How should we model health as a dynamic process?

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Abstract

Health is a complex dynamic process that impacts many economic decisions in ways that remain poorly understood. This chapter comprehensively reviews how health is modelled in the literature, showing that baseline models typically fail to take into account how persistence and frequency of health shocks vary by past health history and magnitude and direction of past shocks. Methods from the earnings dynamics literature are adapted to produce improved health persistence estimates. This chapter also investigates how medical biomarker data can be incorporated in dynamic models of health as a proxy for underlying health. There is significant scope for further work in this area as more medical data becomes available to researchers.

JEL classifications: I10, I31, C5

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Contents

1 Introduction

Health is an important determinant of an individual's economic decision making, affecting labour supply, consumption, family composition and access to government-provided insurance. Accurately modelling health as a dynamic process is needed to answer several important and open questions in the literature, including what is the relationship between health and earnings inequality, how effective are the current government-provided safety nets for those who fall ill, and how best should governments respond to the increasing economic burdens of chronic disease, rising disability rates and an ageing population. There has been a significant amount of reduced-form work on these questions (Prinz et al. (2018) provides a good summary). More recently, structural approaches have been used to better understand some of the complex endogeneity between health and economic decision making. These models typically capture health dynamics in a highly simplified way to minimise computational burden. The contributions made by this chapter are relevant to both these reduced form and structural approaches.

This chapter makes three key contributions to the literature. Firstly, it provides a comprehensive assessment of the different ways health dynamics have been modelled in the literature. Researchers have typically borrowed from the earnings dynamics literature and modelled health as a simple linear process such as an autoregressive moving average process, often discretised as a first-order Markov process, or the sum of a permanent and transitory shock. To the author's knowledge, there have been no prior attempts to systematically evaluate these modelling approaches and their underlying assumptions. I use Understanding Society data, a commonly-used UK household panel dataset, to replicate the most common models of health dynamics using standard panel data techniques. I then evaluate how well they capture key features of the health process, focussing on estimates of persistence and cross-sectional heterogeneity caused by different health shock realisations. I show that an ARMA(1,1) model with a large AR(1) coefficient close to one and a moderately-sized negative MA(1) coefficient fit the data reasonably well. An alternative linear model that combines a permanent process and a transitory process can be a desirable alternative as it allows for two types of shocks with different properties, but at the expense of some very strong and potentially incorrect persistence assumptions.

One of the most important components of health dynamics to accurately model is persistence. An individual is likely to respond very differently to a highly persistent health shock compared to a moderately persistent one. In the ARMA(1,1) model, the AR(1) term captures the average persistence of health from last period, which is then modified by the MA(1) term depending on the magnitude of the prior period's error term. I show that persistence heterogeneity is much greater than captured by this model, and varies systematically by past health and the features of the health shock. On average, health shocks are more persistent if they are negative (a decline in health rather than an improvement), if the individual was in poor health prior to the shock, and if the health shock is large. The standard linear models of health dynamics do not capture this heterogeneity, and therefore tend to be overly-optimistic in modelling the health dynamics of those with a history of poor health who experience additional negative health shocks.

A related limitation is that these models are not particularly effective in capturing the different distributions of health shock risks that individuals face. While an ARMA(1,1) model can be estimated using GMM techniques that are fairly robust to various error distribution assumptions, the most obvious application of the model would impose a mean-zero independent and identically distributed (i.i.d.) normal error distribution, while the model that is a sum of a random walk and a moving average transitory process has a normally-distributed error term. I document several ways that the error terms, which I interpret as health shocks, deviate from an i.i.d. normal distribution. First, there is a strong relationship between past health and the expected distribution of future health shocks. Those in poor health face an increased risk of both large negative and large positive health shocks, while the variance of health shocks faced by those in good health is much lower. The variance of health shocks is higher for negative shocks than positive shocks, even when controlling for past health. Finally, the baseline models do not accurately replicate the higher order moments of the data.

The second contribution of this chapter is to address many of the limitations of these standard linear models of health by adapting a recent panel data technique from the earnings dynamics literature. I use Arellano, Blundell and Bonhomme (2017)'s quantile-based method to produce non-linear persistence estimates that allow for a large amount of heterogeneity. One attraction of this framework is that it allows for

persistence to vary depending on the size and sign of the health shock that occurs in period t, which cannot be done using the standard linear models due to the endogeneity between the shock and persistence estimates that relate health in period $t-1$ to health in period t. Applying this framework to my health data produces persistence estimates that range from 0.6 to 1.2, depending on prior health and characteristics of the shock in period t. This framework is able to capture that the persistence of the health process is higher among individuals in poor prior health, and that positive health shocks are typically less persistent than negative health shocks. These improved persistence estimates better capture the health risks faced by individuals, with implications for our understanding of the impact of health on economic decisions such as labour supply and consumption. I also estimate an extended version of this framework that is able to strip out time-invariant unobserved heterogeneity from the persistence estimates, and consider the wider applicability of the framework by applying it to produce non-linear persistence estimates of an index of mental health.

Finally, this chapter investigates how best to use increasingly-available medical data to improve health modelling. These data are available for a subset of individuals in the Understanding Society dataset. Previous studies have shown that biomarker data such as inflammation markers and steroid hormones in the blood can predict future adverse health outcomes in ostensibly healthy people (Davillas and Pudney, 2020*a*). To the author's knowledge, this data has never been used to better model health dynamics. I find that incorporating the biomarker data into my models of health dynamics does not improve their persistence estimates. However, the data can be used to better model the different health risks individuals face. I show that the ARMA(1,1) model performs less well in cases where the biomarker data suggest that an individual's underlying health is very poor. These are typically cases where an individual does not report any serious health conditions, but they face a significantly elevated risk of negative health shocks. This is an important source of risk to capture. I also find that variation in biomarker data is strongly correlated with the variation captured by the fixed effect component of the persistence estimates produced using the Arellano, Blundell and Bonhomme (2017) framework. This suggests that biomarker data can be used to better understand and model individual heterogeneity in health outcomes, a topic that remains poorly understood.

The remainder of this chapter is structured as follows. Section 2 reviews the

relevant literature and Section 3 describes the data, focussing on the construction of indices to capture observed and underlying health. Section 4 reviews the baseline dynamic health models in the literature and section 5 identifies their limitations. Section 6 applies methods from the earnings literature to produce non-linear estimates of persistence. Section 7 concludes.

2 Literature Review

There is a body of literature that develops methods of aggregating survey health data into an index of overall health, which I summarise in the data section of this chapter. However, answering questions on the relationship between health and economic outcomes often require us to take a stance on how health evolves over time. There is some reduced form work on this question (O'Donnell, Van Doorslaer and Van Ourti, 2015), but the most common approach in the literature has been to apply the vast literature on modelling earnings dynamics to model health as a simple linear process, most commonly as an ARMA(p,q) process or the sum of a persistent and a transitory component. In the structural literature, a discrete version of this approach; a first-order Markov process with a small number of discrete health states, has commonly been used. However, the implications and limitations of these models has only very recently begun to be examined in the literature. I review the modelling health as a dynamic process literature, highlighting the gaps that this chapter seeks to fill.

The canonical papers that model the time series properties of the mean of earnings, such as Lillard and Willis (1978), MaCurdy (1982), and Abowd and Card (1989) use panel data to fit ARMA-type processes to earnings data. A recent example of this approach applied to health data is Blundell et al. (2020), who represent health ($\widetilde{h}_t$) using the error correction model: $\widetilde{h}_t = \pi_t + \varepsilon_t$. The persistent component (π_t) evolves as a random walk: $\pi_t = \pi_{t-1} + \eta_t$, and ε_t is a MA(0) transitory component. Alternative specifications in the literature include modelling the persistent component as an AR(1) or higher order process so the effect of a shock to the persistent component decays over time, and adding more structure to the transitory component, such as by incorporating moving-average terms (Blundell et al., 2016), or by modelling health as a stock that decays (Wallenius, 2020). To reduce dimensionality, health processes that are included in structural models are typically discretised into a first-order Markov process that models transitions between discrete health states. There are many examples: Palumbo (1999), French (2005), De Nardi, French and Jones (2010), Attanasio, Kitao and Violante (2010), French and Jones (2011), Capatina (2015), Jung and Tran (2016), Braun, Kopecky and Koreshkova (2017), Imrohoroglu and Zhao (2018), Jolivet and Postel-Vinay (2020), Nygaard (2021) and Amengual, Bueren and Crego

(2021). Earlier structural papers typically only modelled two health states, good and bad health, while more recent papers tend to include additional states, for example Jolivet and Postel-Vinay (2020) model four states of mental health: good, average, poor and severe. Some of these papers endogenise the health process by incorporating the impact of choices such as unhealthy consumption of cigarettes (Nygaard, 2021) or medical expenditure choices (Prados, 2018). Zweifel, Breyer and Kifmann (2009) model people choosing the level of health investment to marginally alter their transition probabilities between different health states. An important distinction between these Markov models and ARMA models is that the latter imposes a symmetry between positive and negative health shocks. Markov models do not have this feature, and the data suggest that the transition probability from good to poor health differs from the transition probability from poor health to good health.

Both ARMA and Markov models emphasise the state dependency of the health process. This can downplay the importance of individual heterogeneity in explaining the large cross-sectional variance in health observed in the data. Halliday (2008) finds that individual characteristics that trace back to childhood and early adulthood play an important role in determining how long health shocks persist, while the importance of state dependence varies significantly. However, he acknowledges that his first-order Markov model with only two health states limits his ability to pin down state dependence. Hauck and Rice (2004) similarly emphasise the importance of individual heterogeneity relative to state dependence in modelling mental health transitions. Pashchenko, Porapakkarm and Nardi (2017) find that variation in health transitions due to 'health types' is much larger than variation due to state-dependence for men with a high-school education. Of particular interest is some ongoing work recently presented by De Nardi (2024), which identifies health types with different expected health trajectories. Performing clustering analysis on frailty measures, they identify five health types that they label as vigorous resilient, fair-health resilient, fair-health vulnerable, frail-resilient, and frail-vulnerable. They find that these types explain a large share of subsequent health trajectories of older adults, and significantly outperform forecasts of health trajectories based on initial health and a rich set of observables. These classifications are based on a clustering algorithm and the authors do not attempt to explain what causes these different health types. However, the authors do highlight the recent empirical literature that emphasises the life-long

economic consequences of genetics and early childhood experiences such as Barth, Papageorge and Thom (2020), Conti and Heckman (2010), Case, Fertig and Paxson (2004), Harris et al. (2016) and Cronqvist and Siegel (2015). Understanding the nature of this individual heterogeneity is of central importance to answering questions such as what causes the relationship between health and education, or health and earnings inequality, which currently remains poorly understood.

Some of the recent papers containing structural models have made progress in capturing additional complexity of health dynamics, most commonly by adding an extra variable that varies health shock risk such as 'health type' or 'underlying health'. Pashchenko, Porapakkarm and Nardi (2017) augment a standard first-order Markov model of health with transition probabilities that also depend on the duration of the current health spell and 'health type', which is a proxy for individual heterogeneity and affects transition likelihood. They find evidence of 'duration dependence' where the longer that someone has stayed in a particular state of health the less likely they are to transition states next period. This is not consistent with a low-order Markov process of health dynamics. Salvati (2021) incorporates a similar fixed-effect variable which is described as a proxy for high or low health into her model of health. She embeds a health equation into her life-cycle model that consists of an AR(1) process, a binary fixed effect term, a labour-market health interaction term, and various independent variables. Ozkan (2017) models two types of health capital: physical health capital that determines survival probability and preventative health capital that is subject to health shocks and can be modified by health investment. Keane, Capatina and Maruyama (2020) make progress in modelling individual heterogeneity by incorporating an asymptomatic health risk variable estimated with medical data. In this model, individuals have functional health that is subject to three types of shocks: predictable and persistent shocks, unpredictable and persistent shocks, and unpredictable and transitory shocks. Asymptomatic health risk captures conditions such as high cholesterol, high blood pressure and high BMI that do not directly affect daily life but increase the probability for future predictable adverse shocks to functional health. While these models have made progress in capturing health dynamics, these equations tend to be a small component of large and complex structural models with computational demands that limit what these models can capture. The 'black-box' nature of these models can make it difficult to understand the mechanics

of the interactions between health and other variables. This chapter identifies some of limitations of modelling health in this way.

3 Data

The main dataset used in this chapter is Understanding Society - the UK Household Longitudinal Study. This is a longitudinal, nationally representative dataset with good coverage of health, education, employment, family life and income variables. I build an unbalanced panel using waves 1-12 of the study, which include observations from 2009–2021. Excluding a small number of individuals with insufficient health information results in a sample of 265,830 observations from 29,886 unique individuals. Table 1 reports the summary statistics of this sample and indicates good coverage over age, education, family type and employment.*

3.1 Health index construction

In many settings, the theoretically-ideal health index would be an overall stock of health measure, or a related concept such as a work-capacity index. Such an index would be continuous and bounded from below (death). Since these are unobservable concepts, we can instead construct a proxy index by aggregating various health data from household panel surveys. The available data can be grouped into three main categories. Objective health data are data on specific diagnoses and disabilities. Subjective health data are based on survey respondents' assessment of their own health. A third category of data is medical data such as pulse, blood pressure readings, blood tests or genomic data that can be used to predict health outcomes. Some of these medical data, such as genetic information, may be plausibly exogenous to any experiences or choices of the individual, which can be valuable for statistical analysis.

The limitations of each of these categories of health data as proxies for overall health has been thoroughly evaluated in several papers (Blundell et al. (2021) provides a good summary of this literature). To briefly summarise, objective measures are vulnerable to omitted variable bias, they can only capture a subset of relevant conditions, and often lack disease severity information. The rate of omission of life-changing medical diagnoses such as heart attacks and strokes reported by survey

*There is some gender imbalance in the sample (57% female, 43% male). This mostly reflects the raw Understanding Society data, which is split 55% female and 45% male. My sample is then further female skewed by men being more likely to enter the sample as proxies where partial information is provided about them by another household member, but they are unable or unwilling to respond themselves. Therefore, I do not have their subjective health scores and drop them from the sample.

Table 1: Summary statistics

	men	women
age		
<30	17,205	24,567
30-39	17,026	24,991
40-49	21,968	30,142
50-59	21,938	28,848
60-69	19,815	24,204
70-79	12,489	13,950
80-89	3,553	4,448
90+	282	404
education		
below GSCEs	25,743	31,135
GSCEs	31,755	41,889
A-level	13,476	16,622
degree	43,302	61,908
family type		
cohabitating/married	80,910	95,630
widowed	3,315	10,427
separated/divorced	7,111	16,091
single	22,772	29,068
number of children		
0	86,026	106,463
1	11,818	19,822
2	12,271	18,718
3	3,299	5,088
4+	862	1,463
currently employed		
yes	72,589	88,493
no	41,500	62,779
occupation class		
professional	6,324	4,820
managerial & technical	28,854	34,801
skilled non-manual	9,532	24,154
skilled manual	18,719	9,105
partially skilled	7,269	14,316
unskilled	2,651	2,005
N (observations)	*114,276*	*151,554*

respondents has been found to be surprisingly high when compared to linked hospital admission data, suggesting measurement error could be large (Caraballo et al., 2020). Subjective health measures are fairly crude and vulnerable to reporting error and justification bias. For a given disease presentation, people will vary hugely in how poorly they rate their health and to what degree they report that the disease has a negative impact on their life (French and Jones, 2017). Medical data are less commonly collected in household surveys and there is limited research on how best to use them to model health.

A challenge in the literature has been how best to use these data to construct an overall health index that minimises these biases and approximate the ideal theoretical health concept. Lack of consensus on this question has contributed to the ongoing uncertainty of the relationship between health and employment (Blundell et al., 2021). For example, large differences have been found when estimating the impact of poor health on labour supply using objective or subjective health data (Anderson and Burkhauser, 1984). To reduce these biases, a common approach has been to instrument subjective health data with objective data. This approach is still regularly used, with Blundell et al. (2020) being a recent example, although the approach is not without criticism. Bound (1991) argues that the different types of biases affecting subjective health measures roughly offset, so that incorporating objective health data adds little value and may increase bias. Alternative approaches to aggregating health data have included taking the first principle component over a large number of objective measures (Poterba, Venti and Wise, 2017), constructing multiple indices (Blau and Gilleskie, 2001), and converting medical conditions into World Health Organisation disability weights that represent the magnitude of health loss associated with specific health outcomes, which can then be aggregated (Prados, 2018). A helpful contribution was made by Blundell et al. (2021) who comprehensively evaluated the different approaches in the literature to identify how should health data be combined to best represent overall health. They conclude that objective measures, provided that a large enough set of them are used, subjective measures, and subjective measures instrumented with objective measures can produce similar estimates of the impact of health on employment, and any of these modelling approach can reasonably be used. This finding was broadly supported by Hosseini, Kopecky and Zhao (2022), who compares the performance of a 'frailty index' that aggregates objective indicators

with a subjective health index, and an index constructed using principal component analysis, and similarly finds that the predictive power of the different approaches to be broadly comparable.

I follow the literature and use both subjective and objective health data to construct a single health index that functions as a proxy for an individual's overall stock of health. The subjective data comes from the survey question '*In general, would you say your health is: poor, fair, good, very good or excellent?*'. The objective health data used is reported in Table 2.

