The welfare effects of nonlinear health dynamics*

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Abstract

We generate a continuous measure of health to estimate a non-parametric model of health dynamics, showing that adverse health shocks are highly persistent when suffered by people in poor health. Canonical models cannot account for this pattern. We incorporate this health dynamic into a life-cycle model of consumption, savings, and labor force participation. After estimating the model parameters, we simulate the effects of health shocks on economic outcomes. We find that bad health shocks have long-term adverse economic effects that are more extreme for those in poor health. Furthermore, bad health shocks also increase the disparity of asset accumulation among this group of people. A canonical model of health dynamics would not reveal these effects.

Keywords: Health, Nonlinear dynamics, Life-cycle model, Inequality, Savings
JEL codes: I14, D15, J22 ,J26

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1 Introduction

What is the long-term impact of severe health shocks compared to milder ones, and how do they affect individuals' economic outcomes? Poor health is often linked to lower economic status, so it is important to examine whether severe health shocks have a more lasting effect and whether asymmetrical shocks worsen existing inequalities. To address these questions, we introduce a continuous health measure and estimate its dynamic using a flexible non-parametric panel data methodology. We incorporate this health dynamic into a life-cycle model of consumption, savings, and labor supply decisions to simulate the effects of various types of shocks on aggregate economic outcomes and measures of inequality.

We derive a continuous health measure by combining several objective health indicators using a latent variable model inspired by the approach of Bound (1991). To better replicate the dynamic we observe in the data, we allow health to evolve according to a flexible, nonlinear dynamic process characterized by persistent and transitory components. Drawing from Arellano, Blundell and Bonhomme (2017), we assume the persistent component evolves following a non-parametric process with nonlinear persistence and age dependence. This approach's flexibility captures persistence patterns that cannot be captured by a "canonical" first-order Markov process.

We incorporate this health dynamic into a standard life-cycle model where agents receive utility from consumption, leisure, and bequest of assets. They make decisions regarding labor market participation, hours worked, and savings every period while facing uncertainty over health, wages, and life expectancy. Health is a determinant of wages and life expectancy. Following the literature, health affects the time constraint, changing the tradeoff between consumption and leisure. The model also includes a fixed cost of work to fit the evidence on labor market participation and hours worked.

We estimate this model with a Simulated Method of Moments procedure using data from the English Longitudinal Study of the Ageing (ELSA), a panel of English households aged 50 and above. We perform the estimation in two steps. First, we estimate the parameters of the health, wage, and survival processes, which we assume are exogenous to the life-cycle model. Next, we estimate the model parameters (the coefficient of risk aversion, the preferences for bequests, the costs of bad health, and of working) using the processes for wages, health, and life expectancy estimated in the first step. To highlight the importance of using a nonlinear process, we also estimate

a model specification using a "canonical" health process whose persistent component evolves according to an AR(1) model.

The nonlinear process estimates reveal that health indeed follows a complex, nonlinear dynamic. Specifically, negative shocks are more severe when starting from a state of poor health and exhibit greater persistence than positive shocks. When individuals start in good health instead, good shocks exhibit only slightly higher persistence than negative shocks. These asymmetries cannot be replicated by the standard canonical process, lacking the flexibility needed to account for such complexities. Capturing these asymmetries is also challenging when health is represented as a binary variable or when it assumes a small number of discrete values. This difficulty arises from the reliance on arbitrary health cutoffs implicitly used to discretize the health measure, undermining a more nuanced understanding of its dynamics.

The moments we match to estimate the model parameters are the age profiles of accumulated assets, labor force participation across different health quantiles, and hours worked. Our simulated moments align closely with data moments, regardless of whether we employ the nonlinear or the canonical model of health dynamics. We simulate several individual histories to assess the impact of the nonlinear and canonical processes on economic outcomes and compare their results. The two models generate small but non-negligible differences in aggregate outcomes. The differences between the models are substantial for individuals who start with poor initial health. Specifically, the canonical model overstates the asset accumulation of individuals below the 10th percentile of health at age 50 by approximately 20 percent. These disparities are even more pronounced for individuals starting with assets below the median.

We use the life-cycle model to simulate aggregate outcomes after health shocks of different magnitudes to examine the role and distributional consequences of health and its persistence. Specifically, we simulate individuals who begin at the age of 51 with different levels of the persistent component of health (specifically the 10th, 50th, and 90th percentile of its distribution). At the age of 52, we impose to the persistent component of health a good, an intermediate, and a bad shock. These shocks are designed to bring the persistent component of health to the 10th, 50th, and 90th percentile of its distribution, conditional on its starting value.[1]

The health asymmetries resulting from different shocks in the nonlinear model

[1]This simulation exercise is inspired by a similar approach proposed by Arellano, Blundell and Bonhomme (2017) when studying income dynamics.

generate substantial economic disparities. Specifically, bad health shocks are more severe and long-lasting on individuals who start with low wealth and are in poor health compared to those in better initial conditions. For example, among individuals starting at age 51 with £10,000 in assets and at the 10th percentile of the persistent component of health, a bad shock induces an accumulation for the survivors at terminal age of £35,000 less than after a median shock. In contrast, starting from the median of the persistent component of health, a bad shock results in an accumulation of £17,000 less than after a median shock. Starting from £200,000 in assets, the same difference reduces to £9,000 and shows little dependence on the initial level of health.

We find that bad shocks generate higher dispersion in accumulated assets among people who start with poor wealth and health relative to people subject to better shocks. A decomposition exercise where we turn off the impact of health on each component of the model affected by health (wages, time endowment, and mortality) reveals that the effects of the components are not additive (nonlinearities are important) and that the adverse effects of bad health are mostly due to its time cost.

The time cost of bad health can be alleviated by investment in infrastructure, mandates, subsidies, incentives that facilitate work-from-home, and other time-saving measures helping workers in poor health. To assess the value individuals place on their time availability, we simulated a counterfactual where we relaxed the time constraint of individuals in poor health. The consumption equivalent of transferring 6 hours per week to individuals below the 20th percentile of health is 0.125%, a meaningful improvement in overall well-being. Such policy has a large effect on the recipients' labor participation in both its intensive and extensive margins; over one-half of transferred hours are used to increase work participation. A monetary transfer of £60 per week, the average cost of purchasing a home-care worker for an equivalent time, would have a higher consumption equivalent but would depress participation and hours worked.

