 5 cov, 25–65, variance only 65-95, Men by education

	ρ	σ_α^2	σ_γ^2	$\sigma_{\alpha\gamma}$	σ_u^2	σ_ε^2
A. Unrestricted						
All	0.7387	0.0008	4.25e-06	4.57e-05	0.0005	0.0006
	(0.0548)	(0.0001)	(3.6e-07)	(8.94e-06)	(0.0001)	(0.0001)
Highschool	0.7922	0.0007	3.83e-06	0.0001	0.0006	0.0006
	(0.0627)	(0.0002)	(5.03e-07)	(1.53e-05)	(0.0002)	(0.0002)
College	0.7454	0.0006	5.88e-06	4.59e-06	0.0005	0.0003
	(0.0831)	(0.0001)	(4.38e-07)	(9.28e-06)	(0.0001)	(0.0001)
B. Restricted						
All	1.0084	0.0010	–	–	0.0009	0.0002
	(0.0010)	(0.0001)	–	–	(3.31e-05)	(1.15e-05)
Highschool	1.0059	0.0011	–	–	0.0009	0.0003
	(0.0012)	(0.0002)	–	–	(0.0001)	(1.83e-05)
College	1.0139	0.0004	–	–	0.0007	0.0001
	(0.0013)	(0.0001)	–	–	(3.48e-05)	(1.00e-05)

Table 7: PSID samples: 5 cov, 25–65, variance only 65-95, Women by education

4 Cross-sectional distribution

In "Changes with Age in the Distribution of a Frailty Index" they assume a Gamma distribution.

5 Relationships between Frailty and Other Key Variables

In Section 2, we show that frailty index gives a more accurate picture of how an individual's health evolve with age, due to its various of advantages over SRHS. In this section, we move one step further by investigating whether frailty index is also capable of better capturing the impact of health on other key variables, such as survival probability and individual productivity, over the life cycle. Specifically, we study and compare two versions of regression models, in which we use frailty index and SRHS to measure health status respectively. We are particularly interested in the impact of frailty on survival probability, wage, and earnings.

We first investigate the impact of frailty on survival probability by running the following regression model:

$$SP_{it} = \beta_1 f_{it} + \beta_2 f_{it}^2 + \beta_3 t + \beta_4 t^2 + \beta Educ_{it} + \epsilon_{it}. \tag{6}$$

Here SP_{it} is the survival probability of agent i from age t to $t+1$, and $Educ_{it}$ is the years of schooling of that agent at age t. We include the quadratic terms here to capture any possible nonlinearity in the relationship between survival probability and frailty. The results from

this regression are reported under column 1 in Table 8. The impact of frailty on survival probability is highly significant, with a z score of 12. The second and third columns in the same table report the regressions results with different control variables.

For comparison, we repeat the same regressions with SRHS variables replacing the frailty variables. The results from these regressions are reported in Table 9. The estimation results from the two versions of regression models are qualitatively similar. However, as can be noted, the R squares of the frailty regressions are larger than those of the SRHS regressions, suggesting that frailty index has a better predictive power for survival probability than SRHS.

Variable	Coefficient	Z Score	Coefficient	Z Score	Coefficient	Z Score
Frailty	4.7734	12.09	4.7426	12.4	7.1001	20.55
Frailty^2	-3.6926	-5.78	-3.5030	-5.64	-5.8357	-10.22
Age	-0.0095	-1.53	-0.0107	-1.77		
Age^2	0.0003	6.06	0.0003	6.5		
Years of School	-0.0240	-4.02				
Constant	-3.1489	-15.87	-3.4326	-19.28	-3.2094	-77.11
R^2	0.2840		0.2840		0.186	

Table 8: Survival Probits Regression: Frailty Index

Variable	Coefficient	Z Score	Coefficient	Z Score	Coefficient	Z Score
SHLT=2	0.0912		0.1194		0.1911	
SHLT=3	0.3065		0.3406		0.4786	
SHLT=4	0.6377		0.6683		0.9300	
SHLT=5	1.1528		1.1865		1.5960	
Age	-0.0107		-0.0122			
Age^2	0.0003		0.0004			
Years of School	-0.0136					
Constant	-3.1408		-3.3117		-2.9130	
R^2	0.2794		0.2793		0.1429	