Table 2: Objective health indicators

Objective measure	Data
Disabilities (specified as causing 'some difficulty' or 'much difficulty')	12 indicators: manual dexterity, mobility, lifting/moving objects, continence, hearing, sight, communication/speech, memory/ability to concentrate and learn, recognising danger, physical co-ordination, personal care, other
Mental wellbeing	General Health Questionnaire (GHQ) Caseness measure. Measures common mental health problems e.g. depression, anxiety, somatic symptoms, social withdrawal to detect those at risk of developing psychiatric disorders.
Ever diagnosed with condition	asthma, congestive heart failure, coronary heart disease, angina, heart attack, stroke, emphysema, hypothyroidism, chronic bronchitis, liver condition, epilepsy, hypertension, multiple sclerosis, COPD, osteoarthritis, rheumatoid arthritis, other arthritis, cancers: bowel/colorectal, lung, breast, prostate, liver, skin, other, diabetes: type 1, type 2, gestational and other, anxiety, depression, psychosis/schizophrenia, bipolar/manic depression, eating disorders, PTSD, other emotional/nervous/psychiatric condition, other chronic condition
Still have previously diagnosed condition	Conditions: asthma, congestive heart failure, coronary heart disease, angina, hypothyroidism, chronic bronchitis, liver condition, epilepsy, hypertension, COPD, osteoarthritis, rheumatoid arthritis, cancers: bowel/colorectal, breast, prostate and skin, type 2 diabetes, anxiety, depression, eating disorders, PTSD
Hospital out-patient	1-2 days, 3-5 days, 6-10 days, >10 days in the past year
Hospital in-patient	1-2 days, 3-5 days, 6-10 days, >10 days in the past year

To construct a single health index, I follow the approach of Blundell et al. (2020) and estimate an ordered probit of an individual's subjective reported health on a rich dataset of objective health measures, and then take the predicted values from this regression to be the individual's health index. I run the following ordered probit regression where H^*_{it} is the unobserved continuous latent general health variable and H_{it} is the observed ordinal general health score assessed by the individual in period t. $H_{it} = \{1, 2, 3, 4, 5\}$ where 1 = poor, 2 = fair, 3 = good, 4 = very good, and 5 = excellent. X_{it} is a vector of objective measures and some additional controls, and ϵ_{it} is the individual error term. The included controls are age, sex, an employment dummy, occupation class, and month and year dummies. The Pseudo-R square from these ordered probit regressions is around 0.2. Each wave is estimated separately, and a sample of the regression output is reported in Appendix A.2.

$$H^*_{it} = X^{'}_{it}\beta_t + \epsilon_{it}, \quad \epsilon_{it} \sim \mathcal{N}(0,1) \;\; \forall \; i = 1...N, t = 1...T$$

$$H_{it} = j \quad \text{if } \mu_{jt} < H^*_{it} < \mu_{j-1,t} \quad j = \{1, 2, 3, 4, 5\}$$

The probability that individual i selects general health value j in period t is:

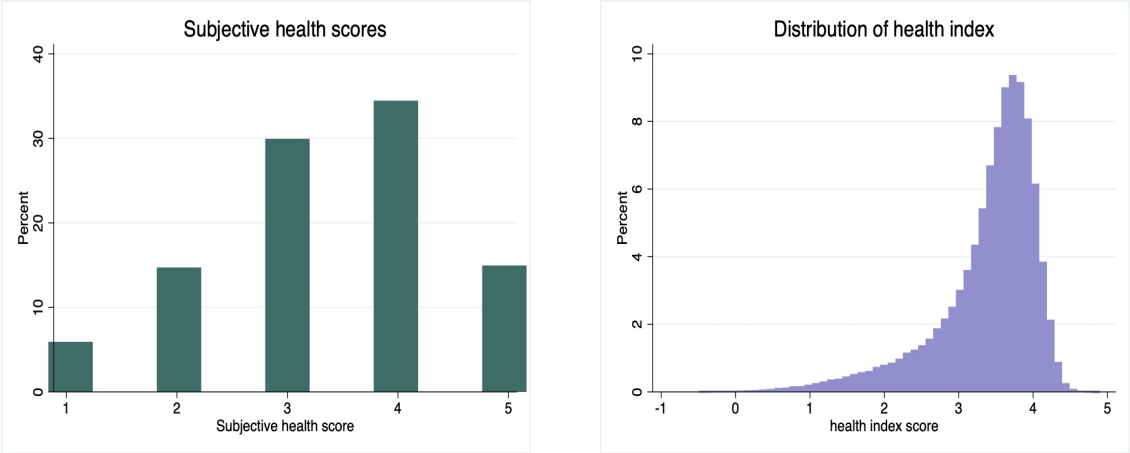
$$Pr(H_{it} = j) = \Phi(\mu_{jt} - X^{'}_{it}\beta_t) - \Phi(\mu_{j-1,t} - X^{'}_{it}\beta_t)$$

I then map the ordered probit fitted values onto the general health scores using a linear regression of the subjective scores onto the predicted values, and re-calculating the fitted values. The distribution of the original subjective health scores and constructed health index is shown in Figure 1.

The constructed health index can be interpreted as the average subjective health score reported by all individuals with the same medical diagnoses and disabilities, controlling for individual characteristics such as age and sex. The distribution of these scores is left-skewed due to a large tail of individuals in poor health, and its kurtosis is around double that of a normal distribution, with many individuals bunching around the modal score.

Figure 2 indicates that differences in health index values between men and women are small. I include men and women in the same regression to calculate the health indices but include a gender dummy variable to allow for variation by gender. Average

Figure 1



The left hand side figure shows the raw health data; the right hand side shows the constructed health index data distribution

health index scores gradually decline with age, although they are fairly stable between the ages of 55-65. Variance in health scores increases with age, especially from the age of around 50. To strip out this decline in health by age, I demean the health index by regressing the health index against age, higher powers of age up to order four, sex, and month/year dummies. The residuals from this regression become the 'demeaned health index' that I use to model health as a dynamic process in subsequent chapters of this thesis

Figure 2: Distribution of health index values by age and gender

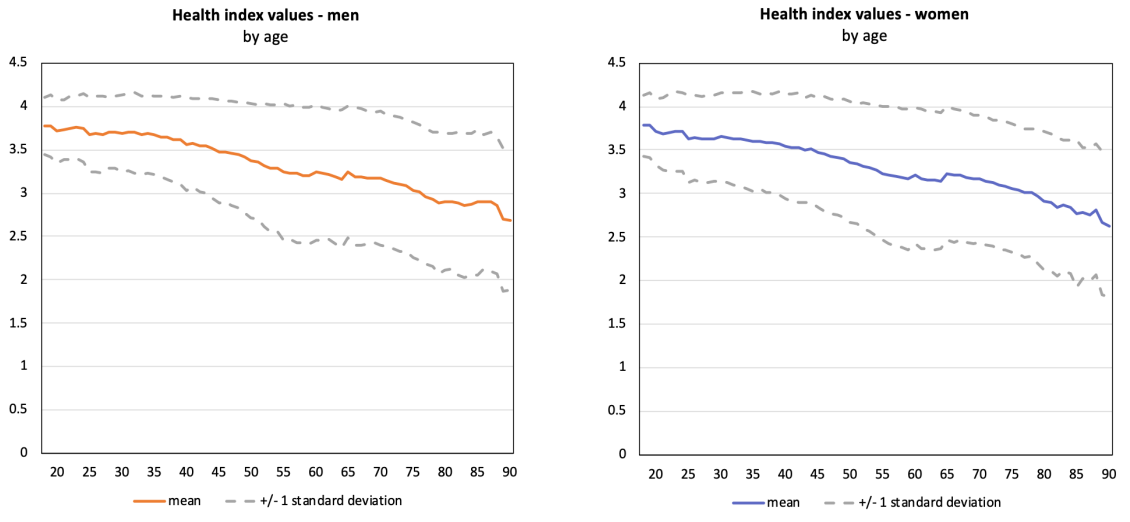
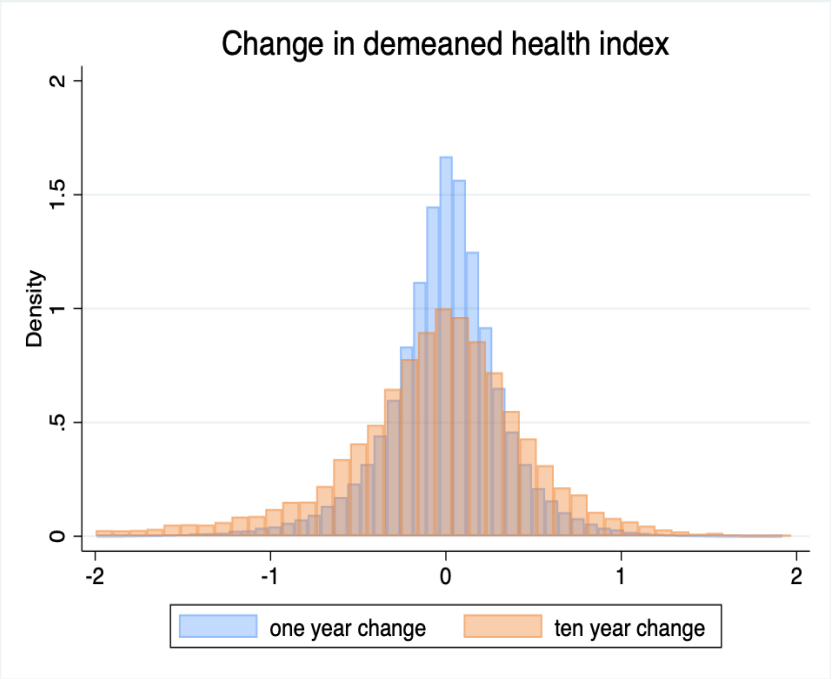


Figure 3 shows the distribution of changes to an individual's demeaned health

index over one year and ten years. In both cases the distribution has a slight negative skew, of -0.5 and -0.8 respectively. The approximate symmetry of shocks supports the use of simple linear ARMA models that impose symmetry of shocks.

Figure 3



A potential concern is that attrition rates vary systematically by health. In my dataset, around 18 per cent of observations do not have an observation next period, either due to attrition or missing data. Estimating a linear probability model of attrition indicates that those in the lowest health quintile are two percentage points more likely to not report health data next period relative to those in better health. However, the literature is fairly sanguine about the risks of using health indices for economic research when there is differential attrition risk by health (Jones, Koolman and Rice, 2006; Pudney and Watson, 2013). I choose to follow this literature and do not atempt to adjust for attrition rates in my subsequent modelling of health dynamics. Further analysis of attrition in my dataset is reported in Appendix A.1.

3.2 Allostatic scores from biomarker data

Between 2010–12, a subset of 8,465 individuals from waves 2 and 3 of the main Understanding Society survey were visited by a nurse for a physical health check and gave a blood sample. I use this biomarker (biological marker) data to construct a second index that approximates a component of underlying health called 'allostatic load'. This is a medical concept that reflects the risk from the cumulative effects of exposure to physical, psychosocial and environmental stressors that increase the risk of developing chronic diseases (Group, 2001). As a measure of cumulative wear and tear to the body, allostatic load is theoretically quite close to overall health stock or working capacity, although it cannot capture mental health or physical injury or disability.

Biomarker data can be used to improve health dynamics modelling for several reasons. They can be measured with less error than other health data that rely on an individual accurately describing their health. The availability of biomarker data is likely to grow rapidly following the increasing popularity of wearable health technology such as smart watches. They can help predict future health and mortality risk in ostensibly healthy individuals (Davillas and Pudney, 2020*b*,*c*). Davillas and Pudney (2020*a*) find that combining subjective health data with biomarker data significantly improves their predictions of future disability risk. This is because biomarker data incorporate health information such as kidney function and hormonal balance that may not be known by the individual, and because it offsets people's bias towards over-weighting certain health information such as obesity and blood pressure, and under-weighting other information such as strength and lung function. Biomarker data can also help disentangle the endogeneity between health and economic outcomes, and have been used to better understand the income-health gradient (Davillas, Jones and Benzeval, 2019), the impact of economic insecurity and childhood economic circumstances on health (Niedzwiedz et al., 2017; Davillas and Jones, 2020), and comparing the health impact of becoming re-employed in poor quality work compared to remaining unemployed (Chandola and Zhang, 2017).

To construct the allostatic score index, I normalise and then aggregate the biomarker data. I follow the approach of Davillas and Pudney (2020*a*) and take the simple average of the z-scores of 12 biomarkers and physical indicators reported in Table 3. I

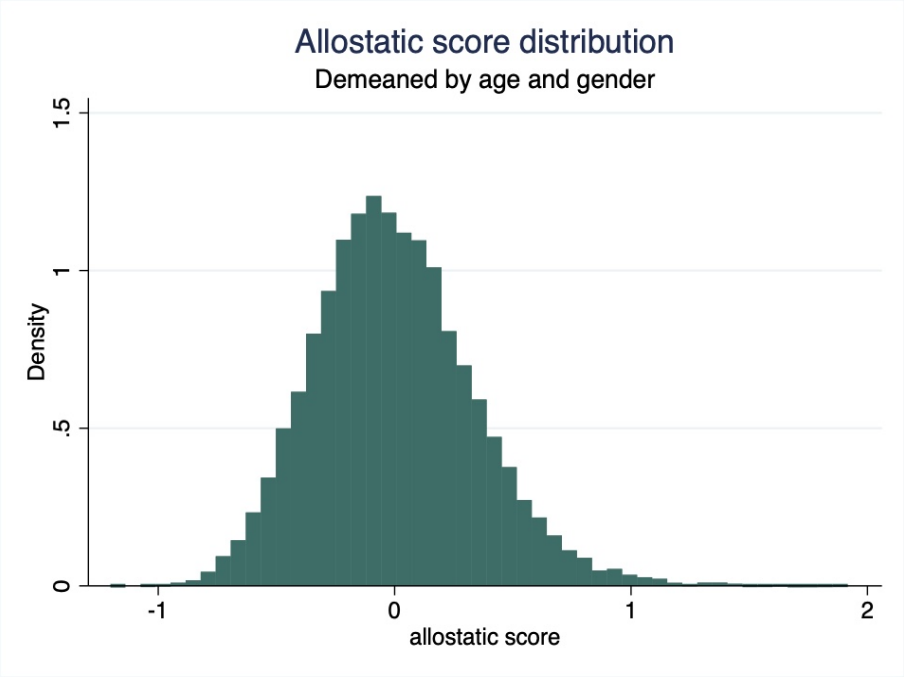
then demean the allostatic scores by age and gender to match how I constructed my health index. The subsequent distribution is approximately normal (Figure 4).

Table 3: Biomarkers used in allostatic load index construction

Indicator	Data	Description
waist-to-height ratio	waist circumference, body mass index	obesity indicator
pulse	resting heart rate	lower heart rate associated with more efficient heart function
blood pressure	systolic, diastolic	two readings treated as separate indicators
lung function	forced vital capacity (FVC)	total amount of air forcibly blown out after a full inspiration using a spirometer
blood sugar	glycated haemoglobin levels (HbA1c)	measures glucose intolerance, a good indicator of diabetes risk
inflammation	C-reactive protein (CRP)	is a protein in the blood that rises in response to general chronic or systemic inflammation. High levels are risk factor for cardiovascular disease and mortality.
kidney function	creatinine	Creatinine is a waste product of muscle function, which is passed through the kidneys and excreted in urine. Glomerular filtration rate (eGFR) calculated using creatinine data according to calculation cited in Benzeval et al. (2014). Indicates how effectively the kidneys are 'cleaning' the blood.
liver function	albumin levels	albumin is main protein made by the liver. Low levels may be indicative of a loss of liver function
steroid hormone	ihydroepian-drosterone sulphate (DHEAS)	one of the primary mechanisms through which psychosocial stressors may affect health. Low levels associated with cardiovascular risk and all-cause mortality
cholesterol	high-density lipoprotein cholesterol (HDL)	'good' cholesterol that helps remove other forms of cholesterol from the bloodstream. High levels lower risk of cardiovascular disease.
grip strength	maximum grip strength	correlated with overall body strength, lower scores associated with decreased physical function, disability and mortality

A limitation of these data is that I only have one set of biomarker observations per individual. However, the predictive content of allostatic scores is fairly stable over time. I show this by regressing my health index against the allostatic score index, varying the time gap between the data used for the health index and the allostatic score (Table 4). An allostatic score has similar predictive power for a

Figure 4



health index based on survey data collected one year later to a health index based on data collected ten years later. This suggests that allostatic scores capture a stable, long-term measure of health. There is a planned second round of biomarker data collection during wave 16 of Understanding Society in 2024–26, which can be used to check the stability of biomarker data over time more formally (Kumari, Al Baghal and Benzeval, 2022).

Table 4: Health index predictive content of allostatic scores

	Number of waves between collection of allostatic score and health index data								
	1-2	2-3	3-4	4-5	5-6	6-7	7-8	8-9	9-10
allo.	0.527***	0.533***	0.526***	0.517***	0.522***	0.562***	0.583***	0.468***	0.439***
	(0.0208)	(0.0207)	(0.0218)	(0.0217)	(0.0219)	(0.0231)	(0.0240)	(0.0240)	(0.0237)
R-sq	0.080	0.082	0.073	0.073	0.075	0.083	0.086	0.061	0.058
Obs	7434	7456	7400	7269	7024	6519	6249	5895	5536

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Alongside biomarker data, genetic data was also collected. However, the Understanding Society genetic data is safeguarded special license data. Therefore, I perform some preliminary analysis using an alternate dataset, The English Longitudinal Study

of Ageing (ELSA), on whether genetic data can be used as an additional health risk indicator when modelling health dynamics. The ELSA dataset reports polygenic scores for a variety of behavioural, emotional and health-related phenotypes, which estimate an individual's genetic propensity to develop various physical and mental health conditions. However, I find that while the polgenic scores do contain additional information on future health outcomes not captured by the the health or allostatic indices, the size of the additional information is too small to significantly improve my modelling of the overall health process. Further details of this analysis is reported in Appendix A.3.