Several articles have documented the important role of health on life-cycle economic outcomes, helping our understanding of the complex interactions between health and economic outcomes. Many structural studies have investigated the role of health in determining different patterns of wealth accumulation (for example Ameriks et al. (2020); De Nardi, French and Jones (2010); De Nardi, French and Jones (2016); Nakajima and Telyukova (2020); De Nardi, Pashchenko and Porapakkarm (2023)) or in explaining heterogeneity in labor supply and earnings over the life cycle (Capatina (2015); French (2005); French and Jones (2011); Hosseini, Kopecky and Zhao (2024);

Low and Pistaferri (2015)).

Most of these studies posit a binary or 3-valued health measure based on the widely available survey question on subjective self-assessed overall health. We depart from this modeling assumption and derive a simple continuous measure based on a large set of (more) objective health indicators in the spirit of Bound (1991), similar to the approach used by Blundell et al. (2016, 2021). Other approaches have been used in the literature: Hosseini, Kopecky and Zhao (2022, 2024) use a continuous measure of health based on a frailty index computed by adding adverse health indicators. Dal Bianco (2023) constructs a continuous health indicator using principal component analysis. Discrete measures of overall health are highly predictive of mortality and generally strongly correlate with objective measures (Idler and Benyamini, 1997; LaRue et al., 1979). However, our continuous measure does not suffer from measurement error and justification bias as much (Bound, Stinebrickner and Waidmann, 2010). Our findings underscore the importance of adopting a continuous health measure to accurately capture and analyze the diverse outcomes observed across different levels of health, especially when focusing on outcome inequality.

In our paper, as in most literature, health evolves exogenously over the life cycle.[2] The dynamic of discrete health is generally assumed to evolve according to a first-order Markov process. An exception is De Nardi, Pashchenko and Porapakkarm (2023), modeling a *binary* health measure with a second-order Markov process with age-dependent parameters, allowing for complex duration-dependence patterns. The few contributions using a continuous measure of health assume that health residuals are the sum of a persistent component, modeled as an AR(1) process, and a transitory one, modeled as i.i.d. shocks (Dal Bianco, 2023; Hosseini, Kopecky and Zhao, 2022, 2024; Blundell et al., 2016). We contribute to the literature on health dynamics, allowing for a more flexible (nonlinear and non-normal) health dynamic and better capturing observed health evolution. We show that using this health process, adverse shocks are costlier, especially for poorer individuals, and generate higher outcome dispersion. We find that a canonical health process could not uncover some of these effects.

We confirm previous findings that poor health has sizable effects on income and asset accumulation (De Nardi, Pashchenko and Porapakkarm, 2023; Capatina, 2015).

[2]Recent contributions to the literature allow endogeneity of health, see Capatina, Kene and Maruyama (2020).

However, unlike previous research, we find that the time cost of bad health, not mortality, is the primary mechanism driving our results. This difference is likely to be driven by modeling assumptions: we depart from Capatina (2015) by introducing in our model bequest motives and the intensive margin of labor supply. The time cost of poor health reduces the available hours to be allocated to work or leisure and significantly affects income through both the extensive and the intensive margin. The limited role of the mortality channel is consistent with bequest motives driving asset accumulation more than survival risk.

Unlike other papers (Capatina, 2015; Hosseini, Kopecky and Zhao, 2024; De Nardi, Pashchenko and Porapakkarm, 2023), we focus on older adults only and not on the entire life cycle because of data limitations: the detailed health information needed to construct a continuous measure of health is typically available only in surveys targeted to the older population.

The rest of the paper proceeds as follows. The next section describes the data we use, the construction of our health measure, and the estimation of the health process. Section 3 describes the life-cycle model and its estimation. In Section 4 we compute the effects of health on aggregate outcomes and their distribution. In Section 5, we simulate the effect of different health *shocks* on the level and the distribution of outcomes. Finally, in Section 6, we report the welfare effects of policies in a counterfactual experiment where we relax individuals' time constraint.

2 Data, Health measurement, and its dynamics

We use data from the first seven waves (2002-2012) of the English Longitudinal Study of Ageing (ELSA), a biennial longitudinal survey representative of the English population aged 50 and above. ELSA contains detailed income and wealth information and several indicators of individuals' physical, mental, and cognitive health status.

We focus on male respondents having a partner. Table 1 shows descriptive statistics conditional on age. Labor supply participation decreases sharply with age, but a small fraction of respondents is still active after age 70. Hours worked, conditional on participation, decreases at age 65 – the state pension age in the UK in the period we consider – from about 2,000 to about 1,000 hours[3]. Accordingly, annual earnings decrease substantially with age, especially after the state pension age. Total

[3]See the complete age profile in Figure 6

Table 1: Descriptive statistics by age

	50-59	60-69	70-90
% working	83.6	43.2	7.5
annual hours worked	1935	1590	942
annual earnings (£)	14,109	9803	2888
wealth (1000 £)	172	205	177

Note: ELSA data, waves 1-7. Men having a partner (about 25,400 observations for 6,700 individuals). Annual hours worked are conditional on working and are computed by multiplying average weekly hours by 46 working weeks. Annual earnings are similarly obtained from weekly earnings, assuming continuous employment during the year. Wealth refers to total wealth, including housing but excluding pension wealth.

wealth increases with age and remains high at older ages, as previously reported in the literature (see, e.g., Blundell et al. (2016)).