Table 9: Survival Probit Regressions: Self-reported Health Status

To explore the impact of frailty on individual productivity, we consider two dependent variables: log wage rate and earnings. Specifically, we study the following regression model:

$$y_{it} = \beta_1 f_{it} + \beta_2 f_{it}^2 + \beta_3 t + \beta_4 t^2 + \beta Educ_{it} + \epsilon_{it}. \tag{7}$$

Here y_{it} is either the log wage of agent i at age t or the earnings of this agent. The results from the wage and earnings regressions are reported in 10. As for comparison, we also study the same regressions with SRHS variables instead of frailty variables, and the results from these regressions are reported under the first column in Table 11. As can be seen, the impact of frailty on productivity is always significant and negative.

Variable	Wage Coefficient	Z Score	Earnings Coefficient	Z Score
Frailty	-1.7319	-12.91	-1.8237	-9.22
Frailty^2	2.0604	4.7	-3.2989	-5.17
Age	0.0713	37.51	0.1464	52.19
Age^2	-0.0007	-31.4	-0.0016	-50.15
Years of School	0.1087	64.86	0.1237	49.63
Constant	-0.0889	-1.88	5.8349	83.24
R^2	0.234		0.207	

Table 10: Wage and Earnings Regressions: Frailty Index

Variable	Wage Coefficient	Z Score	Earnings Coefficient	Z Score
SHLT=2	-0.0641	-6.29	-0.0950	-6.24
SHLT=3	-0.1507	-13.68	-0.2226	-13.52
SHLT=4	-0.2467	-14.95	-0.4686	-19.11
SHLT=5	-0.2637	-7.47	-0.7544	-14.4
Age	0.0750	39.51	0.1537	54.6
Age^2	-0.0007	-33.65	-0.0017	-53.07
Years of School	0.1082	64.26	0.1229	48.9
Constant	-0.1833	-3.96	5.6761	82.56
R^2	0.234		0.2	

Table 11: Wage and Earnings Regressions: Self-reported Health Status

6 Robustness

6.1 Alternative Empirical Moments

Among retirees, We have dropped the covariance moments for individuals older than 65 in our benchmark estimation due the concern about the selection bias. Here we evaluate the sensitivity of our estimations with respect to this decision by exploring alternative choices of empirical moments. In particular we repeat our estimation on two alternative samples. In the first sample we include all variance and covariance moments for all ages. In the second sample we include all variance and covariance moments for but estimate the model only for individuals ages 25 to 65.

Table 12 presents the results from GMM estimation of the restricted and unrestricted versions of the model using the variances and entire auto-covariance structure for all individuals from age 25 to 95 as target moments. Table 13 presents the results from the same estimations using the variances and entire auto-covariance structure for the working-age individuals (from age 25 to 65) as target moments. The results are qualitatively similar to our main estimations. The estimated value of ρ is slightly smaller under both model specifications

when the auto-covariance structure for retirees are included (top panel in Table 12). This is because the auto-covariances decay faster for retirees in the data, and therefore a smaller ρ is needed to match them. On the other hand, the estimated value of ρ is slightly above its counterpart in the benchmark estimation when only the variance and auto-covariances of the working-age individuals are used in the estimation (top panel in Table 13). This is likely due to the fact that in the data the age profile of variance is slightly more convex between age 25 and 65 than for the entire age distribution.

ρ	σ_α^2	σ_γ^2	$\sigma_{\alpha\gamma}$	σ_u^2	σ_ε^2
A. Unrestricted					
0.7551	0.0004	2.74e-6	0.0001	0.0005	0.0006
(0.0337)	(0.0001)	(2.37e-7)	(6.07e-6)	(0.0001)	(0.0001)
B. Restricted					
1.0045	0.0006	–	–	0.0008	0.0002
(0.0008)	(0.0001)	–	–	(2.29e-5)	(9.08e-6)

Table 12: Results – PSID samples: 5 cov, 25–95

ρ	σ_α^2	σ_γ^2	$\sigma_{\alpha\gamma}$	σ_u^2	σ_ε^2
A. Unrestricted					
0.8054	0.0008	7.38e-6	-1.29e-5	0.0006	0.0004
(0.0355)	(0.0001)	(5.34e-7)	(9.44e-6)	(0.0001)	(0.0001)
B. Restricted					
1.0133	0.0007	–	–	0.0008	0.0002
(0.0019)	(0.0001)	–	–	(2.38e-5)	(1.1e-5)