4 Modelling health as a dynamic process

Health is a complex dynamic process that is subject to shocks that vary in magnitude and persistence. Heterogeneity between individuals is also large. In this section, I use panel data techniques to identify how best to model health as a simple linear process. I estimate two baseline models that replicate the two most commonly used approaches to modelling health dynamics: an ARMA(p,q) model, and a linear additive shock model that is the sum of a permanent process and a transitory MA(1) process. I find that an ARMA(1,1) model with a large AR coefficient and a moderately-sized negative MA coefficient best fits the data, although there are circumstances where the extra flexibility of the linear additive shock model in capturing two different shocks may be desirable. I then evaluate how effective these models are in capturing health dynamics accurately. I show that while these models can be appealing due to their simplicity and intuitive interpretation, they have some important limitations that I discuss in detail in the next section.

4.1 ARMA(p,q) baseline model

The two data attributes that I wish to capture in any baseline model of health are the persistence of innovations, and cross-sectional heterogeneity between individuals. My starting point is the simplest linear models that incorporate persistence; the autoregressive moving average (ARMA) class of models. I model health of individual i in period t (h_{it}) as an ARMA(p,q) process that includes a fixed effect μ_i:[†]

$$h_{it} = \sum_{k=1}^{p} \rho_k h_{i,t-k} + \sum_{j=1}^{q} \theta_j \varepsilon_{i,t-j} + \mu_i + \varepsilon_{it} \tag{1}$$

$$i = 1...N, \quad t = 1...T$$

The p lags of the ρ term make up the autoregressive AR(p) components, and the q lags of the θ term make up the moving average MA(q) components. It is important to note that the health process I estimate is based on data that has been detrended by age and gender. This was done by regressing the raw health index

[†]The ARMA models I describe in this section all allow for individual-specific fixed effects unless otherwise stated

against these observable variable and taking the residuals as the detrended health index. This detrending is quite common in the literature, perhaps due to familiarity with modelling the component of earnings growth that is unexplained by observables such as experience and education. Furthermore, detrending by age removes the time trend as health declines over time, reducing the risk that the process is non-stationary. Small changes in survey design between waves is controlled for by including time dummies. Nonetheless, it may be attractive for the researcher to explicitly model the decline in health as people age. I replicate the key empirical work in this section with the original non-detrended health index, and report the results in Appendix A.4. I find that my results are robust to using a non-detrended index.

I test for stationarity, and find that at least a significant proportion of the series is stationary. I use the Born and Breitung (2016) test for panel series correlation, as it is designed to be robust to fixed effects and heteroskedasticity. A non-stationary pure random walk model would result in the autocorrelation of differenced health with its second (and higher) lag to be zero, which is not what we observe. Instead, this pattern of gradually decreasing autocorrelation in first differences is consistent with a persistent autoregressive process or a MA(q) process with a large q.[‡]

In general, the literature finds mixed evidence of health following a random walk process as opposed to a highly persistent one. Blundell et al. (2020) do find evidence of a random walk, while Blundell et al. (2016) estimate the coefficient on the first lag of health to be 0.9-1.1 depending on the sub-sample used, and Heiss, Venti and Wise (2014) estimate an overall coefficient of 0.9. Whether papers model health as highly persistent or permanent processes likely reflects sample selection or health index construction. For example, the use of a dataset such as ELSA or Health and Retirement Study (HRS) that only includes older individuals will have a higher proportion of highly-persistent health shocks compared to a more representative sample by age, which will contain a higher proportion of less-persistent health shocks such as changes in mental health index scores. There are also different ways to construct health indices, and some may place more weight on more permanent health indicators

[‡]It is also interesting to note that the sign of the LM(k) test statistic in levels swaps from positive to negative from lag 4, indicating that health indices are positively correlated over short periods but negatively correlated over long periods. This is inconsistent with an ARMA(1,1) process, and could be explained by a combination of mean reversion and sample attrition. For example, an individual in poor health in period t is likely to also be in poor health in period t+1 or period t+2 but recovers by period t+4 or attrits from the sample

Table 5: Born and Breitung test for panel series correlation

	levels		first difference	
	LM(k)-stat*	p-value	LM(k)-stat	p-value
lag 1	36.30	0.000	-44.24	0.000
lag 2	24.63	0.000	8.36	0.000
lag 3	8.09	0.000	9.20	0.000
lag 4	-14.10	0.000	7.69	0.000
lag 5	-25.87	0.000	3.16	0.002
lag 6	-23.97	0.000	8.52	0.000
lag 7	-25.21	0.000	2.87	0.004
lag 8	-24.45	0.000	1.82	0.068
lag 9	-20.21	0.000	1.90	0.057
lag 10	-15.72	0.000	-1.22	0.224

*LM(k) test statistic is a modified t test of $\zeta = -1/(T-1)$. ζ from equation $h_{it} - \overline{h_i} = \zeta(h_{i,t-k} - \overline{h_i}) + \epsilon_{it}$. k is the lag order being tested

such as disability diagnoses compared to indicators of temporary health conditions such as infectious disease history or mental health indexes. Blundell et al. (2020) used ELSA data and constructed a health index that emphasised disability indicators, therefore it is unsurprising that they find evidence of a random walk.

I begin by estimating an AR(p) model using OLS with various values of p and no accounting for fixed effects, reported in Table 6.[§] The OLS estimates indicate that health is highly persistent, with the sum of coefficients on the lagged health terms consistently around 0.9. A major concern of using OLS is that the coefficient estimates may be spuriously high due to the presence of fixed effects. I strip them out using first differencing and avoid the resultant Nickell bias by using GMM estimation techniques. I use the Arellano-Bond 'Difference GMM' estimator which mitigates Nickell bias by instrumenting the lagged dependent variable terms with further lagged terms in levels. I re-estimate the AR(p) model now accounting for fixed effects, and adopting the following specifications which are selected to be conservative and robust: two-step estimator, time dummies, robust standard errors clustered at the individual level and an 'unadjusted' initial weighting matrix. I include the Windmeijer correction to correct for the usually negative bias in finite samples when the two-step estimator is used (Windmeijer, 2005). To prevent over-proliferation of instruments, I 'collapse'

[§]The sample size for the OLS and GMM estimations differ as the latter typically requires a higher t (additional lags) to generate the moment conditions

the instrument set and only include instruments based on the first to fifth lag of the variable being instrumented. My results are robust to various alternate specifications such as forward orthogonal deviations and different weighting matrices. I report the results of this exercise in Table 7. The MA(0) and MA(1) specifications indicate whether I allow the first lag ($h_{i,t-2}$) to be used as an instrument in the first-differenced equations, which is a valid instrument if the errors follow an MA(0) but not MA(1) process.

Table 6: OLS estimates of the health process as AR(p) model

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
L1.health	0.850***	0.602***	0.535***	0.503***	0.485***	0.470***	0.469***	0.454***
	(382.52)	(165.76)	(117.42)	(96.94)	(82.66)	(70.34)	(62.31)	(49.62)
L2.health		0.300***	0.244***	0.221***	0.213***	0.197***	0.203***	0.208***
		(81.97)	(47.88)	(38.65)	(32.27)	(27.06)	(25.85)	(22.96)
L3.health			0.135***	0.108***	0.0938***	0.0825***	0.0878***	0.0922***
			(32.11)	(18.63)	(13.57)	(10.67)	(10.23)	(9.33)
L4.health				0.0801***	0.0646***	0.0613***	0.0646***	0.0643***
				(16.43)	(9.61)	(7.75)	(7.15)	(6.23)
L5.health					0.0533***	0.0350***	0.0352***	0.0423***
					(8.93)	(4.29)	(3.79)	(3.89)
L6.health						0.0592***	0.0335***	0.0351**
						(8.45)	(3.55)	(2.99)
L7.health							0.0176*	-0.00891
							(2.17)	(-0.73)
L8.health								0.0149
								(1.48)
Observations*	228,886	182,016	146,353	117,013	89,994	69,512	52,415	37,934

Standard errors in parentheses; clustered standard errors, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 7: Difference-GMM estimates of the health process as an AR(p) model

	AR(1)		AR(2)		AR(3)		AR(4)		AR(5)	
	MA(0)	MA(1)	MA(0)	MA(1)	MA(0)	MA(1)	MA(0)	MA(1)	MA(0)	MA(1)
L1.health	0.241***	0.944***	0.478***	0.971***	0.571***	1.008***	0.574***	1.184***	0.614***	0.979***
	(0.00949)	(0.0342)	(0.0152)	(0.0528)	(0.0196)	(0.0991)	(0.0235)	(0.156)	(0.0281)	(0.134)
L2.health			0.147***	-0.0106	0.203***	-0.0453	0.223***	-0.190	0.241***	-0.00671
			(0.00748)	(0.0186)	(0.0101)	(0.0560)	(0.0125)	(0.103)	(0.0154)	(0.0888)
L3.health					0.0681***	-0.0101	0.0871***	-0.0605	0.0916***	-0.00324
					(0.00678)	(0.0195)	(0.00861)	(0.0375)	(0.0106)	(0.0352)
L4.health							0.0351***	-0.0170	0.0380***	-0.00104
							(0.00688)	(0.0158)	(0.00894)	(0.0169)
L5.health									0.00398	-0.00925
									(0.00850)	(0.0106)
AB test, order 1, z score	-60.29	-31.22	-51.10	-18.73	-45.02	-9.10	-38.71	-6.38	-34.47	-6.14
AB test, order 1, p value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
AB test, order 2, z score	11.19	16.04	-1.79	9.89	2.00	4.58	-1.28	3.68	-2.32	2.64
AB test, order 2, p value	0.000	0.000	0.075	0.000	0.045	0.000	0.199	0.000	0.020	0.008
AB test, order 3, z score	-1.92	0.06	4.42	-0.47	-1.91	-0.54	0.75	0.66	2.65	2.49
AB test, order 3, p value	0.056	0.955	0.000	0.637	0.056	0.589	0.456	0.508	0.008	0.013
Hansen J test stat	550.65	0.91	127.31	2.51	35.64	6.83	28.25	5.51	20.60	10.99
Hansen J test p value	0.000	0.823	0.000	0.473	0.000	0.078	0.000	0.138	0.000	0.012
Moment conditions	16	15	16	15	16	15	16	15	16	15
Observations	222,095	222,095	184,734	184,734	151,622	151,622	121,698	121,698	94,513	94,513

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

These results strongly suggest that an ARMA(1,1) model best suits the data. All MA(0) specifications that include instruments based on the immediately preceding lag result in a strong rejection of the null of the Hansen J test, indicating that the model is wrongly specified. However, excluding this instrument, which the MA(1) specifications do, is typically sufficient to change the result of this test and fail to reject the null. For example, excluding $h_{i,t-2}$ as an instrument for $\Delta h_{i,t-1}$ and only using $h_{i,t-3}$ and earlier lags leads to the non-rejection of the null. This strongly suggests that the errors follow an MA(1) process. Crucially, the Arellano-Bond autocorrelation tests also identify autocorrelation only up to the second order in most specifications. For all MA(1) specifications (except AR(5)), I find no evidence for third-order autocorrelation in the error terms, which is the key requirement for validity of the instruments used if I allow for the error component to follow a MA(1) process. In addition, when I exclude the first lag as an instrument, the point estimate of the coefficient on the first lag of health is much higher at around unity while the coefficients on all the subsequent lags are small and not significant. This suggests that including only one lag of health is sufficient.

Incorporating some additional moment conditions by using Blundell-Bond 'System GMM' estimation leads to improved ARMA(1,1) estimates. It is well known that the Arellano-Bond estimator does not function well when persistence is high. At the limit, if health follows a random walk ($\rho_1 = 1$) then the difference GMM instruments are uninformative. Blundell and Bond (1998) suggest that there is a risk of serious finite sample bias at ρ_1 values of 0.8 and higher, although they show that the bias is smaller with very large samples. The System GMM estimator typically performs much better in these circumstances. The additional moment conditions can also contribute to more precise coefficient estimation. This is particularly helpful as having to only use further lags as instruments due to the MA(1) error structure increases the risk of weak instruments. The additional initial moment restriction of $\mathbb{E}(\varepsilon_{it} h_{i1}) = 0$ that is required for System GMM estimation is not a particularly onerous restriction for my data. Blundell and Bond (2023) state that this restriction holds automatically if the same process has generated the series for long enough before the start of the sample period. Since my first observation occurs at least 18 years after the the start of the health process, at birth, this may not be an unreasonable assumption.

Table 8 reports the AR(p) model coefficients estimated using System-GMM and

allowing for MA(1) errors. Differences between the Difference and System GMM coefficient estimates are small, although using System GMM leads to much more precisely estimated coefficients, especially for the first lag. The coefficient estimates of the first lag are mostly not significantly different for the AR(1) AR(2) and AR(3) specifications, and the coefficients on additional lags are typically not significant. Therefore, including only one lag is sufficient to capture the persistence dynamics of this model.

Table 8: System-GMM estimates of the health process as an AR(p) model

	AR(1)	AR(2)	AR(3)	(AR4)	(AR5)
			MA(1) assumption		
L1.health	0.872***	0.901***	1.032***	1.113***	1.149***
	(0.0123)	(0.0310)	(0.0790)	(0.0864)	(0.0980)
L2.health		-0.00940	-0.0775	-0.126**	-0.135**
		(0.0170)	(0.0430)	(0.0448)	(0.0481)
L3.health			-0.0194	-0.0361	-0.0432*
			(0.0166)	(0.0199)	(0.0217)
L4.health				-0.0160	-0.0115
				(0.00844)	(0.0116)
L5.health					0.00608
					(0.00720)
AB test, order 1 z score	-53.86	-26.25	-11.81	-11.8	-10.78
AB test, order 1 p value	0.000	0.000	0.000	0.000	0.000
AB test, order 2 z score	19.19	12.69	6.27	6.88	6.32
AB test, order 2 p value	0.000	0.000	0.000	0.000	0.000
AB test, order 3 z score	0.032	-0.54	-0.39	0.08	1.75
AB test, order 3 p value	0.974	0.588	0.693	0.935	0.080
AB test, order 4 z score	0.867	0.54	-0.16	-0.36	-1.32
AB test, order 4 p value	0.386	0.592	0.871	0.721	0.188
Hansen J test stat	6.42	8.259	8.109	11.27	35.24
Hansen test p value	0.170	0.143	0.23	0.127	0.000
Moment conditions	16	17	18	19	20
Observations	222,095	184,734	151,622	121,698	94,513

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

I conclude that the best single-equation linear specification to capture the health process is the following ARMA(1,1) that accounts for fixed effects:

$$h_{it} = 0.87h_{i,t-1} - 0.33\varepsilon_{i,t-1} + \eta_i + \varepsilon_{it} \tag{2}$$

Since the AR term has already been estimated as 0.87, I estimate that the co-

efficient on the MA(1) term (θ) is -0.33 by re-arranging the ARMA(1,1) model as: $h_{it} - 0.87h_{i,t-1} = \tilde{h_{it}} = \eta_i + +\theta\varepsilon_{i,t-1} + \varepsilon_{it}$. This is now a simple MA(1) process that can be estimated using GMM. I use the following three variance and covariance moments and report the coefficient estimates in Table 9:

$$\text{Var}(\tilde{h_{it}}) = \mathbb{E}\eta_i^2 + (1+\theta^2)\mathbb{E}\varepsilon^2$$

$$\text{Cov}(\tilde{h_{it}}, \tilde{h_{i,t-1}}) = \mathbb{E}\eta_i^2 + \theta\mathbb{E}\varepsilon^2 \quad \text{Cov}(\tilde{h_{it}}, \tilde{h_{i,t-2}}) = \mathbb{E}\eta_i^2$$

Table 9: GMM estimates of MA(1) process

	ρ =0.87
η_i	0.0380***
	(0.00285)
θ	-0.334***
	(0.00593)
ε_{it}	0.335***
	(0.00125)
Observations	222,095

The reported estimates in this table refer to the variance of the error components η_i and ε_{it}
Standard errors in parentheses,* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

As a robustness exercise, I check whether there are large subgroups with health dynamics that are better captured by a different linear model. If this were the case, describing the health dynamics of the entire sample using a single ARMA(1,1) model may be misleading. I use the Sarafidis and Weber (2015) K-means clustering algorithm to divide the sample into as many clusters as required for the estimated slope coefficients of an AR(1) model to be the same within each cluster, accounting for individual-specific fixed effects. The algorithm divides my sample into two groups, containing 40 and 60 per cent of the sample respectively. This suggests that only two groups are needed to capture any heterogeneity in model coefficients. I then estimate AR(p) models separately for each group using GMM, re-assessing whether including one lag is sufficient and whether the error structure follows an MA(1) process. The regression tables are reported in Appendix A.5, as well as some summary statistics for each group. I determine that the models for the two groups that best fit the data are an AR(2) and ARMA(1,1) respectively.

$$\text{Group 1: } h_{it} = 0.79h_{i,t-1} + 0.09h_{i,t-2} + \eta_i + \varepsilon_{it}$$

$$\text{Group 2: } h_{it} = 0.83h_{i,t-1} - 0.54\varepsilon_{i,t-1} + \eta_i + \varepsilon_{it}$$

The two models are quite similar. Both capture that health is a highly persistent process, and have an additional term that helps distinguish between highly-persistent health shocks such as chronic health conditions, and transitory health shocks. I conclude that an ARMA(1,1) model is sufficient to describe the entire sample and slope heterogeneity is not a significant concern.