2.1 Health measurement

We construct a continuous index of health using a latent variable model. Let ψ_{it} be 'true' health. We do not observe ψ_{it}, but only self-reported subjective health h^s_{it}, a noisy measure of its latent true value, equal to the sum of ψ_{it} and an error term μ_{it}:

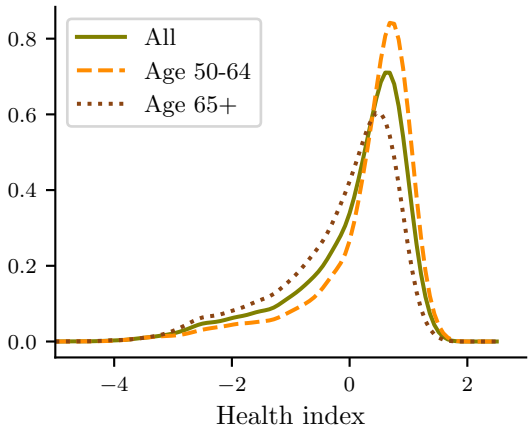
$$h^s_{it} = \psi_{it} + \mu_{it}. \tag{1}$$

Subjective measures have been criticized because they are subject to measurement error, and they have been proved sensitive to justification bias (Bound et al., 1999; Lindeboom and van Doorslaer, 2004). Measurement error and justification bias are captured by μ_{it} in Equation (1). To account for these potential biases, one methodology used in the literature is to instrument subjective measures of health with objective health indicators (see e.g., Stern (1989) and Bound (1991)).[4]

In Equation 2, we substitute ψ with a linear combination of the set of objective health conditions Z. However, as it is unlikely that we observe all the relevant health dimensions, ψ is equal to $Z'_{it}\alpha + \xi_{it}$, with ξ_{it} being a residual term. The identifying assumption is that objective health indicators Z are not related to μ. This assumption

[4]In a recent contribution, Blundell et al. (2021) shows this approach produces similar estimates of the effect of health on employment than one using subjective health measures, provided that a sufficiently rich set of objective measures is used.

Figure 1: Health index distribution



Note: Cross-sectional distribution of the health index in the data (Equation ($h_{it} = Z'_{it}\hat{\alpha}$), solid line, and conditional on age less than 65 (dashed line), or over 65 (dotted line).

might be violated if objective health indicators (i) are affected by justification bias and/or (ii) by measurement error. We assume that objective health indicators (i) are not affected by justification bias and (ii) measurement error in objective health indicators, if present, is not related to measurement error in subjective health indicators, as in Blundell et al. (2021).

$$h^s_{it} = (Z'_{it}\alpha + \xi_{it}) + \mu_{it} \tag{2}$$

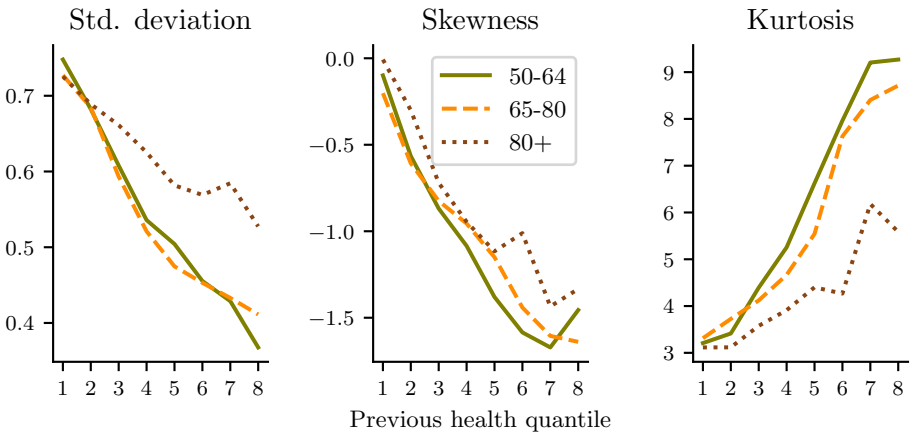
We follow Blundell et al. (2021) and define h^s_{it} as the first component of a principal component analysis conducted on three subjective health indicators included in ELSA data: self-reported general health, work-related health limitations and activity-related health limitations. Among the objective health indicators Z, we include eyesight, hearing, mobility indicators, ADL (limitations with activities of daily living), IADL (limitations with instrumental activities of daily living), depression, heart diseases, other diseases (chronic lung disease, asthma, arthritis, ...), eye problems, incontinence, BMI and grip strength (an objective measure of muscular strength). [5]

We define the continuous health index to be the (standardized) predicted values from equation 2, $h_{it} = Z'_{it}\hat{\alpha}$.

The resulting distribution of the health index is left-skewed, as shown in Figure 1,

[5]The complete list of variables and descriptive statistics are shown in External Appendix B.

Figure 2: Moments of health shocks by age and previous health quantiles



Note: ELSA data, waves 1-6. Men having a partner observed in at least two waves. 3-quantile moving average

and the probability of smaller values increases with age. Its shape is consistent with the one obtained by Hosseini, Kopecky and Zhao (2022) using an alternative health index computed as the sum of adverse health indicators (frailty index).

Figure 2 shows three moments of the health shocks distribution (health shocks are defined as $\Delta h_t = h_t - h_{t-2}$, recall that our data is biennial) conditional on age and initial level of health (h_{t-2}). For low levels of initial health, the shock distribution is almost normal, with a standard deviation of about 0.7. As the initial level of health increases, the distribution has a smaller standard deviation, becomes left skewed, and shows excess kurtosis. For older individuals, the distribution exhibits higher dispersion, larger negative skewness, and lower levels of excess kurtosis. This evidence suggests that health is a highly nonlinear process with some age dependence and justifies the adoption of a flexible specification to reproduce its dynamic.

2.2 Health dynamic

We define the health process as the sum of two components:[6]

$$h_{it} = \eta_{it} + \varepsilon_{it}, \quad i = 1, \ldots, N, \quad t = 1, \ldots, T \tag{3}$$

where η_{it} is a persistent component following a first-order Markov process, whereas ϵ_{it} is a transitory health shock with mean zero, independent of t and η. Hereafter, we use notation h_{it} to denote health residuals. We construct h_{it} as residuals from regressing the health index on a set of demographics, which includes a third-order polynomial in age, year of birth, education, and an indicator for having a partner. We include these controls to capture individual heterogeneity.