Table 13: Results – PSID samples: 5 cov, 25–65

6.2 Comparison to HRS

Figure 6 shows the same statistics for the HRS data. Both the variances and the auto-covariances show patterns similar to those in the PSID data. The variance increases monotonically with age from ages 65 to 95,[24] and the auto-covariances also decay as the lag order increases. In addition, the auto-covariances decay faster at older ages. As explained previously, this is most likely due to the selection bias that highly frail individuals are more likely to die and, as a result, are less likely to contribute to higher-order auto-covariances

Table 14 shows the results of estimation for unrestricted and restricted process in HRS data. The unrestricted process shows much higher persistence in HRS relative to PSID. Also the variance of individual specific parameters are much higher relative to PSID. This is expected. Individuals enter HRS at much older age. Therefore, the initial frailty in HRS sample has must higher variance due to the fanning out that occurs at younger ages. The

[24]Note that the non-monotonicity of the variance over the 50–65 age range is a robust feature of the HRS sample that is most likely due to survey design.

dispersion in growth rate of individual profiles σ^2_γ is also larger in HRS relative to PSID.This is likely due to that fact that age profile of variance in HRS has more curvature relative to PSID (it is more convex). However, the correlation between individual level and growth fixed effect is now negative.

The estimation of the restricted model in HRS is very similar to that of PSID (bottom panel of Table 14), with the exception of higher variance of level fixed effect.

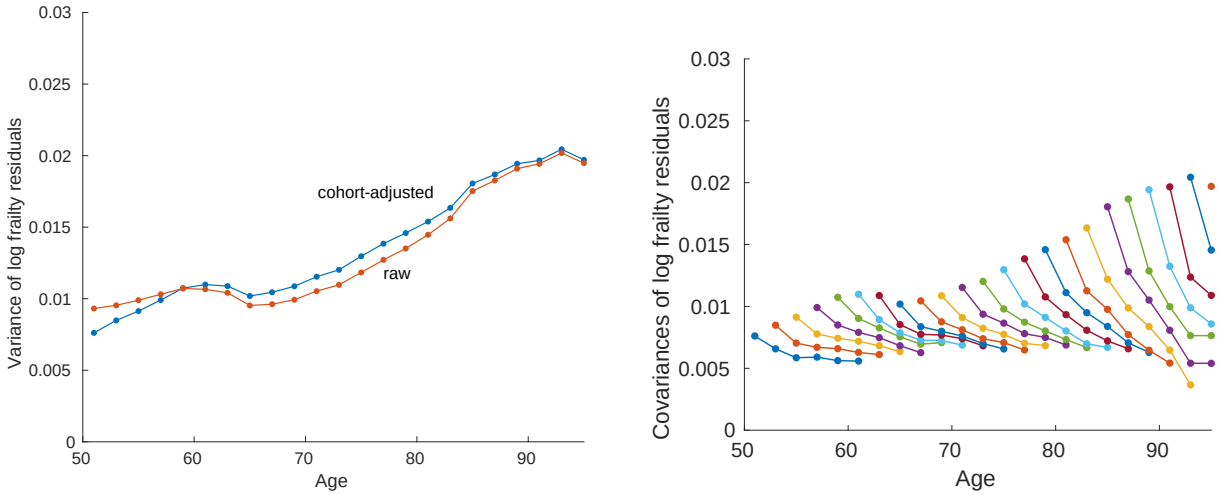


Figure 6: Raw and cohort-adjusted variances (left, darker lines) and cohort-adjusted covariances (right) of the residuals, R_{it}, by age in HRS. The lighter lines in the left panel are the raw and cohort-adjusted variances from the PSID and are provided to facilitate comparison.