4.2 Linear additive shock model

I conclude this section with estimating a slightly different model that allows for more flexibility in capturing shock persistence, but at the expense of imposing other restrictions. A specification used very commonly in the earnings dynamics literature, and sometimes in the health dynamics literature, relaxes the restriction of individuals being subject to only one type of shock. Instead, the variable is modelled as the sum of two independent random processes: a permanent shock process which is typically a random walk, and a transitory process which is either an MA(0) or MA(1):

$$y_{it} = p_{it} + \upsilon_{it}$$

$$\text{permanent process: } p_{it} = p_{i,t-1} + \zeta_{it}$$

$$\text{transitory process: } \upsilon_{it} = \varepsilon_{it} - \theta\varepsilon_{i,t-1}$$

An additive classical measurement error $r_{it} \sim N(0, \sigma_r^2)$ can also be included. This clear distinction between permanent and transitory shocks reflects the influence of Friedman's Permanent Income Hypothesis on earnings dynamics research, but it is also conceptually attractive as researchers can cleanly classify most income shocks as either temporary, such as overtime or one-off-bonuses, or permanent, such as a job change (Meghir and Pistaferri, 2004). A similar intuition for health shocks being divided into permanent shocks such as a physical disability and temporary shocks such as some mental health episodes is compelling, and adopted in papers such as Blundell et al. (2020) and Blundell et al. (2016).

The canonical moment conditions used to estimate these models require that the permanent process is a random walk to achieve identification. This is a strong assumption for my data. It is challenging to distinguish between highly persistent and random walk processes in small-T panel data with significant individual heterogeneity, and I cannot reject that the coefficient on the lagged health term is 1 in many of the ARMA(p,q) models I estimated using GMM. However, my serial correlation tests do indicate that a reasonable proportion of the data are best characterised as following a persistent process rather than a random walk. It is also unclear how robust the resulting coefficient estimates are to small violations of the random walk assumption implied by the moment conditions. Proceeding with caution, I use the following moment conditions to estimate health as the sum of a permanent walk and MA(1) transitory process. Letting g_{it} be a change in h_{it} (equivalent to $h_{i,t} - h_{i,t-1}$) we can identify the variance of the permanent component using the following moment condition from Meghir and Pistaferri (2004):

$$\mathbb{E}(\zeta_{it}^2) = \mathbb{E}\left[g_{it} \left(\sum_{j=-(1+q)}^{1+q} g_{i,t+j} \right) \right]$$

. Since we cannot separately identify the variance of any measurement error, the variance of the transitory shock, and θ, we can only use the moment conditions to place bounds on these coefficients with the following moment conditions:

$$\sigma_r^2 = \mathbb{E}(g_{it}, g_{i,t-1}) - \frac{(1+\theta)^2}{\theta}\mathbb{E}(g_{it}, g_{i,t-2})$$

$$\sigma_\varepsilon^2 = \frac{\mathbb{E}(g_{it}, g_{i,t-2})}{\theta}$$

By setting σ_r^2 to zero we can estimate the lower or upper bound of θ, which we assume is bounded between -1 and 1. The sign of $\mathbb{E}(g_{it}, g_{i,t-2})$ defines the sign of θ. In my case it is negative, therefore the maximum value of θ is the case where $\sigma_r^2 = 0$. I use these moment conditions to estimate the variance of the two shocks, as well as the coefficients of the MA(1) transitory process. These estimates are reported in Table 10. My estimates of the magnitude of the variances of the two shocks are quite similar to the findings of Blundell et al. (2016), although they do not find evidence of a MA(1) transitory process. Blundell et al. (2020) obtain quite different results

and argue that transitory and permanent shocks contribute fairly equally to health variance. However, they use a very different estimation strategy and do not use these canonical moments from the earning dynamics literature.

Table 10: Coefficient estimates of linear additive shock model

variable	estimate
$\mathbb{E}(\zeta_{it}^2)$	0.155***
	(47.26)
σ_ε^2 if $\sigma_r^2 = 0$	0.050***
	(40.89)
θ if $\sigma_r^2 = 0$ (upper bound)	-0.072***
	(-6.42)

t statistics in parentheses, *p<0.05, *p<0.01, *p<0.001

If transitory shocks do not explain much variation of the overall health process, then using an ARMA(p,q) model that only allows for one type of shock is sufficient. My results suggest that the permanent process is responsible for the majority of the variance in the health process over time, although the transitory process does make some contribution. In addition, the ARMA(p,q) model does not require the persistent shock to follow a random walk. I conclude that the ARMA(p,q) model is a superior fit for my data.

I further consider the limitations of these two models in the next chapter, and suggest some improvements.

5 Capturing more complex dynamics

The two linear health models estimated in the previous section are simple to use and incorporate into more complex structural models. However, there is a cost to their simplicity. Since the baseline ARMA(p,q) model attempts to capture the average persistence of a health shock, it imposes uniformity of persistence on shocks of different sizes, for positive and negative health shocks, and for individuals with very different levels of health and health histories pre-shock. I find evidence of significant heterogeneity in persistence once I allow persistence to vary by these characteristics. Simple extensions of the ARMA(p,q) baseline model can capture some of this variation, however we can make further progress with more sophisticated modelling approaches, which I discuss in the subsequent chapter. In addition, I do not need to assume stationarity or that the error terms follow a white noise process for my ARMA(p,q) coefficient estimates to be valid. However, I document some features of the error distributions that are important to capture when modelling the heterogeneity in health shock risk that individuals face. I show that biomarker data can be used to capture some of the elevated negative health shock risk faced by some individuals.

This section focusses on the ARMA(1,1) model as my preferred linear model, but most of the limitations I identify can also be applied to the linear additive shock model. De Nardi, Fella and Paz-Pardo (2019) provide a good summary of the key limitations of this model when applied to earnings data which are equally valid when using health data. They key model assumptions they identify that do not match the data are: age independence of the second and higher moments of the conditional distribution of both the transitory and persistent components, normality of the shock distribution, and linearity of the process of the persistent component.

5.1 Recent health history

The average persistence of a health shock varies significantly depending on the health history of the individual prior to the shock taking place. This makes intuitive sense; someone's capacity to recover from an illness is a function of how healthy they were just prior to getting sick. The MA term in an ARMA(1,1) model takes into account the size of the shock last period, but there is significant additional persistence information in the level of health. The simplest way to capture this would be to add

an interaction term to the baseline ARMA(1,1) model that assigns individuals to a quintile of their health just before the shock, and interact it with the lagged health term, which I report in Table 11.

Table 11: ARMA(1,1) model with interaction dummy for lagged health level quintile

	Difference-GMM	System-GMM
Q1 - lagged health index	0.966***	0.923***
	(0.0697)	(0.0163)
Q2 - lagged health index	1.102***	0.910***
	(0.159)	(0.104)
Q3 - lagged health index	0.754***	0.665***
	(0.100)	(0.0719)
Q4 - lagged health index	0.901***	0.829***
	(0.0569)	(0.0433)
Q5 - lagged health index	0.871***	0.868***
	(0.0349)	(0.0260)
AB test, order 1 z score	-24.76	-56.093
AB test, order 1 p value	0.000	0.000
AB test, order 2 z score	14.3	20.835
AB test, order 2 p value	0.000	0.000
AB test, order 3 z score	0.0567	0.0915
AB test, order 3 p value	0.9548	0.927
Hansen J test stat	25.925	67.035
Hansen J test p value	0.0388	0.000
Observations	222,095	222,095

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

GMM estimation specifications identical to those used in baseline model in section 4

This exercise of relaxing the restriction that the autoregressive parameter is common across quintiles of the lagged health index suggests that persistence is the highest for those with prior bad health, and lowest for those with prior average health. However, these results should be taken with extreme caution as the Hansen test strongly rejects the validity of the over-identifying restrictions used by both the Difference-GMM and System-GMM estimators. This approach also is unable to allow coefficients to depend on the sign or magnitude of the health shock between periods $t-1$ and

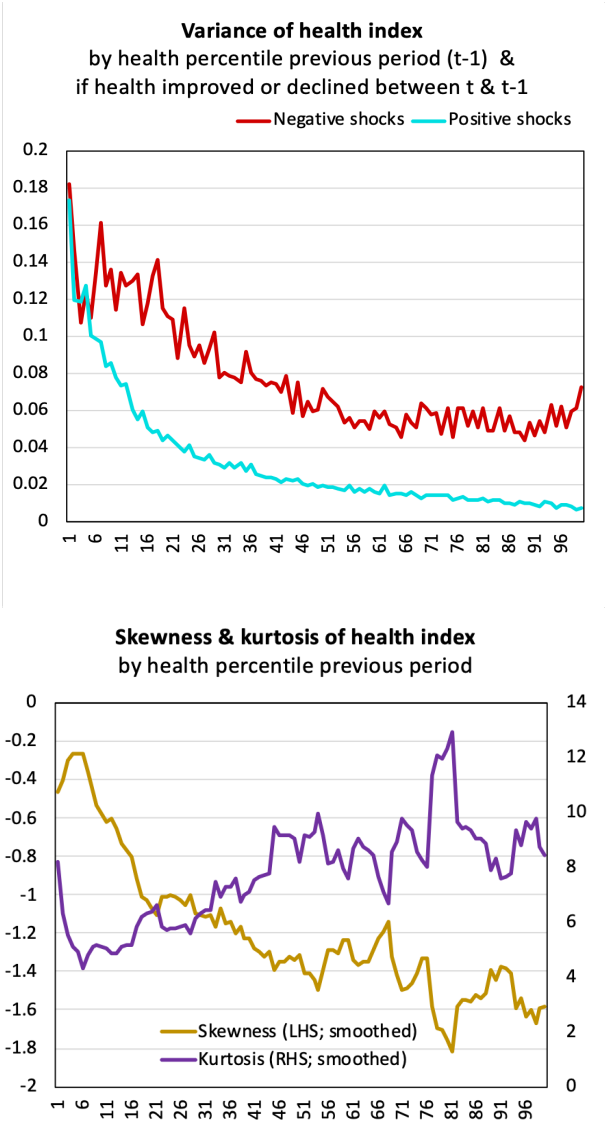
t. I adopt more complex econometric techniques in a later section of this chapter to capture this heterogeneity in the persistence of health shocks.

As well as the relationship between recent health history and persistence, there is also a relationship between recent health history and the expected distribution of future health shocks. This is difficult to capture in a simple linear model but is an important component of health risk to capture. To illustrate the relationship between past health and the expected distribution of health shocks, I graph the higher moments of the health index (variance, skewness, and kurtosis) as a function of that individual's health percentile in the previous period, where 1 is the bottom health percentile of all individuals and 99 is the highest health percentile in Figure 5. The variance depicted in the top panel is calculated separately for the subset of individuals who experienced a 'positive health shock', meaning their reported health improved between the current and immediately prior period, and those who suffered a negative health shock, which is defined as the opposite.

I find that variance, skewness, and kurtosis all systematically vary by health the previous period. Notably, those in poor health have more volatile health in subsequent periods, with increased risk of both large negative and positive changes to their health relative to those in good or average health. This elevated risk is difficult to capture using simple linear models. One plausible way of capturing this feature of the data, is estimating an autoregressive conditional heteroskedasticity (ARCH) model. ARCH models are able to capture differences in variance depending on the size of the error term in the previous period. For example, a large shock in period $t-1$ may mean a large shock is more likely in period t. Figure 6 compares the histograms of health changes between period $t-1$ and t for those who experienced a greater than one standard deviation change in health in the prior period (between $t-2$ and $t-1$) to those who did not. It shows that large changes in health is associated with more volatile health next period, and that, on average, a large negative health shock is associated with an improvement in health next period, and the opposite holds for those who experienced a positive health shock in the prior period. Interestingly, the distribution of health changes for those who experienced large positive or negative shocks in the prior period is closer to a normal distribution than the distribution of health changes of those who experienced neither. For this group, there is very little mass in the tails as stable health in the past is correlated with stable health in the

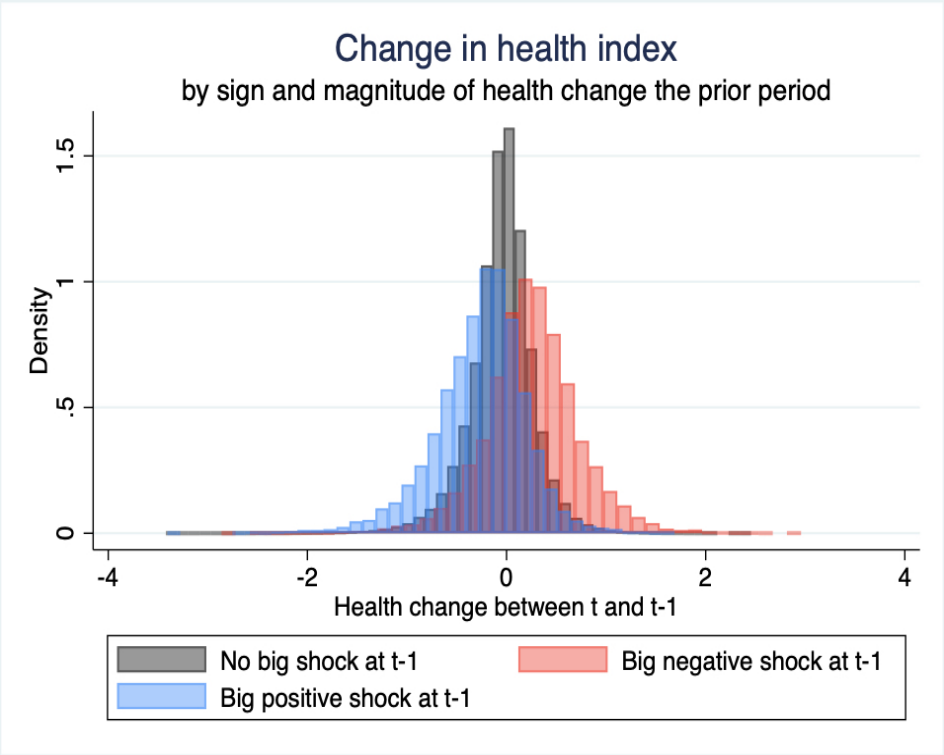
next period.

Figure 5



x-axis is individual's health percentile in the previous period, where (99=highest health percentile. y-axis is units of higher moment being graphed

Figure 6



I assess this relationship between errors and variance more formally by estimating an ARCH(1) model with the following specification for health variance: $\exp(\gamma_0 + \gamma_1 \varepsilon_{i,t-1}^2 + \gamma_2 \varepsilon_{i,t-1})$. The γ_2 term accounts for possible heterogeneity between positive and negative shocks. I describe my estimation procedure in Appendix A.6, but I do not find any evidence that γ_1 or $\gamma_2 \neq 0$, and therefore do not find evidence of ARCH effects in my data. However, this specification only models the relationship between shock magnitude in two consecutive periods. I do find evidence that individuals who experience a large negative health shock are more likely to experience another large negative health shock in subsequent years. However, a majority of these later shocks occur several years afterwards, which cannot be captured in an ARCH(1) model and requires a more complex econometric approach. Table 12 reports the number of large negative shocks, defined as at least one standard deviation fall in the detrended health index, experienced by those of different ages in the sample. Conditional on experiencing one negative shock, individuals are more likely to experience a second. For example, those aged 20-29 at the beginning of the sample period have a 16 per cent chance of experiencing a negative shock in the next decade, but 28 per cent

of those who experienced one negative shock experienced a second negative shock, with an average gap between shocks of four years. The average gap between negative health shocks rises with age.