To estimate the health process dynamic, we use the quantile-based panel data method developed by Arellano, Blundell and Bonhomme (2017). Let $Q_x(\tau|.)$ be a conditional quantile function that denotes the τ'th conditional quantile of x. The persistent component of the health process can be written as

$$\eta_{it} = Q_\eta(u_{it}|\eta_{i,t-1}, t) \qquad u_{it} \sim \text{Uniform}(0,1), \quad t > 1. \tag{4}$$

With this quantile-based formulation, the persistence of η when hit by a shock of rank τ can be defined as:

$$\rho(\tau|\eta_{i,t-1}, t) = \frac{\delta Q_\eta(\tau|\eta_{i,t-1}, t)}{\delta \eta_{i,t-1}}. \tag{5}$$

This formulation allows persistence to depend upon both the past realization of η and the current shock realization. A special case of (4) is $\eta_{it} = \rho\eta_{i,t-1} + \nu_{it}$, an AR(1) process, with persistence equal to ρ, the autoregression parameter.[7]

We use low-order Hermite polynomials (ψ_k) to parametrize the quantile function $Q_\eta(\tau|.)$. The marginal distributions of the initial persistent component η_{i1} and of the

[6] Because our health data is biennial, for ease of notation in this section one time period corresponds to two years. In External Appendix C, we explain how we reconcile the biennial health process to the annual time span we adopt in the life-cycle model.

[7] See Blundell et al. (2016), Hosseini, Kopecky and Zhao (2022) and Dal Bianco (2023) in which the health persistent component is modeled as an AR(1) process.

transitory component ϵ_{it} follow similar specifications:

$$Q_\eta(\tau|\eta_{i,t-1}, age_{it}) = \sum_{k=0}^{K} a_k^\eta(\tau)\psi_k(\eta_{i,t-1}, age_{it})$$
$$Q_{\eta_1}(\tau|age_{i1}) = \sum_{k=0}^{K} a_k^{\eta_1}(\tau)\psi_k(age_{i1})$$
$$Q_\epsilon(\tau|age_{it}) = \sum_{k=0}^{K} a_k^\epsilon(\tau)\psi_k(age_{it})$$

The functions $a_k^\eta(\tau)$ are modeled as piecewise linear splines on the grid $\tau_1 < \ldots < \tau_P, \in (0,1)$, where P is the number of gridpoints. The intercept coefficients $a_0^\eta(\tau)$ are specified as the quantiles of an exponential distribution on $(0, \tau_1]$ and $[\tau_P, 1)$ with parameters λ_-^η and λ_+^η respectively. Analogous empirical specifications apply to $a_k^{\eta_1}(\tau)$ and $a_k^\epsilon(\tau)$.[8]

To understand the implications of this flexible representation, which we refer to as the *nonlinear model*, we compare it with a *canonical model*, which assumes a standard AR(1) process for the persistent component and a normal distribution for the transitory iid shocks.[9]

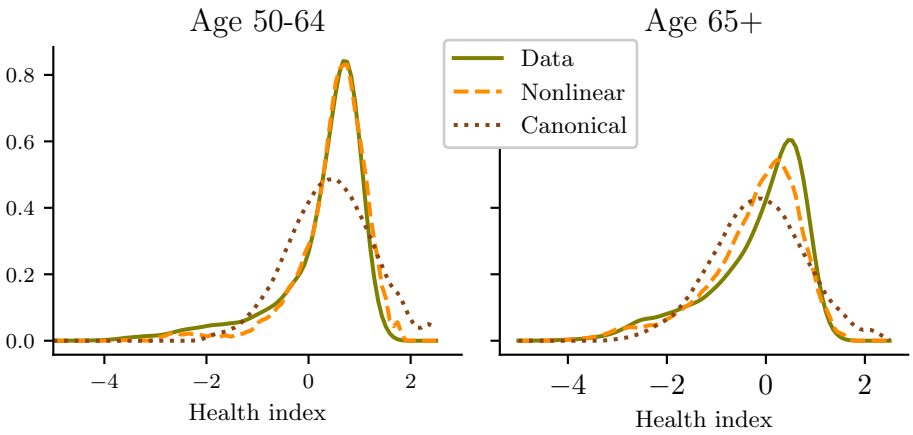
Figure 3 compares the cross-sectional distributions of the health index derived from the *nonlinear* (dashed line) and the *canonical* (dotted line) models. The nonlinear model fits the health index distribution from the data much better than the canonical model, especially for younger individuals. The canonical model is unable to replicate the index's skewness, overstating high values of health and understating low values. Figure H.2 in External Appendix H reports the Nonlinear model fit of the conditional dispersion, skewness, and kurthosis, which are well reproduced. The Nonlinear model also matches the transition probabilities between quintiles of health quite well (see External Appendix H Figure H.3).

The left panel of Figure 4 reports the average value of ρ_t in the nonlinear model as a function of current and previous health deciles computed from the data. The

[8] As in Arellano, Blundell and Bonhomme (2017), we set the number of gridpoints P to 11. For functions ψ_k, we use Hermite polynomials of degree 2 in age and of degree 3 in $\eta_{i,t-1}$.

[9] We adopt Arellano, Blundell and Bonhomme (2017)'s terminology in defining the canonical model. Note that the typical model adopted in the literature studying life-cycle models with health does not adopt an autoregressive process, since it uses only a 2 or 3-valued health indicator. We use the autoregressive model as a comparison to highlight the importance of nonlinearities of the dynamic process.