ρ	σ^2_α	σ^2_γ	$\sigma_{\alpha\gamma}$	σ^2_u	σ^2_ε
A. Unrestricted					
0.8697	0.0059	8.50e-6	-0.0001	0.0009	0.0009
(0.0245)	(0.0002)	(8.27e-7)	(2.05e-5)	(0.0001)	(0.0001)
B. Restricted					
1.0074	0.0059	–	–	0.0014	0.0002
(0.0021)	(0.0002)	–	–	(3.54e-5)	(1.79e-5)

Table 14: Results – HRS samples: 5 cov, 51–65, variance only 65-95

A Appendix

A.0.1 PSID Data

We use 2003–2015 PSID data. The PSID is biennial over this period. We do not use years prior to 2003 because the PSID expanded its disability and health-related questions in 2003 to include questions on specific medical conditions, activities of daily living (ADL's) and instrumental activities of daily living (IADL's) which we rely on to construct individuals' frailty indices. For the base sample, the only restriction is that a person is a household head

or the spouse of a household head and at least 25 years of age. A good description of the PSID household head definition is in Heathcote et al. (2010). The base sample consists of 18,548 individuals (8,765 men, 9,784 women; 13,224 household heads, 7,128 spouses).

Table 15 lists the variables we used to construct the frailty index for PSID respondents. The index is constructed by summing the variables in the first column of the table using their values which are assigned according to the rules in the second column. Then dividing this sum by the total number of variables observed for the individual in the year. The construction of this frailty index mostly follows the guidelines laid out in Searle et al. (2008), and uses a set of PSID variables similar to the index created in Yang and Lee (2009).

A.0.2 HRS Data

Our HRS sample is constructed using the 1998–2014 waves of the HRS/AHEAD. We restict the sample to individuals aged 51 or older. The base sample consists of 36,102 individuals (16,041 men, 20,061 women).

Table 16 lists the variables we used to construct the frailty index for HRS respondents. The index is constructed in the same way for HRS respondents as for PSID respondents. In contrast to Searle et al. (2008) and the PSID, the HRS index is comprised of a larger number of variables measuring mental/cognitive health.

A.0.3 MEPS data

We use 2000–2014 MEPS data. The base sample consists of MEPS respondents aged 25 years and older. The base sample consists of XXX individuals (XXX men, XXX women). Table 17 lists the variables we used to construct the frailty index for MEPS respondents. The index is constructed in the same way for MEPS respondents as for PSID and HRS respondents.

A.1 Frailty index vs. Self-reported Health Status in HRS and MEPS

In section 2 we report the comparison between SRHS and frailty index in PSID data. We argue, based on the reported data, that frailty index is more accurate indicator of individuals health status. In this section we repeat this comparison for HRS and MEPS data. All of the patterns we report for PSID in section 2 can be also seen in HRS and MEPS. However, HRS does not have many observations for individuals younger than 50. Therefore, we report HRS results only for people older than 50.

Figure 7a shows average frailty index at each age within each SRHS category in HRS. The figure clearly shows higher average frailty index for each SRHS at any age. Also, similar to PSID data there is large variation in average frailty index by age for the same SRHS. Figure 7b shows similar pattern in MEPS.

Table 15: Health Variables used to construct frailty index for PSID respondents

Variable	Value
Some difficulty with ADL/IADLs:	
Eating	Yes=1, No=0
Dressing	Yes=1, No=0
Getting in/out of bed or chair	Yes=1, No=0
Using the toilet	Yes=1, No=0
Bathing/showering	Yes=1, No=0
Walking	Yes=1, No=0
Using the telephone	Yes=1, No=0
Managing money	Yes=1, No=0
Shopping for personal items	Yes=1, No=0
Preparing meals	Yes=1, No=0
Heavy housework	Yes=1, No=0
Light housework	Yes=1, No=0
Getting outside	Yes=1, No=0
Ever had one of following conditions:	
High Blood Pressure	Yes=1, No=0
Diabetes	Yes=1, No=0
Cancer	Yes=1, No=0
Lung disease	Yes=1, No=0
Heart disease	Yes=1, No=0
Heart attack	Yes=1, No=0
Stroke	Yes=1, No=0
Arthritis	Yes=1, No=0
Asthma	Yes=1, No=0
Loss of memory or mental ability	Yes=1, No=0
Psychological problems	Yes=1, No=0
Other serious, chronic condition	Yes=1, No=0
BMI ≥ 30	Yes=1, No=0
Has smoked ever	Yes=1, No=0