Table 12: Number of large negative shocks over 10 years, population share by age†

Age in first wave	0 shocks	1 shock	2+ shocks	average yrs b/tween shocks
20-29	0.801	0.155	0.044	3.5
30-39	0.784	0.169	0.047	3.7
40-49	0.744	0.198	0.058	4.2
50-59	0.722	0.222	0.056	4.4
60-69	0.704	0.242	0.054	4.6
70-79	0.610	0.300	0.090	4.5

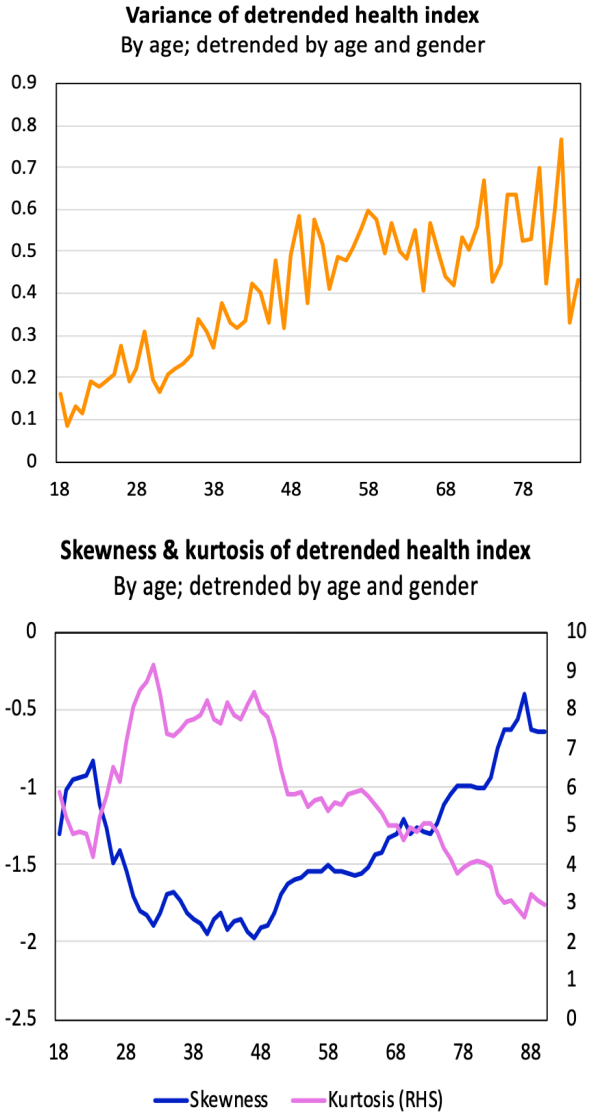
†shocks of at least one standard deviation

5.2 Age and model stationarity

A different source of heterogeneity in persistence and shock distribution is age of the individual. Age is closely related to the statistical property of stationarity. Since the time dimension of my panel data is fairly short, stationarity is difficult to assess. However, the ARMA(1,1) process I estimated is stationary in the long run, provided that $\rho + \theta \neq 0$, $\rho < |1|$, and some not particularly onerous restrictions are imposed on the distribution of ε_{it}. Stationarity implies that the moments of the data are age independent. For the first moment, this is mechanically achieved by detrending the health index by age and age polynomials. However, higher moments of the detrended health data are not age-independent. Figure 7 shows the second, third and fourth moments of the detrended health data by age. Older individuals are more likely to experience health shocks, and so the standard deviation of the health index increases with age. The distribution of the detrended health index of older people is less negatively skewed, reflecting their increased propensity to experience positive health shocks. Young people are much less likely to experience positive health shocks as their health is typically good and so cannot be improved further. The health index distribution for young people is platykurtic and so extreme health changes are rarer,

while the kurtosis for older people is close to a normal distribution. Higher moments do systematically vary by age, which should be captured in life-cycle models or models that consider the long-term impacts of health shocks on economic outcomes. This can be achieved by imposing a shock sequence that is a function of age rather than assuming a normal distribution for the error term. The ARMA(1,1) coefficients I estimate using GMM are robust to conditional heteroskedasticity and the patterns of kurtosis and skewness I identify. Assuming mean-zero errors and no serial correlation of the errors is sufficient for this to be the case (Arellano and Bond, 1991). However, higher moments are an important component of capturing the health risk people face.

Figure 7

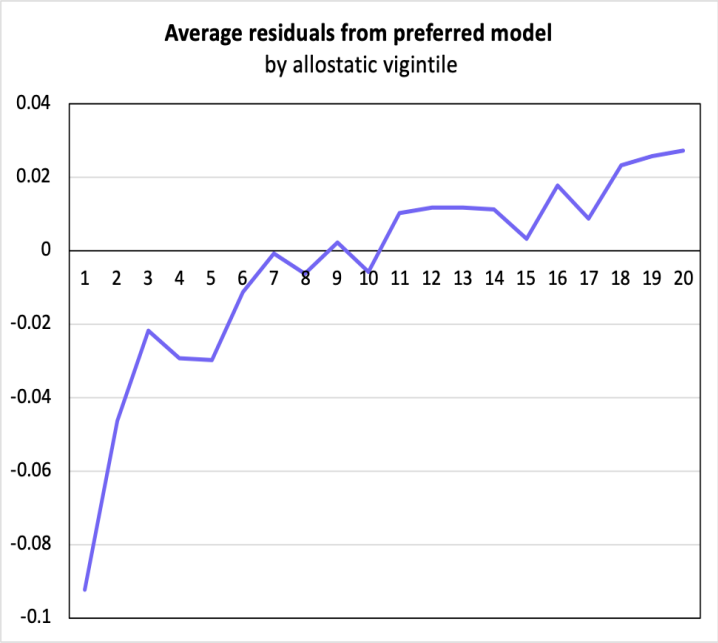


5.3 Underlying health

I conclude with considering how allostatic scores can be used as an additional data source to improve the performance of linear health dynamics models. I find that the main informational content of allostatic scores relates to the likelihood of a large negative health shock in the future. In addition, I find that allostatic scores do not help predict the persistence of already realised health shocks (see Appendix A.7 for further details).

The ARMA(1,1) model has the worst performance when predicting the health of individuals with poor allostatic scores. Figure 8 shows the average difference between the level of health predicted in period t using my preferred ARMA(1,1) model as estimated using System GMM (see section 4.1 for further details), and actual health in period t by allostatic score vigintile. These are the residuals for the health equations in levels, averaged for each allostatic score vigintile. The biggest forecast misses occurs for the population with the worst 10 per cent of allostatic scores, indicating very poor underlying health. If someone is in average health in period $t-1$ but has bad underlying health, they are much more likely to be hit by a large negative shock in period t. In these cases, the ARMA(1,1) model performs the most poorly and significantly overestimates their level of health. This result is in line with previous research that finds that biomarker data can predict future negative health outcomes among ostensibly healthy people (Davillas and Pudney, 2020*c*). This increased propensity to experience a large negative health shock is an important source of risk to capture in models of health dynamics.

Figure 8



6 Non-linear health dynamics

The complex dynamics of health, as described in the prior sections, can be better understood by adapting the latest panel data techniques. I estimate the health process using a non-linear panel data framework developed by Arellano, Blundell and Bonhomme (2017). This method is from the earnings literature, although it has been applied to a small number of non-earnings contexts, such as non-linear productivity and investment dynamics in firms (Melcangi and Sarpietro, 2024).[¶] A major attraction of this method is that it allows for heterogeneity in persistence to depend on the size and direction of the health shock that occurs in period t. This is not possible to do using the methods used to estimate the ARMA(p,q) models due to the fundamental endogeneity between the shock in period t and the persistence estimates that relate health in period $t-1$ to health in period t. This framework also allows persistence estimates to vary by the level of health in period $t-1$, which I previously showed can have a large impact on persistence estimates.

Adapting the Arellano, Blundell and Bonhomme (2017) framework to a health context produces persistence estimates that range from 0.6 to 1.2. While the linear

[¶]Dal Bianco and Moro (2022) have written a working paper concurrent to this one that also applies this framework to a health context

methods from the prior section produce persistence estimates around the midpoint of these estimates, this range is large enough to have meaningful implications for economic decision making. People faced with a health shock with persistence at the lower end of this range are likely to behave quite differently to those facing a much more persistent health shock. I also document some interesting patterns in how persistence estimates vary depending on whether the shock at period t is positive or negative, the magnitude of the shock, and the level of health immediately prior to the shock. I find that negative health shocks are more persistent than positive health shocks, and that negative health shocks are more persistent if someone was in poor health prior to the shock. I also estimate an additional model that includes fixed effects as an additional source of heterogeneity. Accounting for fixed effects does reduce the persistence estimates a little, especially for those in poor health who experience large negative shocks. I find some evidence that the size and sign of the fixed effect is correlated with allostatic scores which helps us understand the variation captured by the fixed effect. I conclude this section by extending this method to better capturing the complex dynamics of other health indicators by estimating the non-linear persistence of an index of mental health.

6.1 Non-linear persistence estimates of overall health

The non-linear framework of Arellano, Blundell and Bonhomme (2017) models their variable of interest as the sum of a persistent component (η_{it}) and a transitory innovation (ε_{it}). The linear model estimated in the previous section as also the sum of a permanent component and transitory innovation can be considered a special, highly-restrictive case of Arellano, Blundell and Bonhomme (2017)'s framework. The persistent component is assumed to follow a general first-order Markov process, and so the η_{it} terms are dependent over time, although the nature of their dependence does not need to be specified, allowing for flexible temporal dynamics. The τth conditional quantile ($\tau \in (0,1)$) of this persistent component, given η_{it-1}, is $Q_t(\eta_{it-1}, \tau)$. υ_{it} is then defined as a random process such that:

$$\eta_{it} = Q_t(\eta_{i,t-1}\upsilon_{it}), \text{ where } (\upsilon_{it}|\eta_{i,t-1}, \eta_{i,t-2}\ldots) \sim \text{Uniform(0,1)}$$

. The quantile function maps draws of υ_{it} from a uniform distribution into quantile draws for the persistent component. The transitory component ε_{it} is assumed to be mean-zero, independent over time, and independent of $\eta_{i,t-s}$ for all s, and is assumed to also include any measurement error. This method allows for general forms of heteroskedasticity, conditional skewness and kurtosis in η_{it}. A caveat to this specification is that it excludes the possibility for the transitory component to follow an MA(1) process, which I do find some evidence for when estimating the baseline models. The t subscript refers to age. The permanent and transitory components are assumed to be mean-independent of age t, but the conditional quantile functions and marginal distributions of the transitory component may all depend on t. Non-linear persistence (ρ_t) of the persistent component can then be defined as:

$$\rho_t(\eta_{i,t-1}, \tau) = \frac{\partial Q_t(\eta_{it-1}, \tau)}{\partial \eta}, \quad \rho_t(\tau) = \mathbb{E}\frac{\partial Q_t(\eta_{it-1}, \tau)}{\partial \eta}$$

$\delta Q_t / \delta \eta$ is the partial derivative of Q_t with respect to its first argument, and the expectation is taken with respect to the distribution of η_{t-1}. This approach estimates persistence as the derivative effect of how much the persistent component of earnings in period t varies with the persistent component of earnings in period $t-1$ when hit with a shock in period t. I estimate $\rho_t(\eta_{i,t-1}\tau)$ of the health process, which is the persistence of $\eta_{i,t-1}$ when hit by shock υ_{it} with rank τ.

A major attraction of this method is that it allows for one shock (such as a very large or small realisation of υ_{it}) to wipe out the memory of past shocks. This incorporates an important additional source of heterogeneity in health shock persistence that is unavailable in the simple linear models. This allows, for example, a big negative shock in period t, such as a sudden permanent severe disability, to wipe out the persistence of past shocks. By contrast, the ARMA(p,q) and simple linear additive shock models cannot allow ρ to vary by any features of the shock that occurs in period t. Despite its computational complexity, the method is easy to use as Arellano, Blundell and Bonhomme (2017) make available full MATLAB replication files.[‖] Furthermore, De Nardi, Fella and Paz-Pardo (2019) propose a simulation-based method to discretize nonlinear and non-normal stochastic processes, so that these estimates

[‖]All replication files and supplementary material can be downloaded from: `https://onlinelibrary.wiley.com/doi/abs/10.3982/ECTA13795`

can be incorporated into a life-cycle model with minimal state-space cost.

To estimate the model, the quantile functions for ε_{it}, η_{i1} and η_{it} are first parameterised as low order Hermite polynomials. Since the persistent and transitory components of the process are not seperately observable, the estimation algorithm begins with an initial guess for the coefficients and then iterates sequentially between draws from the posterior distribution of the latent persistent component and quantile regression estimation until convergence is achieved. The algorithm used is closely related to the stochastic EM algorithm (Diebolt and Celeux, 1993), although the quantile specification of the model avoids the need for a likelihood-based approach to estimation.

I apply this method to estimating the persistence of health, and report the results in Figure 9 and Table 13 by deciles for the magnitude of the shock at period t and health decile in period *t-1*. Since the health index has been de-meaned by age, the health shocks are approximately symmetric, so the lowest decile consists of large negative health shocks, the median decile consists of very small health shocks or unchanged health, and the highest decile consists of very large positive shocks. I report both the persistence estimates for the overall health process, and just the persistent component η_{it}, which strips out the transitory component from the overall estimates. The persistent-component-only estimates are on average higher, with two notable exceptions; large positive shocks experienced by those in poor prior health, and large negative shocks experienced by those in prior good health. Transitory shocks are likely to be more important in these cases.

I find that persistence of health shocks varies greatly, depending on past health, shock size and sign. While the average of my estimates is approximately the estimate of persistence from my baseline models, my non-linear persistence estimates range from 0.6 to 1.2. Such variation has significant implications for economic decision making. Furthermore, there is a large difference in the persistence of positive health shocks and negative health shocks. Large negative health shocks are almost twice as persistent as large positive health shocks. Another notable result is that those in poorer health pre-shock take much longer to recover from a negative shock relative to those in better health pre-shock. Individuals who are both in poor health in period $t-1$ and then experience a large negative health shock in period t have an estimated persistence coefficient of 1 or more, suggesting that a negative shock is

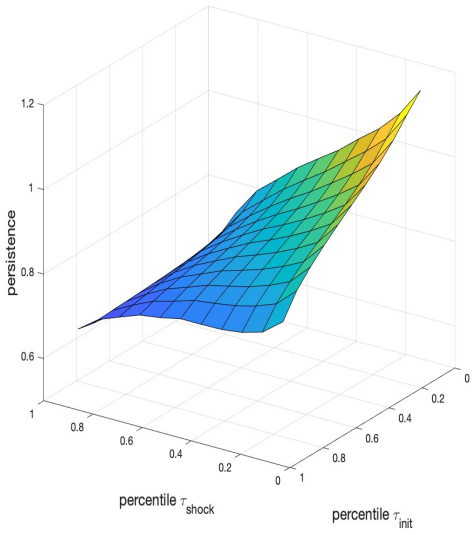
Table 13: Non-linear health persistence estimates

	Shock size percentiles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
	Overall health persistence										
1	1.16	1.10	1.05	1.01	0.97	0.94	0.90	0.86	0.82	0.76	0.67
2	1.13	1.06	1.01	0.98	0.94	0.91	0.87	0.83	0.79	0.73	0.65
3	1.08	1.02	0.98	0.94	0.91	0.88	0.85	0.81	0.78	0.72	0.64
4	1.05	0.99	0.95	0.92	0.89	0.86	0.83	0.80	0.77	0.71	0.64
5	1.02	0.96	0.93	0.90	0.87	0.85	0.82	0.79	0.76	0.71	0.64
6	0.99	0.93	0.90	0.88	0.86	0.83	0.81	0.78	0.75	0.71	0.64
7	0.96	0.91	0.88	0.86	0.84	0.82	0.80	0.78	0.74	0.71	0.65
8	0.93	0.88	0.86	0.84	0.82	0.81	0.79	0.77	0.74	0.70	0.65
9	0.90	0.85	0.83	0.82	0.80	0.79	0.78	0.76	0.73	0.70	0.65
10	0.87	0.82	0.80	0.79	0.78	0.78	0.76	0.75	0.73	0.70	0.66
11	0.81	0.77	0.76	0.75	0.75	0.75	0.74	0.74	0.72	0.70	0.66
	Persistent component of health shocks										
1	1.45	1.25	1.17	1.11	1.06	1.02	0.98	0.92	0.86	0.77	0.56
2	1.37	1.21	1.15	1.09	1.05	1.01	0.98	0.93	0.87	0.80	0.62
3	1.28	1.15	1.11	1.06	1.03	1.00	0.96	0.92	0.88	0.81	0.67
4	1.21	1.10	1.07	1.03	1.01	0.98	0.95	0.92	0.88	0.83	0.71
5	1.14	1.05	1.03	1.00	0.99	0.97	0.94	0.91	0.88	0.84	0.74
6	1.08	1.01	1.00	0.98	0.97	0.95	0.93	0.91	0.88	0.84	0.77
7	1.02	0.97	0.97	0.95	0.95	0.94	0.92	0.90	0.88	0.85	0.79
8	0.96	0.93	0.94	0.93	0.93	0.92	0.91	0.90	0.88	0.86	0.82
9	0.89	0.89	0.91	0.90	0.91	0.91	0.90	0.89	0.88	0.87	0.85
10	0.81	0.83	0.86	0.86	0.88	0.89	0.88	0.89	0.88	0.88	0.89
11	0.68	0.74	0.79	0.80	0.84	0.85	0.86	0.87	0.88	0.89	0.95

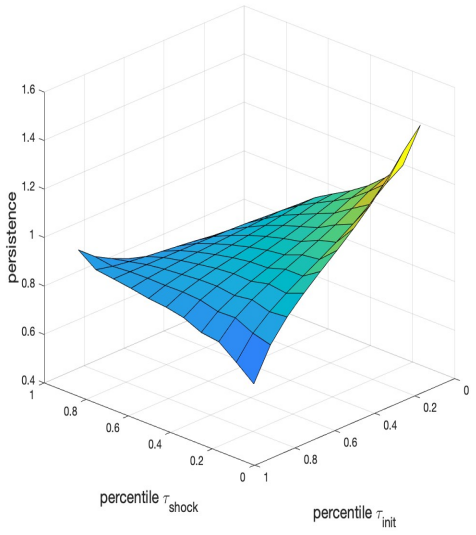
*1=most negative, 11=most positive

Figure 9: Non-linear persistence estimates

(a) Health ($\eta_{it} + \varepsilon_{it}$)



(b) Persistent component only (η_{it})



likely to be permanent for these individuals. By comparison, an ARMA(p,q) model will underestimate the persistence of a large negative health shock and overestimate the pace and magnitude of recovery, especially for those in poor past health.