Figure 3: Health distribution: data and model simulations



Note: Kernel density estimates of the distribution of health index (Data), and simulated data from the nonlinear and canonical model

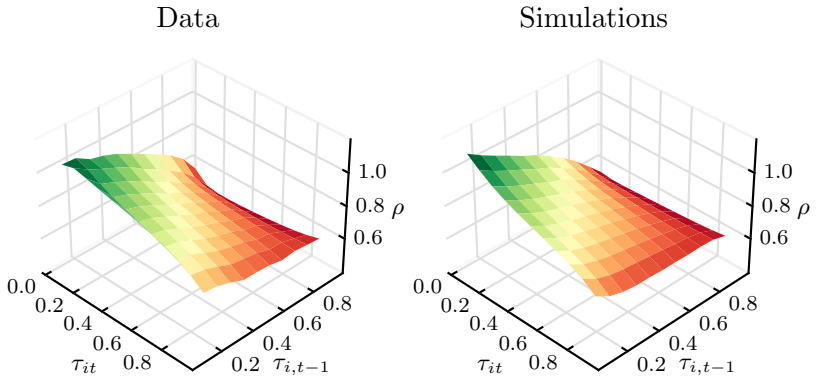
nonlinearity of the health persistence from model simulations (right panel) reproduces the persistence in the data very well. Persistence depends on values of both current and previous health shocks, ranging from about 0.59 to 1.13. For individuals with a small value of the permanent component of health, bad shocks are much more persistent than good shocks (see the left portion of the surface sloping down from a maximum of 1.13 to a minimum of 0.59). Instead, for individuals starting with high values of the permanent component, persistence decreases at a much smaller rate with the value of the current shock (from 0.74 to 0.62).[10]

The canonical model estimate of ρ is 0.953 (s.e. 0.015), by construction independent upon the current period's value of the shock and the previous period's value of the permanent component of health (see External Appendix C.2 for details).

To understand the implications of persistence following a health shock in the nonlinear and canonical models, we simulate individual health histories using the estimated processes. We set the persistent component of the health process at percentiles 10, 50, and 90 of the health distribution at age 51 ($\tau_{init} = .1, .5, .9$). We expose individuals to three shocks at age 52: a bad, median, and good shock that respectively place them at the 10th, 50th, and 90th percentiles of the health distribu-

[10]See Figure H.4 in External Appendix H nonparametric bootstrapped 95% confidence intervals of the nonlinear persistence.

Figure 4: Persistence, nonlinear model



Note: Estimates of the average derivative of the quantile function ρ (Equation 5), computed directly from data (left panel), and from simulations of the nonlinear model estimates, adding the predictions of the persistent and transitory component (right panel).

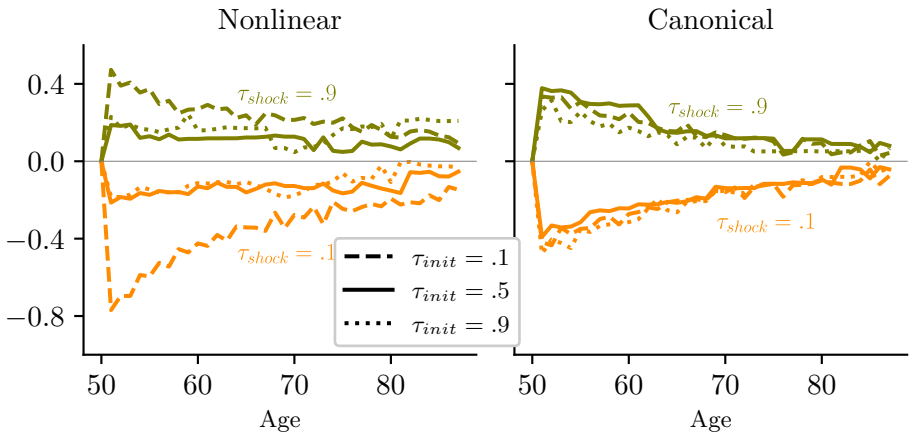
tion conditional on τ_{init}.[11] The resulting health dynamics of the good and bad shock, relative to the median shock, are shown in Figure 5.

The canonical model results in symmetric health patterns after the good and bad shock, close in magnitude for different τ_{init}.[12] The patterns resulting from the nonlinear health process, instead, are asymmetric and depend substantially on the initial value of the persistent component of the health process. In particular, the left panel of Figure 5 shows that a bad shock is worse and more persistent, relative to a median shock, when starting in bad or median health than when starting with good health. Good shocks lead to higher levels of health when starting in good health (relative to a median shock), but their magnitude is quantitatively smaller in absolute value than bad shocks.

[11] We impose the shock at 52, and not 51, because of the biennial nature of the health information in our data. See External Appendix C for details on how we reconcile the biennial health process to the annual time span of the life-cycle model.

[12] In theory, the three lines of each shock level in the right panel of Figure 5 should overlap. The discrepancies observed in the figure are due to approximation errors from the discretization of the health variable.

Figure 5: Health after different health shocks



The figures illustrate the age profiles of the difference in health between individuals subject to a permanent component of health shock τ_{shock} ($\tau_{shock} = 0.9$ green lines and $\tau_{shock} = 0.1$ orange lines) and individuals subject to $\tau_{shock} = 0.5$, starting from different initial levels of the permanent component (τ_{init}). Averages over 10,000 simulated histories per type of shock.

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3 The life-cycle model

We model the behavior of males living in a couple who enter the model at age 50 ($t = 1$), start receiving pension income at 65 (the state pension age in the UK in the period covered by our data), and may work until 70. In each period t, the individual receives utility (U_t) from consumption (c_t) and leisure (l_t). When he dies, he values bequest according to a bequest function, b_t.

Subject to the time and budget constraints outlined below, the individual maximizes his present discounted utility. Current utility depends on consumption and leisure according to the CRRA specification:

$$U(c_t, l_t) = \frac{1}{1-\nu}(c_t^{\gamma} l_t^{1-\gamma})^{1-\nu} \tag{6}$$

where ν is the relative risk aversion parameter and γ is the consumption weight. Upon death, the agent also receives utility from bequests of assets a according to:

$$b(a_t) = \phi_B \frac{(a_t + K)^{(1-\nu)\gamma}}{1-\nu}. \tag{7}$$

Parameter ϕ_B measures the intensity of bequest motives, whereas K regulates the curvature of the bequest function and allows for a finite utility of zero bequests.