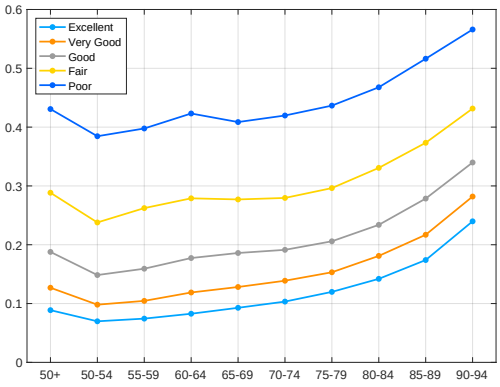
Table 16: Health Variables used to construct frailty index for HRS respondents

Variable	Value
Some difficulty with ADL/IADLs:	
Eating	Yes=1, No=0
Dressing	Yes=1, No=0
Getting in/out of bed	Yes=1, No=0
Using the toilet	Yes=1, No=0
Bathing/shower	Yes=1, No=0
Walking across room	Yes=1, No=0
Walking several blocks	Yes=1, No=0
Using the telephone	Yes=1, No=0
Managing money	Yes=1, No=0
Shopping for groceries	Yes=1, No=0
Preparing meals	Yes=1, No=0
Getting up from chair	Yes=1, No=0
Stooping/kneeling/crouching	Yes=1, No=0
Lift/carry 10 lbs	Yes=1, No=0
Using a map	Yes=1, No=0
Taking medications	Yes=1, No=0
Climbing 1 flight of stairs	Yes=1, No=0
Picking up a dime	Yes=1, No=0
Reaching/ extending arms up	Yes=1, No=0
Pushing/pulling large objects	Yes=1, No=0
Cognitive Impairment:	
Immediate Word Recall	+.1 for each word not recalled (10 total)*
Delayed Word Recall	+.1 for each word not recalled (10 total)*
Serial 7 Test	+.2 for each incorrect substraction (5 total)
Backwards Counting	Failed test=1, 2nd attempt = .5, 1st attempt = 0
Identifying obejcts & Pres/VP	.25 for each incorrect answer (4 total)
Identifying date	.25 for each incorrect answer (4 total)
Ever had one of following conditions:	
High Blood Pressure	Yes=1, No=0
Diabetes	Yes=1, No=0
Cancer	Yes=1, No=0
Lung disease	Yes=1, No=0
Heart disease	Yes=1, No=0
Stroke	Yes=1, No=0
Psychological problems	Yes=1, No=0
Arthritis	Yes=1, No=0
BMI ≥ 30	Yes=1, No=0
Has smoked ever	Yes=1, No=0

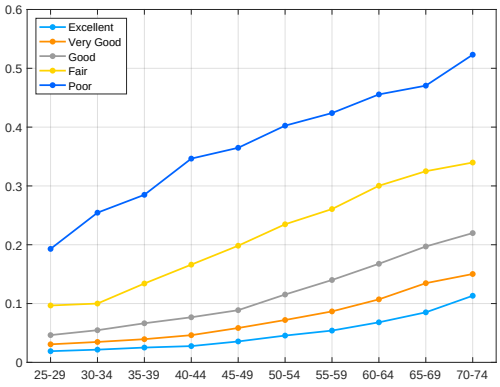
*For the 1994 HRS cohort, 40 questions were asked (instead of 20) for word recall. In this year, each missed question receives weight 0.05.

Table 17: Health Variables used to construct frailty index for MEPS respondents

Variable	Value
Need help with ADLs	Yes=1, No=0
Need help with IADLs	Yes=1, No=0
Use assistive technology	Yes=1, No=0
Limitation impacts work/housework/school	Yes=1, No=0
Any difficultly with the following:	
Walking three blocks	Yes=1, No=0
Standing for 20 minutes	Yes=1, No=0
Bending/Stooping	Yes=1, No=0
Lifting 10 pounds	Yes=1, No=0
Walking up 10 steps	Yes=1, No=0
Using fingers to grasp	Yes=1, No=0
Reaching over head	Yes=1, No=0
Ever been diagnosed with:	
High Blood Pressure	Yes=1, No=0
Diabetes	Yes=1, No=0
Cancer	Yes=1, No=0
Emphysema	Yes=1, No=0
Angina	Yes=1, No=0
Coronary Heart Disease	Yes=1, No=0
Other Heart Disease	Yes=1, No=0
Heart Attack	Yes=1, No=0
Stroke	Yes=1, No=0
Asthma	Yes=1, No=0
Arthritis	Yes=1, No=0
High Cholesterol	Yes=1, No=0
Other serious, chronic condition	Yes=1, No=0
BMI ≥ 30	Yes=1, No=0
Cognitive Limitations	Yes=1, No=0
K6 Depression Score	0–25, rescaled to 0–1