6.2 Fixed effects

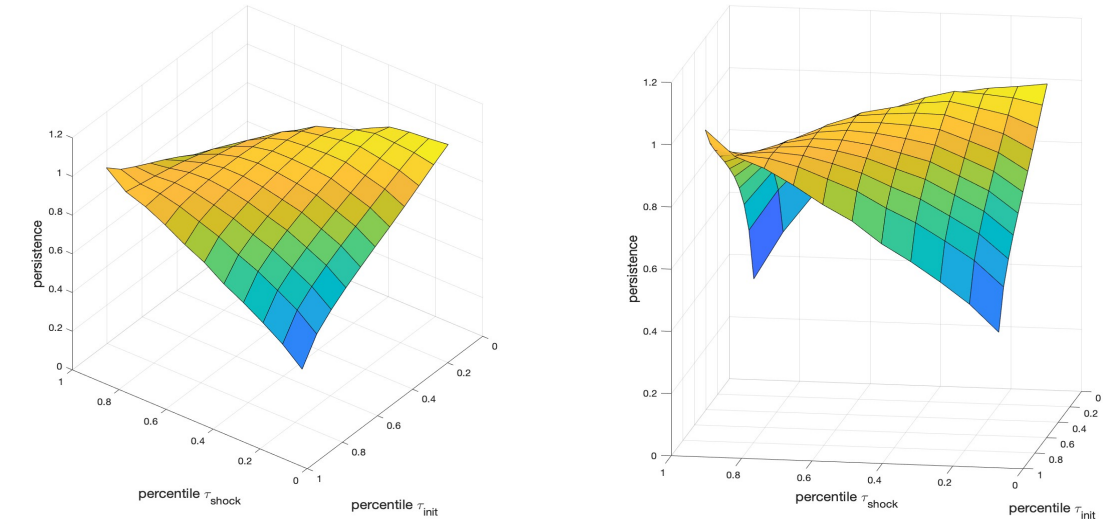
These non-linear persistence estimates demonstrate the crucial importance of allowing for heterogeneity in health shock features and health history when estimating persistence. Time-invariant, individual fixed effects are an additional important source of heterogeneity, and not accounting for them may bias the persistence estimates upwards. The literature also emphasises the importance of individual heterogeneity, such as initial conditions from childhood, education, or generic variation, as potentially more important than state dependence in determining health outcomes (Halliday, 2008). I re-estimate persistence allowing for fixed effects by using an extension to the Arellano, Blundell and Bonhomme (2017) framework included in their supplementary appendix. I find that accounting for fixed effects does reduce the persistence estimates, and the magnitude of the reduction varies by past health and shock magnitude. The reductions are largest for those in prior poor health who experience a large negative health shock. Therefore, the extremely high persistence previously observed for this group partially reflects fixed effects, although the new persistence

estimates remain high. Accounting for fixed effects also removes the asymmetry between positive and negative shock persistence.

To capture time-invariant fixed effects, the persistent component η_{it} is now defined as being equal to $Q_t(\eta_{i,t-1}, \zeta_i, \upsilon_{it})$ where ζ_i is the fixed effect. I report the new persistence estimates in Figure 10 and Table 14. The two graphs that make up Figure 10 illustrate the same data, but I rotate the plane around the persistence axis to better illustrate the range of the persistence estimates.

I report the estimates for the persistent component rather than overall health as estimates of this component are most likely to be overstated by not accounting for fixed effects.

Figure 10: Persistent component of health, accounting for fixed effects



Several of the key results from the original non-linear persistence estimates are unaffected by accounting for fixed effects. The range of the persistence estimates, depending on past health and characteristics of the shock at period t remain large, ranging from 0.3 to 1.0 depending on the features of the shock at period t and past health. Persistence estimates in cases of negative health shocks continue to be much higher for individuals in poor health, and the opposite is true for positive health shocks.

On average, the persistence estimates are smaller when fixed effects are taken into account. This is mostly driven by reductions to the persistence estimates in cases of negative shocks at period t. The largest reductions are observed for the largest decile

Table 14: Persistent component of health, accounting for fixed effects

	Shock size deciles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
1	1.01	0.99	0.99	0.97	0.92	0.86	0.81	0.72	0.62	0.50	0.34
2	0.96	0.98	0.99	0.99	0.97	0.93	0.90	0.84	0.75	0.66	0.52
3	0.91	0.94	0.96	0.98	0.97	0.95	0.93	0.89	0.82	0.74	0.63
4	0.86	0.90	0.93	0.96	0.96	0.95	0.94	0.91	0.86	0.79	0.70
5	0.81	0.86	0.90	0.93	0.94	0.95	0.95	0.92	0.88	0.83	0.76
6	0.76	0.82	0.86	0.90	0.92	0.93	0.94	0.93	0.90	0.86	0.80
7	0.71	0.78	0.83	0.87	0.89	0.92	0.93	0.93	0.91	0.88	0.84
8	0.66	0.74	0.79	0.83	0.86	0.89	0.91	0.93	0.92	0.90	0.88
9	0.61	0.69	0.74	0.79	0.83	0.87	0.89	0.92	0.92	0.92	0.92
10	0.54	0.62	0.68	0.74	0.78	0.83	0.87	0.90	0.92	0.93	0.97
11	0.41	0.50	0.57	0.63	0.68	0.75	0.80	0.86	0.91	0.95	1.03

*1=most negative, 11=most positive

of negative shocks, where persistence estimates fall by 0.3-0.4. Accounting for fixed effects has little impact on the persistence estimates when there are positive shocks. As a result, while the original non-linear estimates were much higher for negative shocks than positive shocks, this difference disappears when we take fixed effects into account. Accounting for fixed effects also reduces the persistence estimates for those in very poor health in $t-1$ who experience a positive shock in period t. These results suggest that those who experience large negative shocks, or have a history of poor health, are also more likely to have some unobserved time-invariant trait that subtracts from overall health, such as poor underlying health, and this partially explains the persistently very poor health we observe after large negative shocks and among those with poor health in the prior period. This result is not symmetrical for those who experience positive shocks.

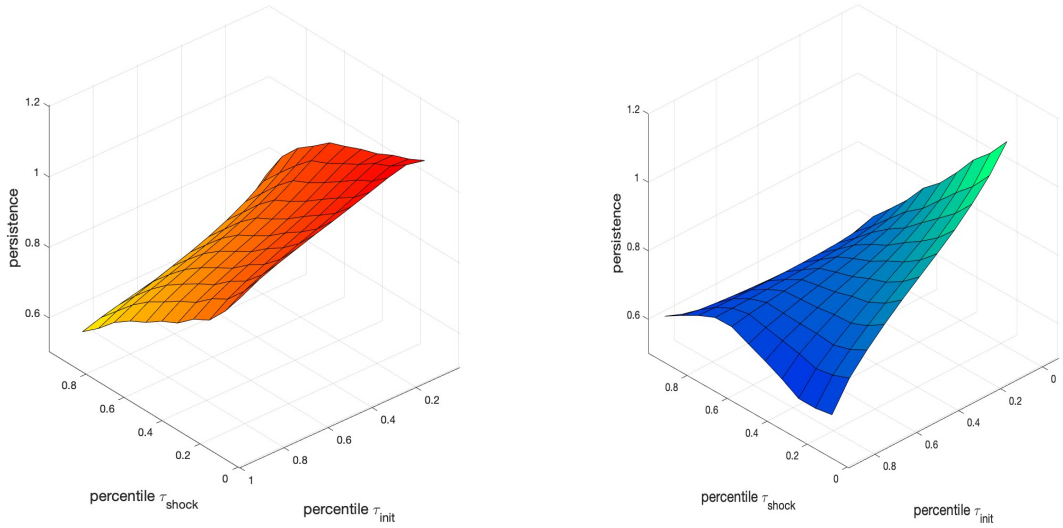
While these fixed effects cannot be observed directly, I do find some evidence that they are related to allostatic scores. Allostatic scores attempt to measure underlying health, which may be associated with vulnerability to suffer negative health shocks, and propensity and speed of recovery from them. I divide my sample into two groups based on whether allostatic scores are above or below the sample median allostatic score, and then re-estimate persistence for these two groups (Figure 11). High allostatic scores indicate poor underlying health while low allostatic scores indicate good

underlying health. These estimates are for the entire index, rather than just the persistent sub-component.

Figure 11: Non-linear overall persistence estimates; by allostatic score

(a) High allostatic scores (poor underlying health)

(b) Low allostatic scores (good underlying health)



I observe significant differences in the persistence estimates of those with better and worse allostatic scores. Those with worse underlying health experience more persistent negative health shocks and less persistent positive health shocks. The biggest difference between them is that the persistence estimates for those who suffered a large negative health shock but were in good prior health are about 0.5 units lower than for the group with worse allostatic scores. There are two possible reasons why these persistence estimates vary by allostatic score. Allostatic scores may be correlated with the persistence of shocks that people experience. For example, those with worse underlying health may be more vulnerable to highly persistent chronic health conditions. Alternatively, there may be a high correlation between allostatic scores and fixed effects. I find that the persistence differences between the higher and lower allostatic score groups can be significantly reduced if I use the estimation procedure that takes fixed effects into account. This suggests that these differences mostly reflect fixed effects. I show this in Table 15, which reports the difference between the non-linear persistence estimates that takes fixed effects into account for the groups with good and bad allostatic scores. I subtract the estimates for the group with bad

(above average) allostatic scores from the group with good (below average) allostatic scores; the difference now only ranges from -0.2 and 0.1.

Table 15: Difference between persistent component estimates of poor and good allostatic health groups, accounting for fixed effects

	Shock size deciles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
1	0.1	0.1	0.1	0.1	0.1	0.1	0.0	0.0	0.0	0.0	-0.1
2	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	-0.1
3	0.1	0.0	0.0	0.0	0.0	0.0	-0.1	-0.1	-0.1	-0.1	-0.1
4	0.1	0.0	-0.1	0.0	0.0	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1
5	0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1
6	0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1
7	0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.2	-0.2	-0.2	-0.1
8	0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.2	-0.2	-0.2	-0.2	-0.1
9	0.1	-0.1	-0.1	-0.1	-0.1	-0.1	-0.2	-0.2	-0.2	-0.2	-0.1
10	0.1	-0.1	-0.1	-0.1	-0.1	-0.2	-0.2	-0.2	-0.2	-0.2	-0.1
11	0.2	-0.1	-0.1	-0.1	-0.2	-0.2	-0.2	-0.2	-0.2	-0.2	-0.1

*1=most negative, 11=most positive

This result strongly suggests that allostatic scores capture some aspect of fixed effects that are helpful to include when modelling health dynamics. There is significant scope for further research on modelling these fixed effects and identifying whether they relate to, for example, education, early childhood experiences, or genetics.

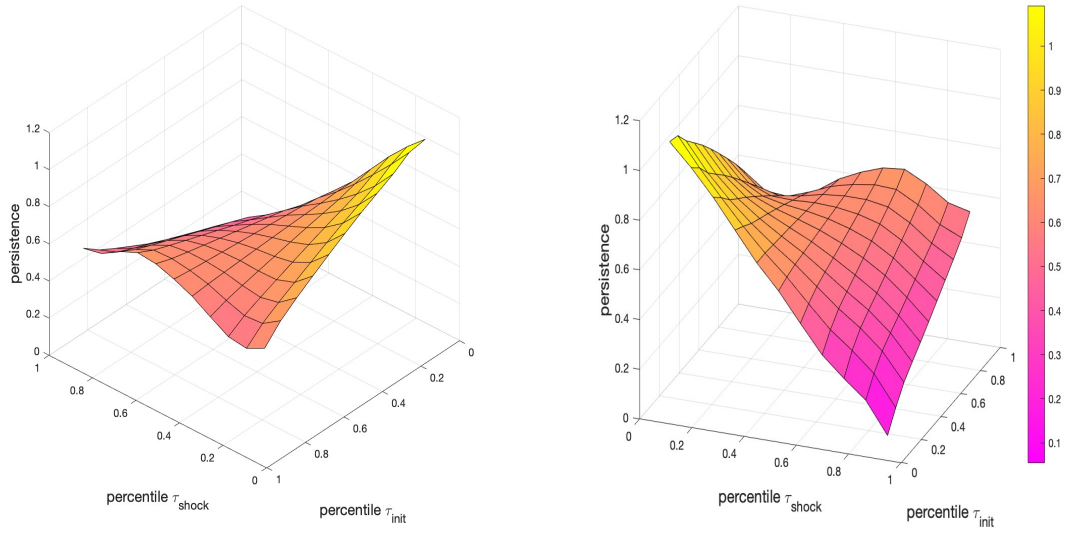
6.3 Mental health persistence

This chapter has focussed on modelling overall health. However, the methods used in this chapter can be easily applied to other health indices used in the economics literature, which may have very different persistence profiles. I calculate the persistence of GHQ scores, which are a sub-component of my health index and can be considered a measure of overall mental health. GHQ (general health questionnaire) is a questionnaire designed to identify non-psychotic and minor psychiatric disorders such as anxiety and depression, and provides a mental health score ranging from 1 to 36. I de-trend the raw GHQ scores from age, gender, and time trends, and first estimate an ARMA(p,q) model as a linear baseline. I find that GHQ scores can be represented as an ARMA(1,1) model in a similar manner to an overall health index,

although the level of persistence is lower, with the coefficient on the lagged health term estimated as 0.7. I report these ARMA estimation results in Appendix table 30.

I then follow the same procedure as above and calculate the non-linear persistence of the mental health index using the Arellano, Blundell and Bonhomme (2017) framework. I report a table of my persistence estimates by past health shock and past health decile in Appendix table 31 and illustrate the estimates in Figure 12. The two graphs show the same data, but I rotate the plane around the persistence axis to better illustrate the range in the persistence estimates. These estimates are for the complete mental health index, rather than just the persistent component.

Figure 12: Persistence of mental health index



In some ways, the mental health persistence graphs resemble the overall health persistence graphs. In both cases, the persistence of shocks varies significantly depending on past health history and magnitude and sign of the shock in period t, outcomes tend to be worse if the individual is in prior bad health, and negative shocks are more persistent than positive shocks. However, there are some significant differences, which may be important to capture when considering the impact of mental health shocks on economic decision making; the literature on this is very nascent (Jolivet and Postel-Vinay, 2020; Abramson, Boerma and Tsyvinski, 2024). For those with good prior mental health, persistence estimates are around 0.5-0.6, and do not vary much by shock sign or magnitude. However, for those with bad prior mental health,

persistence estimates have a huge range. Of note, the persistence of very large negative shocks is over 1, while the persistence of very large positive shocks is around 0.1 This suggests that for individuals already struggling with their mental health, large improvements are highly transient but any further declines are permanent, suggesting a 'downward spiral of despair' mechanism and that the capacity for recovery is limited.

7 Conclusion

This chapter investigated how best to model health as a dynamic process. It evaluated the strengths and weaknesses of the most commonly used approaches in the literature, and adapted recent techniques from the earnings dynamics literature to better capture some of the complexities around modelling shock persistence, frequency and magnitude. It also explored how biomarker data can be used to improve our ability to model health dynamics, although further research in this area is recommended as the increasing availability of genetic and other medical data offers researchers the opportunity to model health in increasingly sophisticated ways.

I conclude with several suggestions of ways to further develop this research. First, as the Arellano, Blundell and Bonhomme (2017) non-linear persistence framework becomes better known, other researchers are suggesting modifications and improvements, which could be applied to a health context. For example, Almuzara (2020) develops a 'heterogeneous transitory risk' (HTR) model that offers a sophisticated way of separately identifying the permanent and transitory components of a shock while also permitting dependence between them. This cannot be done using the Arellano, Blundell and Bonhomme (2017) approach. Health could be an interesting application of this model, as many individuals suffer from multiple health conditions, and capturing interactions between different conditions with different persistence profiles, such as a long-term chronic health condition and shorter-term mental health shock, could improve our health dynamics modelling.

Second, the focus of this paper has been to improve our ability to statistically predict health dynamics, because doing so helps us understand how health impacts economic decision making. However, I do not consider to what degree my predictions map onto how individuals understand and predict their own health trajectory. There are different ways to characterise this relationship: as a process of learning as a series of positive and negative health shocks helps people gradually learn their 'health type', or whether individuals have a stable, long-term bias to be overly perssimistic, optimistic, or broadly correct about their future health outcomes which has little relationship to their actual health histories. An interesting avenue for future research is to better understand to what degree people modify their expectations of the frequency and persistence of future health shocks following a period of poor health. Under-

standing this relationship is important as the persistence and distribution of health shocks affects both ex-ante choices (how people prepare in advance for a potential health shock) and ex-post choices (how people respond to a realised health shock). For example, a bad health shock may directly impact someones savings behaviour as they have to stop working for a while and that reduces their income, but it might also affect their savings behaviour by modifying their priors about their future health, which will continue to impact their savings behaviour even when fully recovered. A related improvement could be to separately consider the impact of negative and positive health shocks. Much of the literature focusses on modelling negative health shocks, with little attention paid to recoveries, perhaps because overall health indices heavily feature chronic health conditions and disabilities from which full recovery is unlikely.