In each period, the agent chooses savings a_t, and, only up to the age of 70, labor supply s_t measured in hours worked. With a time endowment set to L, the agent faces a time cost of bad health, which we model as a flexible function $\phi_h(\cdot)$ of health (see Appendix A for details). The time constraint is:

$$l_t = L - s_t - \phi_h(h_t) \tag{8}$$

Health is included in the model by discretizing the transitory and persistent components and the transition matrices of the persistent component at each time t into N dimensional grids.[13] Health uncertainty affects utility through leisure, the wage offer (see below), and the survival probability. We do not consider the medical expenditures channel, which is particularly important in other institutional contexts (De Nardi, French and Jones, 2016; Capatina, 2015; De Nardi, Pashchenko and Porapakkarm, 2023). Medical expenses are less important in the UK, where healthcare is universally provided, at least between age 50 and 70 before the costs of institutionalization arise (they are not covered by the National Health Care system).[14]

Log hourly wages (e) are the sum of a deterministic component, which depends on health and age, and a stochastic one, the sum of a persistent and a transitory shock. We assume that persistence in wages is captured by an AR(1) process and the transitory component captures measurement error only. The budget constraint is:

$$a_{t+1} = (1+r)a_t + s_t \cdot e_t(1 - c_p \mathbb{1}(t < 15)) - tax_t + tr_t - c_t - \phi_w(\mathbb{1}(s_t > 0, t)) \tag{9}$$

Where r is the exogenous interest rate on the accumulated assets, tax_t are taxes, tr_t are transfers, and $\phi_w(\cdot)$ denotes a monetary cost of working.[15] Each agent is endowed with initial pension wealth p, to which he contributes a fraction c_p of his annual earnings; pension wealth is annuitized at a rate r_p at age 65. Transfers tr_t are computed so that consumption c_t is greater or equal to the consumption floor

[13] De Nardi, Fella and Paz-Pardo (2020) use this procedure to incorporate the estimates of a nonlinear model of *earnings* dynamics into a life-cycle model. Additional details are in External Appendix C.

[14] Figure B.1 in the External Appendix shows the marginal importance of out-of-pocket expenditures and voluntary payment schemes in the UK with respect to the US. (Source: OECD statistics.)

[15] We model the fixed cost of work as a monetary cost and not as a time cost (see French (2005)) to improve the fit of the hours worked age profile.

$\underline{c}$. Transfers also include pension income from age 65 onwards. Hours worked s_t are constrained to zero after age 70.

The probability of surviving to period $t+1$ conditional on being alive in period t is a function of age and health in period t, $\pi^{t+1} = \pi(h_t, t)$.[16] We set the terminal age $T = 89$. Time is discounted at factor β. With these assumptions, preferences can be expressed using a recursive formulation with states $X_t = \{a_t, e_t, h_t, p_t\}$, which we use to compute the numeric solution to the optimization problem. The expression for the Bellman equation is in Appendix A.

3.1 Calibration and estimation

Among the preference parameters, we fix the time endowment L to 4880 hours[17], the consumption floor ($\underline{c}$) to £1660, i.e., 10% of male average earnings in the data (as in Capatina (2015)), the risk aversion parameter ν to 4 (in the range of values estimated in French (2005)) and the discount factor β to 0.9756 (as in Low and Pistaferri (2015)). The pension contribution rate c_w is set to 6%, the average contribution rate to Defined Contribution pension funds in the UK, and the annuity rate p_r is 3.78%.[18]

We estimate the life-cycle model using a Simulated Method of Moments, matching the age profiles of average assets, hours worked, and participation by four quantiles of the health distribution (using thresholds at percentiles 20, 30, and 50). The moments to match are obtained from the data controlling for cohort effects (more details are provided in External Appendix F). We match the profile of male respondents born between 1948 and 1952 and having a partner. The same selection is used to derive initial conditions for model simulation. Each simulated individual receives a draw of assets, pension wealth, health, and offered wage from the estimated initial distribution.

Heterogeneity in labor supply by level of health identifies the time cost of being in bad health $\phi_h(.)$. Hours of work and labor force participation pin down the fixed cost of work ϕ_w^1. The age patterns of hours worked and participation help identify the slope of the fixed cost of work ϕ_w^2 and how the fixed cost changes at retirement age

[16] See External Appendix D for details about our computation of the survival process.

[17] The time endowment is set by assuming 305 working days in a year and 16 hours per day to allocate between leisure and work.

[18] The calibrated value for the annuity rate is obtained from the data. We use total pension wealth, compute the actuarially fair annuity rate for each individual aged between 55 and 65 as if he retires at his current age, and take the median value. We consider that age interval because Defined Contribution pensions can be annuitized starting from age 55, and most individuals (about 80%) do it at or before age 65.

Table 2: Nonlinear model parameter estimates

Calibrated			Estimated		
Time endowment	L	4880	Consumption weight	γ	0.4
Consumption floor	$\underline{c}$	1660	Bequest	ϕ_B	0.276
Risk aversion	ν	4		K	838516
Pension annuity rate	p_r	0.0378	Cost of health	$\phi_h(h_{50})$	1662.0
Pension contribution rate	c_w	0.06		$\phi_h(h_{30})$	1682.9
Discount factor	β	0.9756		$\phi_h(h_{20})$	2976.8
Interest rate	r	0.029		$\phi_h(\underline{h})$	2979.4
			Cost of work	ϕ_w^1	2040.9
				ϕ_w^2	30.0
				ϕ_w^3	7.6