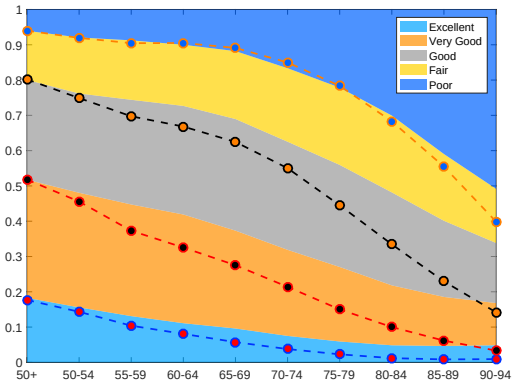
(a) Average frailty index by self reported health status and age. Source: authors calculation using HRS.



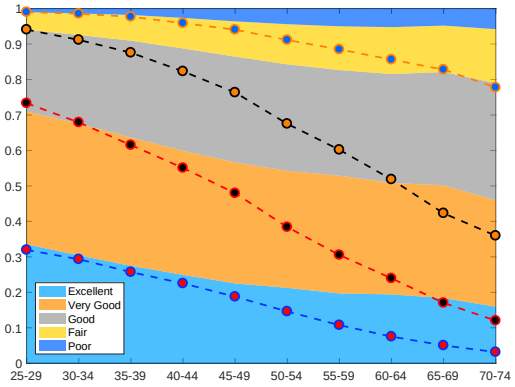
(b) Average frailty index by self reported health status and age. Source: authors calculation using MEPS.

Figure 7

Figure 8a shows how distribution of SRHS and frailty index evolves in HRS as individuals get older. The shaded area is the share of each SRHS at each age. We construct partitions of frailty distribution following the same procedure described in section 2. The only difference is that the frailty cut offs are calculated so that the partitions of the frailty distribution match the distribution of SRHS at age 50-54 (instead of age 25-29). Similar to the PSID data we observe that the distribution of frailty index evolve more rapidly with age. For example, about 20% of 95-99 year olds have 'excellent'/'very good' assessment of their health, while only less than 5% of the have frailty index below the cut off that put them into that category (according to frailty index). Figure 8b shows similar pattern in MEPS data. It worths nothing that the evolution of health status by frailty index in MEPS is remarkably similar to that of PSID (see Figure 2). However, fraction of people in different SRHS categories in MEPS evolves very differently compared to PSID. In particular, in MEPS these fraction are less variable with age. This is another indication that frailty index is more consistent indicator of health status than SRHS.



(a) Distribution of health status by age. The area shows the fraction of each SRHS at each age. The dashed line shows the fraction of health status according to frailty index. Source: authors calculation using HRS.



(b) Distribution of health status by age. The area shows the fraction of each SRHS at each age. The dashed line shows the fraction of health status according to frailty index. Source: authors calculation using MEPS.

Figure 8

Finally Table 18 shows transition probabilities between different health status categories in HRS. [25] As with PSID data we see higher persistence in frailty index, specially for the 'excellent' and 'poor' health status.

Transition Probabilities (%)											
Self Reported Health Status						Health Status by Frailty Index					
	'excellent'	'very good'	'good'	'fair'	'poor'		'excellent'	'very good'	'good'	'fair'	'poor'
'excellent'	50.5	34.9	9.9	2.5	2.2	'excellent'	57.3	33.6	5.0	0.9	0.2
'very good'	11.8	54.1	25.5	5.4	3.2	'very good'	8.7	58.4	28.9	3.5	0.5
'good'	3.0	21.9	49.7	17.8	7.7	'good'	0.7	14.8	59.1	22.9	2.5
'fair'	1.2	6.5	24.8	44.7	22.8	'fair'	0.1	1.3	18.7	60.8	19.1
'poor'	0.5	2.0	7.5	25.4	64.6	'poor'	0.0	0.2	1.4	19.9	78.5

Table 18: Transition probabilities for health status. Left panel: Self Reported Health Status. Right panel: health status by frailty index. Source: authors calculation using HRS.