A final suggestion is that there is significant scope to improve our understanding of the dynamics of sub-components of overall health, such as mental health. I showed that in some important ways, the statistical properties of mental health dynamics differs from the dynamics of overall health. There are lots of potential avenues for future work on this topic. For example, researchers could build up a richer picture of how mental health dynamics vary by observable characteristics such as age, gender or education level, or the interaction between mental health dynamics and economic events such as unemployment ((De Vera, Garcia-Brazales and Lin, 2023) is a current working paper on a closely-related topic). My method of modelling mental health is based on a score from a short questionnaire from the psychology literature, which is common practise in the health economics literature but could be improved, such as by making adjustments for the fact that it is measured as a non-negative integer that is bounded from above and below (Mullahy, 2024), or by incorporating additional data sources, such as high-frequency health information from wearable health technology.

A Appendix

A.1 Attrition

I estimate a linear probability model of attrition to identify the magnitude of any relationship between health and attrition risk. Those in poorest health quantile are around two percentage points more likely to drop out of the sample next period.

Table 16: Linear probability model of attrition

	Missing next period
health index quintile 1 (lowest)	0.0213***
	(8.14)
health index quintile 2	-0.00200
	(-0.80)
health index quintile 3 (baseline)	0
health index quintile 4	-0.00131
	(-0.52)
health index quintile 5 (highest)	0.00166
	(0.62)
age	-0.0205***
	(-5.88)
age squared	0.0577***
	(5.08)
age cubed	-0.0861***
	(-5.61)
age quartic	0.0498***
	(6.75)
sex	-0.00706***
	(-4.34)
Observations	228,886

t statistics in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

In general, the literature is fairly sanguine about the risks of using health indices for economic research when there is differential attrition risk by health. Jones, Koolman and Rice (2006) find that response rates to the British Household Panel Survey (BHPS) vary by health, with elderly or low-income individuals who start the survey

in poor health particularly likely to attrit, buts find that attempting to account for this using inverse probability weights is unnecessary for most research applications. Similarly, Pudney and Watson (2013) investigate the impact of reducing the effort made to chase up non-responders to BHPS and HILDA (an Australian panel dataset) surveys. While they find that the effort exerted to chase up non-respondents changed the sample prevalence of disability and ill health, their subsequent statistical modelling of the relationship between health and unemployment is unaffected. I conclude that the observed level of attrition in my dataset does not pose a significant threat to the robustness of my subsequent analysis.

A.2 Regression output from health index construction

I estimate the health index separately for each data wave using an ordered probit. Below I report the output for the the second last wave of data (wave 11) as an example to show how different objective variables contribute to the final index. Most of the objective variables are dummy variables.

Table 17: Estimation of health index - wave 11

variables	coefficient estimate	t-stat
mobility - some difficulty	-0.701***	(-18.97)
mobility - significant difficulty	-1.069***	(-13.40)
lifting, carrying, moving objects - some difficulty	-0.405***	(-10.77)
lifting, carrying, moving objects - significant difficulty	-0.489***	(-7.65)
manual dexterity - some difficulty	-0.173***	(-3.05)
manual dexterity - significant difficulty	-0.401***	(-3.90)
continence - some difficulty	-0.241***	(-4.81)
continence - significant difficulty	-0.339***	(-3.33)
hearing - some difficulty	-0.0565	(-0.76)
hearing - significant difficulty	-0.0403	(-0.34)
sight - some difficulty	-0.101	(-1.44)
sight - significant difficulty	-0.134	(-1.18)
communication, speech problems - some difficulty	-0.140	(-1.23)
communication, speech problems - significant difficulty	-0.455*	(-2.20)
memory, ability to concentrate, learn, understand - some difficulty	-0.333***	(-6.02)
memory, ability to concentrate, learn, understand - sig. difficulty	-0.398***	(-4.19)
recognise danger - some difficulty	0.116	(0.63)
recognise danger - significant difficulty	0.543*	(2.25)
physical coordination - some difficulty	-0.0759	(-1.29)
physical coordination - significant difficulty	0.111	(0.98)
personal care - some difficulty	-0.144	(-1.91)
personal care - significant difficulty	-0.321**	(-2.87)

other - some difficulty	-0.581***	(-14.95)
other - significant difficulty	-0.745***	(-10.50)
age 20-24	-0.244***	(-4.32)
age 25-29	-0.415***	(-7.33)
age 30-34	-0.452***	(-7.73)
age 35-39	-0.531***	(-9.61)
age 40-44	-0.649***	(-12.00)
age 45-49	-0.653***	(-12.49)
age 50-54	-0.732***	(-14.15)
age 55-59	-0.692***	(-13.66)
age 60-64	-0.764***	(-14.60)
age 65-69	-0.625***	(-11.88)
age 70 and older	-0.616***	(-12.39)
female	0.117***	(6.43)
asthma - ever had	0.0609	(0.29)
arthritis - ever had	0.0407	(0.26)
congestive heart failure - ever had	-0.285	(-0.58)
coronary heart disease - ever had	0.855**	(2.76)
angina - ever had	-0.175	(-0.45)
heart attack - ever had	-0.358	(-1.93)
angina - ever had	-0.0386	(-0.27)
emphysema - ever had	-0.480	(-1.15)
hypothyroidism - ever had	-0.0582	(-0.14)
chronic bronchitis - ever had	-0.532	(-1.15)
chronic liver condition - ever had	0.405	(1.33)
cancer - ever had	0.324*	(2.17)
diabetes - ever had	-0.277	(-1.25)
epilepsy - ever had	0.0659	(0.16)
high blood pressure - ever had	-0.0356	(-0.32)
other chronic condition - ever had	-0.209***	(-3.78)
multiple sclerosis - ever had	0.148	(0.51)
COPD - ever had	0.158	(0.43)
emotional, nervous, psychiatric problem - ever had	-0.252	(-1.08)
other cancer - ever had	-0.590**	(-2.90)
anxiety - ever had	-0.0187	(-0.07)
depression - ever had	-0.267	(-1.41)
asthma - still have	-0.194	(-0.89)
arthritis - still have	-0.128	(-0.78)
congestive heart failure - still have	-0.327	(-0.60)
coronary heart disease - still have	-1.019**	(-3.00)
angina - still have	-0.310	(-0.73)
hypothyroidism or underactive thyroid - still have	-0.0505	(-0.12)
chronic bronchitis - still have	0.144	(0.27)
liver condition - still have	-0.606	(-1.75)
cancer - still have	-0.884***	(-4.44)
diabetes - still have	-0.115	(-0.50)
epilepsy - still have	-0.105	(-0.20)
high blood pressure - still have	-0.134	(-1.14)
COPD - still have	-0.488	(-1.28)
emotional or nervous or psychiatric condition - still have	0.521	(1.91)

anxiety - still have	-0.0641	(-0.24)
depression - still have	0.000729	(0.00)
1-2 visits to hospital outpatient in yr	-0.132***	(-6.50)
3-5 visits to hospital outpatient in yr	-0.384***	(-12.12)
6-10 visits to hospital outpatient in yr	-0.476***	(-9.93)
>10 visits to hospital outpatient in yr	-0.652***	(-9.31)
No job dummy	-0.269***	(-9.84)
professional occupation	0.0977*	(2.02)
skilled non-manual occupation	-0.154***	(-5.00)
skilled manual occupation	-0.1000**	(-2.95)
partly skilled occupation	-0.188***	(-5.45)
unskilled occupation	-0.181**	(-2.58)
GHQ score - 1	0.192	(0.66)
GHQ score - 2	-0.309	(-1.30)
GHQ score - 3	-0.261	(-1.19)
GHQ score - 4	-0.424*	(-2.12)
GHQ score - 5	-0.476**	(-2.60)
GHQ score - 6	-0.531**	(-3.04)
GHQ score - 7	-0.639***	(-3.65)
GHQ score - 8	-0.720***	(-4.11)
GHQ score - 9	-0.851***	(-4.85)
GHQ score - 10	-0.962***	(-5.49)
GHQ score - 11	-1.051***	(-6.01)
GHQ score - 12	-1.163***	(-6.66)
GHQ score - 13	-1.226***	(-6.93)
GHQ score - 14	-1.221***	(-6.82)
GHQ score - 15	-1.268***	(-7.04)
GHQ score - 16	-1.354***	(-7.48)
GHQ score - 17	-1.292***	(-7.10)
GHQ score - 18	-1.433***	(-7.76)
GHQ score - 19	-1.389***	(-7.48)
GHQ score - 20	-1.402***	(-7.47)
GHQ score - 21	-1.447***	(-7.61)
GHQ score - 22	-1.634***	(-8.59)
GHQ score - 23	-1.698***	(-8.95)
GHQ score - 24	-1.497***	(-7.89)
GHQ score - 25	-1.788***	(-8.56)
GHQ score - 26	-1.864***	(-8.35)
GHQ score - 27	-1.823***	(-6.19)
GHQ score - 28	-2.013***	(-7.98)
GHQ score - 29	-1.703***	(-6.75)
GHQ score - 30	-2.027***	(-7.47)
GHQ score - 31	-1.980***	(-7.51)
GHQ score - 32	-1.922***	(-6.90)
GHQ score - 33	-2.272***	(-6.62)
GHQ score - 34	-2.033***	(-8.03)
GHQ score - 35	-1.946***	(-4.30)
GHQ score - 36	-1.527***	(-3.66)
Education - GSCEs	0.0511*	(1.96)

Education - Yr12	0.125***	(3.66)
Education - Degree	0.275***	(10.81)
pregnant	0.174	(1.70)
N	24,987	

t stats in parentheses, exclude reporting time dummies coefficients, *p<0.05, **p<0.01, *** p<0.001

A.3 Genetic data

Polygenic scores (PGS) are scores constructed using genetic data that estimate an individual's propensity to express a phenotype, which is an observable trait. They are calculated from genome-wide association studies (GWAS), which are systematic analyses of genetic variation across the entire human genome and their association with various phenotypes. I make use of the latest (2022) version of the ELSA polygenic scores (Ajnakin and Andrew Steptoe, 2022) and select some of them to capture health conditions with the biggest disease burdens, which I report in Table 18. I use these polygenic scores to create two health aggregates; one capturing chronic physical health conditions, the other mental health. The correlation between the two indices is small.

Table 18: Polygenic Score Aggregation

Physical index	Mental index
Coronary artery disease (2016)	Alzheimer's disease (2019)
Type II diabetes (2018)	Depressive Symptoms
Rheumatoid arthritis	Major depressive disorder (2018)
Myocardial infarction	Anxiety (case-control)
Migrane (2016)	Schizophrenia (2020)
Chronic pain	Bipolar disorders (2021)
Waist-hip-ratio	Subjective wellbeing
	Loneliness

My method of aggregation is identical to how I aggregate the biomarker data. I normalise each PGS and then aggregate them. I consider alternate aggregation methods, including factor analysis and converting each PGS into a binary variable with the highest 10-20 per cent of scores coded as '1', however the resulting indexes are all quite similar. To assess the predictive value of my PGS indices, I regress a health index constructed using ELSA data, that is designed to be as similar as possible to my main health index constructed using Understanding Society data, against normalised

polygenic scores for all the PGS that make up my indices for mental and physical health. I report the results of this exercise in Table 19. The major depressive disorder PGS has the highest predictive power although the predictive power of many of the eight mental health PGS are quite similar. The predictive power of the physical PGS are much more varied, with chronic pain being by far the most important.

Table 19: How well individual polygenic scores predict health index

	components of	
z-score of PGS	mental index	physical index
z score: depressive symptoms	-0.0302***	
	(0.00486)	
z score: major depressive disorder	-0.0521***	
	(0.00515)	
z score: anxiety	0.00763	
	(0.00490)	
z score: schizophrenia	0.0192***	
	(0.00575)	
z score: bipolar	-0.00397	
	(0.00468)	
z score: subjective well-being	0.0119**	
	(0.00433)	
z score: Alzheimer's	-0.0170***	
	(0.00394)	
z score: loneliness	-0.0340***	
	(0.00490)	
z score: arthritis		0.00911*
		(0.00426)
z score: coronary heart disease		0.00193
		(0.00401)
z score: diabetes		-0.0342***
		(0.00409)
z score: chronic pain		-0.112***
		(0.00416)
z score: myocardial infarction		-0.0192***
		(0.00408)
z score: waist-hip ratio		-0.0121**
		(0.00394)
z score: migraines		-0.00235
		(0.00378)
N	37,543	37,546

I then repeat this exercise, but with the PGS scores aggregated into two indices that capture mental and physcial health, and report the results in Table 20. I find that while the both indices based on genetic data are significantly correlated with the level of health index and its variance over time, and contains additional information not captured by lagged health index terms or biomarkers, the size of the coefficients are

very small. I conclude that the coefficient sizes are too small to be a useful addition to modelling the overall dynamics of the health index. However, a more granular approach may be more effective. This could include using only individual PGS with higher predictive power or for diseases with high incidence rates such as diabetes and depression rather than rarer conditions such as schizophrenia.

Table 20: How well aggregated polygenic scores predict health index

	level of health index			variance of health index		
	(1)	(2)	(3)	(4)	(5)	(6)
mental index	-0.00746***	-0.00125*	-0.00768***	0.000810***	0.000392	0.000654*
	(-8.42)	(-2.10)	(-6.31)	(4.48)	(1.93)	(2.52)
physical index	-0.0229***	-0.00476***	-0.0187***	0.00165***	0.000809**	0.00136***
	(-18.28)	(-5.42)	(-10.71)	(6.24)	(2.63)	(3.56)
mental index ^{2}	-0.000496***	-0.000187*	-0.000463**	0.0000500*	0.0000226	0.0000302
	(-4.73)	(-2.45)	(-3.19)	(2.37)	(0.93)	(1.01)
physical index ^{2}	-0.000519*	0.0000618	-0.000417	0.0000270	0.0000366	0.00000785
	(-2.05)	(0.34)	(-1.18)	(0.49)	(0.57)	(0.10)
lagged health index		0.840***			-0.0485***	
		(201.75)			(-29.36)	
allostatic index			-0.385***			0.0330***
			(-18.59)			(10.38)
N	37,543	25,256	18,304	37,412	25,256	18,203

t statistics in parentheses; also control for age and gender, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

A.4 Replication exercise using non-detrended health data

I replicate the preferred baseline ARMA(1,1) model estimated using health data that has not been detrended by age and sex and a Difference-GMM specification. This exercise shows that the persistence estimates are robust to being detrending by age and sex; the slightly higher persistence estimates in this case are due to a gradual decline in health as people age being incorporated into the persistence estimates.

Table 21: Estimation of ARMA(1,1) model with non-detrended health index

	health index
L.health_index	1.038***
	(0.0227)
Hansen J test stat	2.756
Hansen J p value	0.431
N	222,095

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

A.5 ARMA(p,q) groups from clustering analysis

The partition clustering algorithm makes an initial partition of individuals into clusters based on the number of desired clusters, and then reallocates individuals until the final partition minimises the residual sum of the squared objective function. The optimal number of clusters for a given model is selected as the one with the lowest model information criterion (MIC). I describe the main characteristics of the two clusters I obtain after performing clustering analysis in Table 22. Relative to group 2, group 1 contains individuals with worse health on average. The individuals in the two groups are just as likely to experience a large negative health shock of at least one standard deviation (6.6 and 6.7 per cent of observations respectively), but the second group is more likely to experience a positive shock (5.3 versus 6.4 per cent).

Table 22: Group characteristics*

	Group 1	Group 2
mean health index	0.024	0.134
median health index	0.186	0.254
st. dev of health indices	0.670	0.565
mean allostatic score	0.023	-0.046
N	28,644	44,231

* Only include individuals where T=12

I then replicate my difference and system GMM specifications I used for estimating the ARMA(p,q) specification for my main health index for each group separately.

Table 23: GMM estimates of the health process, by cluster

	Difference GMM								System GMM	
	group 1				group 2				group 1	group 2
	MA(0)		MA(1)		MA(0)		MA(1)		MA(0)	MA(1)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
L.health	0.709***	0.791***	0.898***	0.922***	0.104***	0.187***	0.997***	0.893***	0.742***	0.828***
	(0.0203)	(0.0218)	(0.0311)	(0.100)	(0.0119)	(0.0172)	(0.132)	(0.164)	(0.00819)	(0.0461)
L2.health		0.0876***		-0.0207		0.0737***		-0.00265	0.0807***	
		(0.0124)		(0.0822)		(0.0127)		(0.0236)	(0.0106)	
AB test, order 1 z score	-26.87	-27.77	-21.04	-6.27	-35.4	-31.92	-9.46	-6.99	-33.00	-19.15
AB test, order 1 p value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
AB test, order 2 z score	3.44	-1.21	3.59	1.27	3.36	-1.46	6.02	4.2	-1.11	9.39
AB test, order 2 p value	0.001	0.226	0.000	0.203	0.001	0.145	0.000	0.000	0.266	0.000
Hansen J test statistic	63.74	3.39	1.47	1.37	78.77	43.01	1.71	7.19	12.74	4.09
Hansen J p value	0.000	0.495	0.689	0.712	0.000	0.000	0.634	0.066	0.047	0.394
Observations	28,644	26,040	28,644	26,040	44,231	40,210	44,231	40,210	26,040	44,231

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

This exercise shows that Group 1 is best modelled as an AR(2) process and group 2 as an ARMA(1,1) process. Table 24 reports the results of estimating the MA(1) coefficient for group 2, which I estimate to be -0.54.