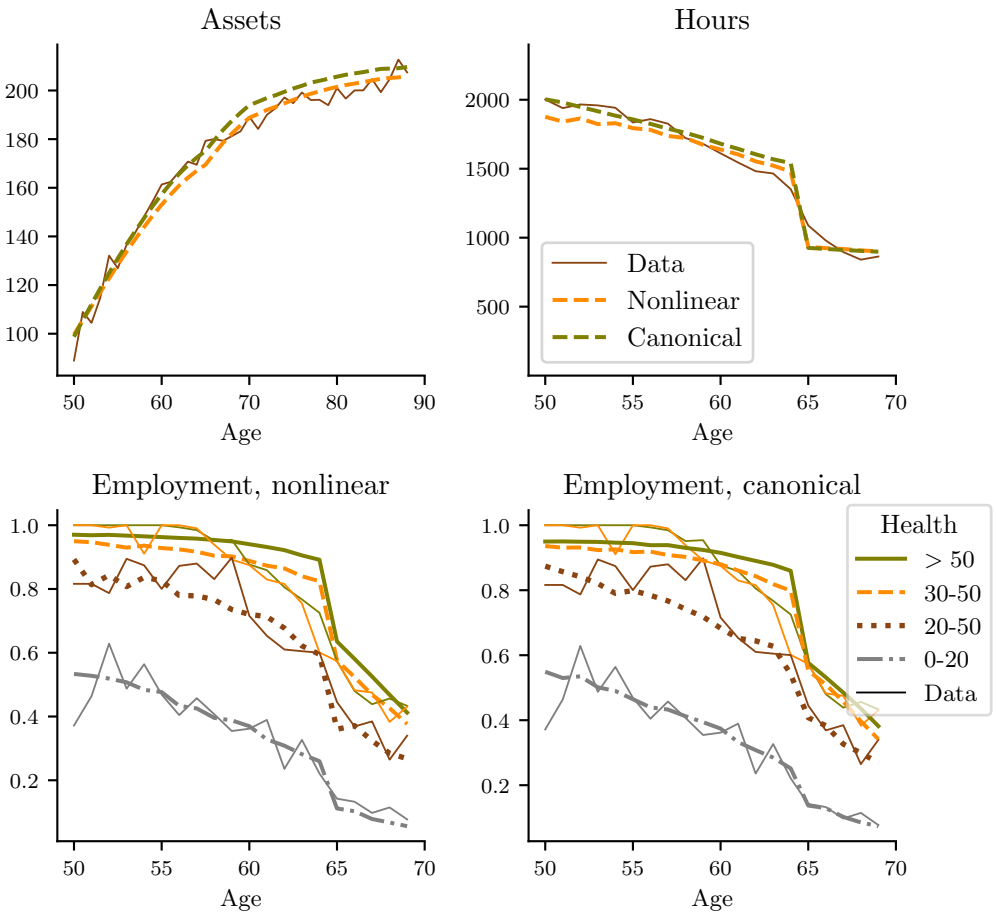
Note: See appendix A for the definition of the parameters of the health function and the cost of work function.

ϕ_w^3. The labor supply profiles identify the consumption weight parameter γ. If death were not stochastic, assets at the time of death would identify the bequest function parameters (ϕ_B and K). When the time of death is uncertain, people save both for precautionary and bequest motives; therefore, these parameters' identification is weak but guaranteed because risk aversion has been fixed.

The nonlinear model estimated parameters are reported in Table 2. Holding labor supply fixed, the estimate for the consumption weight γ implies a coefficient of relative risk aversion for consumption of 2.2 ($\gamma(\nu-1)+1$), in the range of previous estimates in the literature (e.g. French (2005) and Capatina (2015)). To compare the estimated values for the cost of bad health, we compute the average value of our health index when self-reported binary health is respectively fair/poor (-1.06) or excellent/very good/good (0.34). The corresponding time costs are 2677 and 1639. Being in bad health entails a 27% reduction in the time endowment relative to being in good health. The same figure is 21% in Dal Bianco (2023), 12% in French and Jones (2011) and 14-21% in Capatina (2015).

The two bequest parameters imply a marginal propensity to bequeath of 0.98, and that the bequest motive becomes operative when the consumption value of total wealth exceeds £16,500 (see De Nardi, French and Jones (2010) and Dal Bianco (2023)). These values align with those implied by estimated parameters in French (2005). Finally, the fixed cost of work ϕ_w^1 at age 50 corresponds to a monetary value

Figure 6: Estimation fit



Note: Data and simulated age profiles of average assets (in £1000), average hours worked conditional on being employed and employment rate. Simulations of 30,000 histories, nonlinear and canonical models.

equal to 10% of average earnings at age 50, reaching 28% at age 65.

The fit of the models, estimated using both the Nonlinear and the Canonical health processes is displayed in Figure 6. Despite the two alternative health processes producing different health persistence, as shown in Section 2, the fit of the life-cycle models using the linear and the canonical they replicate the dynamic of the targeted moments equally well. Figure H.5 in the External Appendix shows the match for earnings, which reflects both the wage process and the labor supply decision (extensive and intensive margins). The simulated profiles reproduce these non-targeted data moments closely as well. However, the two models' different health dynamics produce

Table 3: Aggregate effects of nonlinear dynamics

Initial health/Variable	Nonlinear		Canonical	
	All	**Low assets**	**All**	**Low assets**
All				
Assets ($\times 1,000$)	164	106	+2.2%	+5.1%
Cumulated earnings (log)	5.32	5.53	+5.3%	+5.5%
Employment rate	0.79	0.87	-6.4%	-3.6%
Hours	1625	1749	+4.4%	+3.5%
Percentiles 0-10				
Assets ($\times 1,000$)	93	48	+20.8%	+44.1%
Cumulated earnings (log)	4.85	4.96	+4.8%	+5.0%
Employment rate	0.70	0.73	-5.8%	+0.5%
Hours	1324	1385	+7.2%	+5.4%
Percentiles 10-25				
Assets ($\times 1,000$)	137	84	+3.1%	+0.6%
Cumulated earnings (log)	5.24	5.45	+5.2%	+5.4%
Employment rate	0.79	0.88	-8.3%	-5.4%
Hours	1536	1633	+5.3%	+3.6%
Percentiles 25-100				
Assets ($\times 1,000$)	177	121	+0.9%	+2.1%
Cumulated earnings (log)	5.39	5.64	+5.4%	+5.6%
Employment rate	0.80	0.89	-6.1%	-3.4%
Hours	1675	1827	+4.0%	+3.4%

Note: The table reports the average value of assets, the log of cumulated earnings, the employment rate of people of working age, and hours worked conditional on working, in the nonlinear model, and percent deviations from the nonlinear model values for the canonical model. Columns "Low assets" condition on individuals with initial assets below the median. Results are reported for All health levels (top panel) and by quantiles of initial health (bottom three panels). Averages of 30,000 simulated histories.

different implications, as we will show below.