A.2 Computation of cohort-adjusted empirical moments

This section describes how we obtain the cohort-adjusted variance and covariance moments that we target in the GMM estimations of the frailty process described in Section 3. The cohort-adjusted moments for the main PSID sample are shown in Figure 3. The corresponding raw moments are in Figure 9.

First, cohorts are defined. Cohort 1 consists of all individuals born before 1911 (this is 35 individuals with birth years from 1905–1910). Cohorts then increment with every two

[25] MEPS contains only one observations of the health conditions used in calculation of frailty index for each individual. Therefore, tracking individual frailty index over time is not possible and transition probabilities are not calculated for MEPS data.

birth years, so that the second cohort includes birth years 1911 and 1912, and the third 1913 and 1914. Denote the number of cohorts by n_c. Next, age groups are defined. Each age group spans 2 years and age groups are non-overlapping. Denote an individual's age group by age_f. The initial OLS regression is then run on the sample of individuals meeting the desired age restrictions for the particular covariance matrix being computed. The means of the squared residuals R^2_{it} for each age group are the raw variance profile. Denote the raw variance of the 35–36 year old age group by R^2_{35}.

To obtain the cohort-adjusted variances and covariances, we first define the individual-specific moments

$$m_i^{t,t+k} = R_{it}R_{it+k},$$

for $k \in \{0, 1, 2, \ldots, 5\}$. We regress these moments on age and cohort dummies as follows:

$$m_i^{t,t+k} = \eta + \sum_{a=0}^{35}\sum_{j=0}^{5} \delta_j^t I[k=j]I[a=25+2t] + \sum_{c=1}^{n_c} \theta_c I[C_i=c] + \varepsilon_{ik}^t.$$

Note that the omitted age effect dummy is δ_0^{35}. Individual-specific cohort-adjusted moments are then given by

$$\tilde{m}_i^{t,t+k} = \hat{\delta}_k^t + \varepsilon_{ik}^t + R^2_{35} - \tilde{m}_0^{35},$$

where $\tilde{m}_{35}$ is the mean variance moment for individuals in the 35–36 year-old age group. The last two terms scale the moments so that the raw and cohort-adjusted variances are the same for this age group. The cohort-adjusted variance/covariance profiles are the means of these individual-specific cohort-adjusted moments for each age group:

$$\tilde{m}_k^t = \sum_i \tilde{m}_i^{t,t+k}.$$

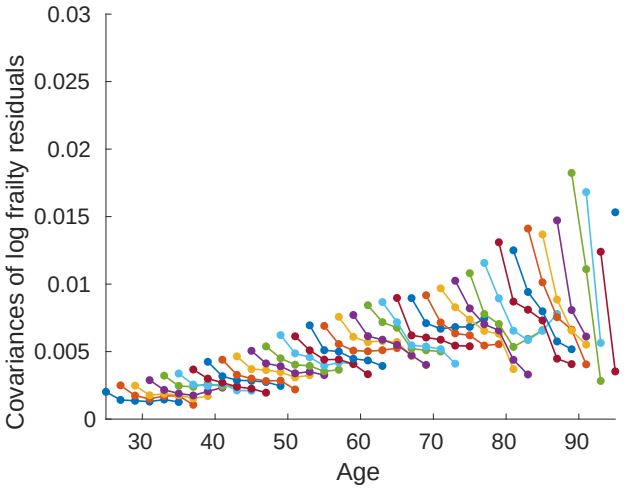


Figure 9: Raw covariances of the residuals, R_{it}, by age in PSID.

A.3 Additional regression results

Table 19 provides results from running the OLS regressions on individuals aged 51–95. There are some important differences between the HRS results and PSID results in Table 2. First, in the HRS the effect of age on frailty becomes stronger with age at a faster rate than is found in the PSID sample. Second, while being male increases frailty in the PSID sample. It lowers frailty in the HRS samples.

	HRS ages 51–95 sample	
Variable	Coefficient	Std. Err.
Age	-0.0063	(0.0005)
Age^2	0.0001	(3.71e-06)
Years of School	-0.0092	(0.0001)
Male	-0.0126	(0.0006)
Married	-0.0265	(0.0006)
Const.	0.2792	(0.0255)
Cohort dummies included		
	N=148,063, R^2=0.1998	

Table 19: OLS regression results for log frailty using HRS sample: ages 51–95.

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