Table 24: GMM Estimation of MA(1) coefficient - group 2

	$\rho = 0.83$
η_i	0.0851***
	(0.00280)
θ	-0.544***
	(0.0249)
ε_{it}	0.289***
	(0.00411)
N	44,231

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

A.6 ARCH model estimates

An autoregressive conditional heteroskedasticity (ARCH) model allows us to accounts for whether individuals who have recently experienced a health shock, captured in the model as a large ε_{it} term, are more or less likely to experience additional health shocks in the subsequent period. I estimate an ARCH(1) model with the following exponential variance specification: $\sigma_{it}^2 = \exp(\gamma_0 + \gamma_1 \varepsilon_{i,t-1}^2 + \gamma_2 \varepsilon_{i,t-1})$. The γ_2 term allows for asymmetry between negative and positive shocks. To estimate ARCH effects using GMM I use the following moment condition derived by Arellano (1995). For robustness I also estimate the more general specification by Meghir and Windmeijer (1999) and obtain similar results.

$$\mathbb{E}\left[h_{i,t-k}\left(\varepsilon_{i,t-1}^2 - \frac{\varepsilon_{i,t}^2(1+\sigma_{it-1}^2)}{(1+\sigma_{i,t}^2)}\right)\right] = 0, \text{ k} = 1...t-3$$

An ARCH effect exists if the γ_1 and γ_2 terms on the lagged error terms in the variance specification (σ_{it}^2) are significantly different from zero. I report my results in Table 25. I find that while the point estimates for the γ terms are reasonable, and suggest that a person who experiences a large health shock has more volatile health next period, especially if they suffer a negative health shock, the estimates are not

statistically significant. I conclude that there is no evidence for ARCH effects in the data.

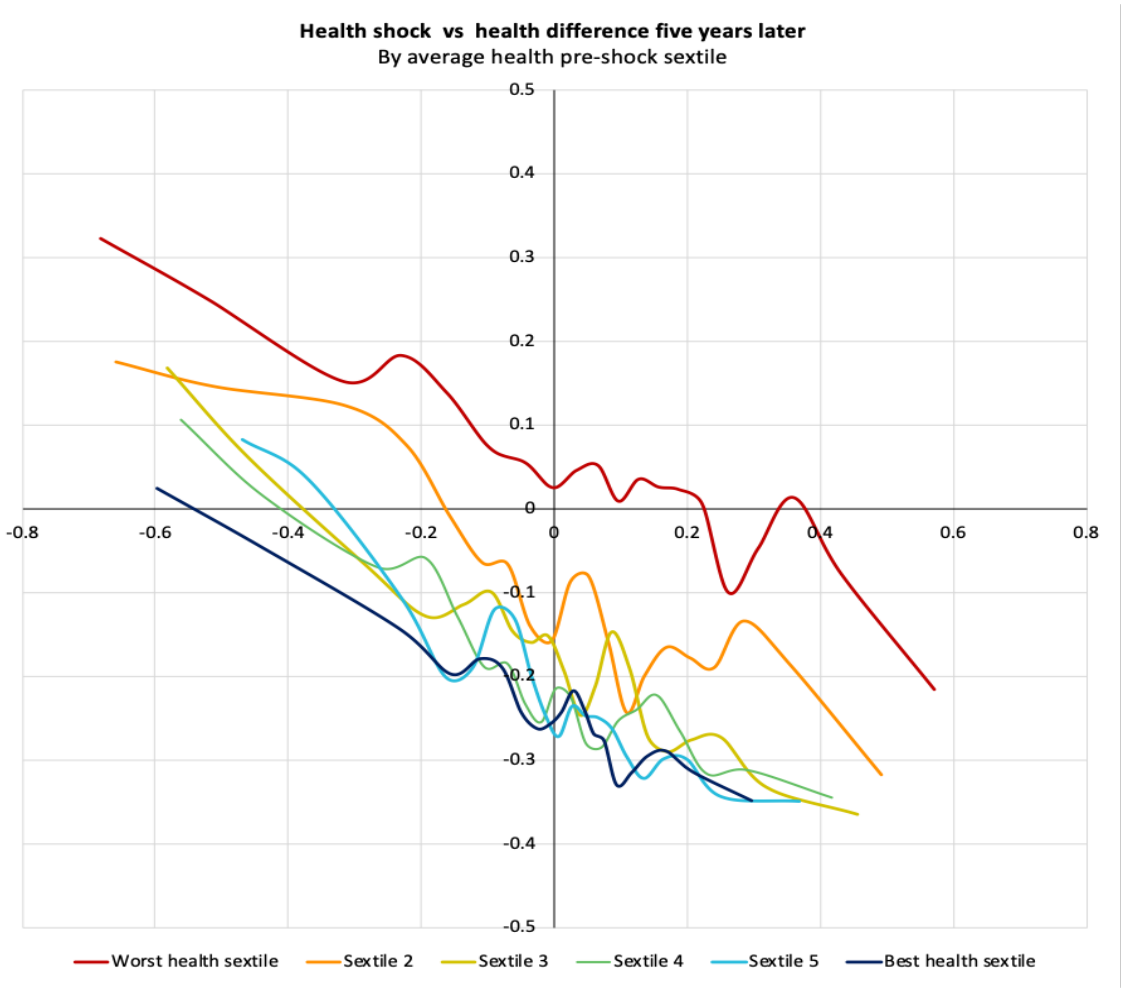
Table 25: GMM estimates of ARCH(1) effect

variables	estimates
γ_0	-2.5176
	(5.7445)
γ_1	0.3301
	(0.5848)
γ_2	-0.1648
	(0.8703)

standard errors in parentheses, $* = p < 0.05$

I also consider shock heterogeneity in a different way. Figure 13 graphs the relationship between the magnitude of a health shock in period t and the extent of recovery/mean-reversion five years later. I plot the initial health shock $(h_{i,t} - h_{i,t-1})$ on the x axis and the difference between health at period t and five years later $(h_{i,t+5} - h_{i,t})$ on the y axis. A steeper downward-sloping line suggests a faster rate of mean-reversion, while a flat line would indicate that there has been no change in health between period t and period $t + 5$. I graph this relationship for each sextile based on average health in the period prior to the shock $(t + 1)$. The design of this graph was adapted from an earnings dynamics graph by Guvenen et al. (2021). Figure 13 shows that there is significantly less recovery from negative shocks than would be predicted by an ARMA(1,1) model with normally distributed white-noise shocks, which would achieve around a 60 per cent mean revision, especially among those in persistently poor health prior to the negative shock. The health dynamics of those whose health is in the bottom sixth of the sample are much less well captured by an ARMA(1,1) model relative to those in the upper two-thirds. This exercise suggests that simple ARMA models are adequate for approximating the health process for the healthier section of the population, but are much less accurate for those with a history of poor health.

Figure 13



A.7 ARMA models with additional allostatic regressors

Adding allostatic scores as an additional regressor, or as a dummy variable for whether individuals have 'bad' allostatic scores interacted with the lagged health term, has very little impact on my linear estimates of health. Table 26 reports the regression output if I include these additional terms in my preferred specification.

Table 26: Health index estimates with additional allostatic score regressors

	(1)	(2)
L.health index	0.890***	0.876***
	(0.0286)	(0.0218)
allostatic scores	0.203	-0.348
	(0.302)	(0.178)
L.health index × allostatic scores		-0.131
		(0.0711)
Observations	70,039	70,039

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

A.8 Additional non-linear health persistence estimates results

I report the full results of re-estimating the non-linear persistence model when the sample is split into two sub-groups based on allostatic score in Tables 27 and 28. Table 29 reports output from the same estimation process, where I separately estimate the health persistence for individuals with higher and lower allostatic scores. However, this time I only report the persistence results for the persistent component ($\rho_t(\tau)$) rather than the entire index. I do not control for fixed effects in either set of results.

Table 27: Non-linear persistence estimates: low allostatic scores sub-sample (healthy)

	Shock size percentiles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
1	1.15	1.09	1.04	0.98	0.92	0.88	0.82	0.76	0.71	0.64	0.55
2	1.08	1.02	0.98	0.93	0.87	0.84	0.79	0.74	0.70	0.63	0.54
3	1.02	0.97	0.93	0.89	0.84	0.81	0.78	0.73	0.69	0.63	0.55
4	0.98	0.92	0.89	0.86	0.82	0.79	0.77	0.72	0.69	0.63	0.55
5	0.93	0.89	0.85	0.83	0.80	0.78	0.76	0.72	0.69	0.63	0.56
6	0.89	0.85	0.82	0.80	0.78	0.77	0.75	0.72	0.68	0.63	0.57
7	0.85	0.81	0.79	0.78	0.76	0.75	0.74	0.72	0.68	0.64	0.58
8	0.81	0.77	0.75	0.75	0.74	0.74	0.74	0.71	0.68	0.64	0.59
9	0.76	0.73	0.72	0.72	0.72	0.73	0.73	0.71	0.69	0.65	0.60
10	0.70	0.68	0.67	0.69	0.70	0.71	0.72	0.71	0.69	0.65	0.61
11	0.62	0.61	0.61	0.64	0.67	0.69	0.71	0.71	0.69	0.66	0.63

*1=most negative, 11=most positive

Table 28: Non-linear persistence estimates: high allostatic score sub-sample (unhealthy)

	Shock size percentiles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
1	1.09	1.06	1.05	1.02	1.01	0.99	0.97	0.93	0.90	0.85	0.77
2	1.10	1.07	1.05	1.02	0.99	0.96	0.94	0.90	0.85	0.80	0.72
3	1.08	1.05	1.02	0.99	0.96	0.93	0.91	0.87	0.82	0.77	0.69
4	1.07	1.02	1.00	0.96	0.93	0.90	0.88	0.84	0.79	0.74	0.66
5	1.05	1.00	0.97	0.94	0.90	0.88	0.85	0.81	0.77	0.72	0.64
6	1.03	0.98	0.95	0.92	0.88	0.85	0.83	0.79	0.74	0.70	0.63
7	1.02	0.96	0.93	0.90	0.86	0.83	0.81	0.77	0.73	0.68	0.62
8	1.00	0.94	0.90	0.87	0.84	0.81	0.78	0.75	0.71	0.66	0.60
9	0.98	0.92	0.88	0.85	0.81	0.79	0.76	0.73	0.69	0.64	0.59
10	0.96	0.90	0.85	0.82	0.79	0.76	0.73	0.71	0.67	0.62	0.58
11	0.93	0.87	0.81	0.79	0.75	0.73	0.70	0.67	0.64	0.60	0.56

*1=most negative, 11=most positive

Table 29: Estimates of coefficient of persistent component $\rho_t(\tau)$

	Shock size percentiles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
Sub-sample with below-average (good) allostatic scores											
1	1.48	1.35	1.25	1.15	1.05	0.97	0.91	0.86	0.80	0.68	0.39
2	1.35	1.21	1.13	1.04	0.96	0.90	0.85	0.81	0.76	0.66	0.46
3	1.24	1.11	1.02	0.96	0.89	0.85	0.80	0.76	0.72	0.64	0.50
4	1.15	1.02	0.95	0.89	0.84	0.80	0.76	0.73	0.69	0.63	0.53
5	1.08	0.95	0.88	0.84	0.80	0.77	0.73	0.70	0.67	0.61	0.55
6	1.02	0.90	0.83	0.79	0.76	0.74	0.71	0.68	0.65	0.60	0.56
7	0.96	0.85	0.79	0.76	0.73	0.71	0.68	0.66	0.63	0.59	0.58
8	0.91	0.80	0.74	0.72	0.70	0.69	0.66	0.64	0.61	0.58	0.59
9	0.86	0.75	0.70	0.68	0.67	0.66	0.64	0.62	0.60	0.57	0.60
10	0.80	0.69	0.65	0.64	0.64	0.63	0.61	0.59	0.58	0.56	0.61
11	0.70	0.61	0.57	0.57	0.58	0.59	0.57	0.56	0.55	0.54	0.63
Subsample with above-average (bad) allostatic scores											
1	1.35	1.25	1.16	1.03	0.96	0.92	0.89	0.83	0.78	0.68	0.52
2	1.26	1.19	1.11	1.00	0.94	0.91	0.89	0.83	0.79	0.71	0.58
3	1.17	1.13	1.05	0.96	0.91	0.89	0.87	0.83	0.79	0.73	0.61
4	1.08	1.07	1.00	0.93	0.89	0.88	0.86	0.82	0.79	0.73	0.64
5	1.01	1.01	0.96	0.90	0.87	0.86	0.85	0.82	0.79	0.74	0.66
6	0.94	0.96	0.92	0.87	0.85	0.85	0.84	0.81	0.79	0.74	0.67
7	0.88	0.92	0.88	0.85	0.84	0.84	0.83	0.81	0.79	0.75	0.69
8	0.82	0.87	0.84	0.82	0.82	0.83	0.82	0.81	0.79	0.75	0.70
9	0.75	0.82	0.80	0.80	0.80	0.81	0.81	0.80	0.79	0.75	0.72
10	0.67	0.76	0.75	0.76	0.78	0.80	0.80	0.79	0.78	0.75	0.73
11	0.53	0.66	0.67	0.71	0.74	0.77	0.77	0.78	0.78	0.76	0.76

*1=most negative, 11=most positive

A.9 Additional mental health persistence estimates results

Table 30 reports the coefficient estimates for the lagged GHQ term when estimating an ARMA(1,1) model for the mental health index using GMM.

Table 30: ARMA(1,1) model of GHQ scores

	Diff-GMM	Sys-GMM
L.ghq	0.610***	0.693***
	(0.0508)	(0.0388)
AB test, order 1, z score	-17.27	-22.72
AB test, order 1, p value	0.000	0.000
AB test, order 2, z score	8.960	11.42
AB test, order 2, p value	0.000	0.000
AB test, order 3, z score	0.158	0.262
AB test, order 3, p value	0.874	0.793
Hansen J test stat	2.223	7.41
Hansen J test p value	0.527	0.115
Observations	222,095	222,095

Standard errors in parentheses, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Incorporating additional lagged terms found to be insignificant

I report some additional results from the non-linear persistence estimates of mental health. First, I report the persistence estimates in Table 31 that accompany the graphs I include in section 6 (Figure 12). Second, I illustrate the impact of taking fixed effects into account when calculating mental health persistence in Figure 14.

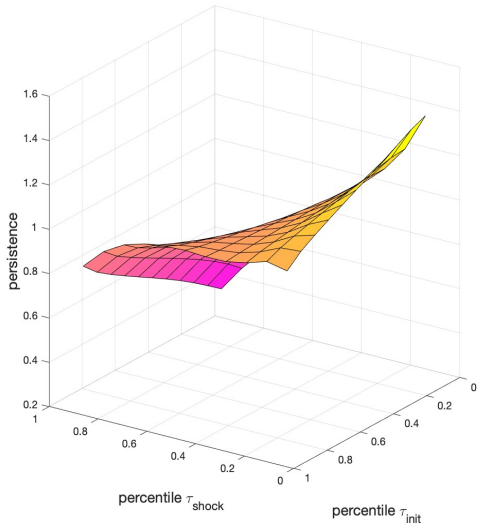
Table 31: Mental health persistence estimates

	Shock size percentiles*										
	1	2	3	4	5	6	7	8	9	10	11
$health_{t-1}$*											
1	1.09	1.00	0.90	0.78	0.66	0.56	0.45	0.34	0.26	0.18	0.06
2	1.08	0.96	0.86	0.76	0.66	0.58	0.49	0.40	0.33	0.25	0.13
3	1.02	0.90	0.81	0.73	0.66	0.59	0.52	0.44	0.38	0.30	0.19
4	0.97	0.86	0.78	0.71	0.65	0.60	0.54	0.48	0.41	0.34	0.24
5	0.92	0.82	0.74	0.69	0.65	0.61	0.56	0.50	0.45	0.37	0.28
6	0.87	0.77	0.71	0.67	0.65	0.62	0.58	0.53	0.48	0.40	0.32
7	0.81	0.72	0.67	0.65	0.64	0.62	0.60	0.56	0.51	0.44	0.36
8	0.75	0.67	0.63	0.63	0.63	0.63	0.62	0.59	0.54	0.47	0.41
9	0.69	0.62	0.60	0.61	0.63	0.64	0.64	0.62	0.57	0.51	0.46
10	0.63	0.56	0.55	0.59	0.62	0.65	0.66	0.65	0.61	0.54	0.50
11	0.54	0.48	0.50	0.55	0.61	0.66	0.69	0.70	0.65	0.59	0.57

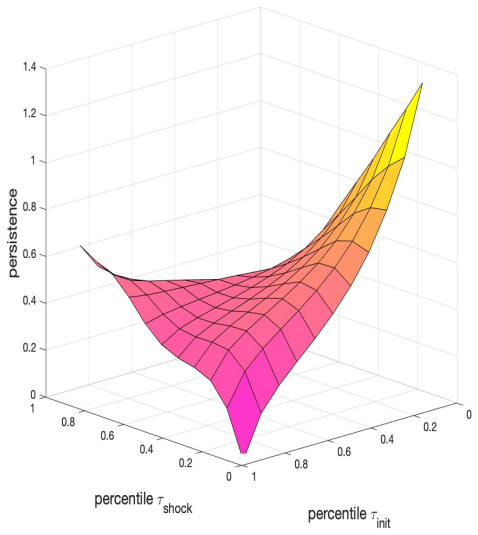
*1=most negative, 11=most positive

Figure 14: Persistence of GHQ index

(a) Persistent component only



(b) Persistent component w FE



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