4 The economic effects of nonlinear health dynamics

To assess the economic consequences of alternative health process specifications, we run 30,000 individual histories and analyze the resulting impact on various economic aggregates and their dispersion.

The first two columns of Table 3 present the computed statistics derived from the Nonlinear model simulations, for all individuals (first column) and individuals with initial assets below the median (second column). Health plays a significant role in exacerbating inequality, particularly among those with lower wealth. Individuals below the 10th percentile of health possess only about 53% of the assets possessed by individuals above the 25th percentile of health (£93 vs. £177 thousand). However, when we shift our focus to individuals below median initial assets, the ratio decreases to 40%. Additionally, the cumulated earnings of the least healthy group are lower by 0.54 log points compared to the healthier category. This difference increases to 0.68 when considering individuals below the median initial assets. In Section 4.2 below, we will delve further into the effects of health on inequality.

On average, the nonlinear and the canonical models (last two columns of Table 3), exhibit moderate differences in outcomes. However, conditioning on the first decile of initial health, we observe notable differences. The Canonical model tends to overestimate assets by over 20%, hours worked by 7%, and cumulated earnings by 5% when compared to the Nonlinear model.[19] Moreover, we observe an understatement of labor participation in the Canonical model. When we further condition on individuals having initial assets below the median, the disparities between the models become even more pronounced. Specifically, the Canonical model forecasts that individuals in the first decile of health and initial assets below the median display assets that are, on average, 44.1% higher compared to the estimates provided by the nonlinear model. In Section 5 below, we will explore how the two health processes also diverge in their predictions of outcomes following health shocks. For the rest of this section, we will focus on analyzing results obtained from the Nonlinear model.

[19] Cumulated earnings are computed as in Guvenen et al. (2022) and Hosseini, Kopecky and Zhao (2024).

Table 4: Decomposition of the effects of health on outcomes

Effects removed	Assets	Earnings	Empl.	Hours	CEV
None (baseline)	164	5.3	0.79	1625	–
(a) Counterfactual health: 90th percentile					
All (relative to baseline)	+17.4%	+7.1%	+10.0%	+20.1%	–
Decomposition:					
Mortality	-0.1%	+0.1%	-0.4%	-0.6%	–
Time cost	+13.4	+5.9	+9.1	+20.8	+1.41
Wages	+2.7	+1.4	+2.5	-0.3	+0.08
Time+Wages	+16.6	+7.0	+10.0	+20.9	+1.54
Mortality+Time	+14.3	+6.0	+9.0	+20.2	–
Mortality+Wages	+2.6	+1.5	+2.1	-0.9	–
(b) Counterfactual health: 10th percentile					
All (relative to baseline)	-21.1%	-8.2%	-16.5%	-22.0%	–

Note: The table reports average outcomes in the baseline model simulations (first row) and percent changes from the baseline after assigning everyone either the mortality rate, time cost of health, or wage offer computed at the indicated percentile of the age-specific health distribution. Assets measured in £1000 averaged throughout the life cycle. Earnings are the log of cumulated earnings at 65. Employment rate and hours averaged from age 50 to 69. Hours are annual, conditional on employment. See Table H.6 for the decomposition at the 10th and 50th percentiles of health.

4.1 Costs of bad health, benefits of good health, and their sources

In our life-cycle model, health affects economic outcomes through three channels: mortality, time available for leisure and work, and wages. To ascertain the relative importance of these channels, we turn them off by assigning to all individuals the mortality, the time cost of bad health, and wages equal to the values these variables assume at a given quantile of the health distribution, conditional on age. These counterfactuals generate both income effects (higher income and time available) and substitution effects (higher hourly wages), as well as dynamic effects from changes in mortality and uncertainty about future health.

Removing the effect of health has very different effects on economic outcomes depending on the reference health level. We report in Table 4 results obtained by

choosing the 90th percentile and the 10th percentile as reference point.[20]

The cost of being in bad health is asymmetric to the benefit of being in good health: removing health effects through all three channels, average assets are 17% higher if health were equal for everyone to the 90th percentile of its age-conditional distribution but 21% lower if health were set at the 10th percentile. Cumulated earnings are 7% higher (8% lower with health at the 10th percentile), employment and hours increase by 10% and 20%, respectively. These findings are in line with those of De Nardi, Pashchenko and Porapakkarm (2023), suggesting that a remarkable portion of the health monetary costs is due to income losses.

The decomposition of these effects reveals that the time cost contributes the most to all outcomes. When health increases, the reduced time cost shifts the static budget constraint outwards, increasing leisure, hours worked, and participation and making saving less costly. The income effect induced by higher wages differs from that induced by lower time costs because the available time does not change. Consequently, hours worked slightly decrease (the substitution effect dominates), and higher earnings and employment generate a more moderate effect on accumulated assets.

Mortality alone has a negligible contribution to all outcomes. This result differs from previous findings in the literature. Capatina (2015), finds that shutting off mortality is the main driver of health effects when everyone is assigned to the high level of health of a 3-valued health variable. There are several explanations for this difference. Our model includes bequest motives to reproduce the high level of wealth owned by individuals at the end of their lives, limiting the role of the mortality channel, which is consistent with bequest motives driving asset accumulation more than survival risk. Differently from Capatina (2015), we also model the intensive margin of labor supply, which plays an important role in the time allocation between leisure and work, and therefore to the cost of bad health. Finally, we only model the behavior of older adults, not the entire life cycle, which might reduce the importance of the mortality risk channel in our setup.

The effect of the three components is not additive, a result of the model's non-linearities. For example, the effect on assets of mortality and time cost combined is more than the sum of the two effects: the reduced cost of time makes it less costly to

[20] In External Appendix H, Table H.6 we report the results when health levels are set at the and 50th percentile, together with the full decomposition of the effects for both the 10th and 50th percentile experiments.

save for the increased lifespan.

We also computed a welfare measure, the consumption equivalent that keeps expected utility at the start of life constant relative to the baseline. Unsurprisingly, health has a large impact on welfare: when the time cost of health is set at the value taken by those with health at the 90th percentile, the magnitude of the gain in terms of consumption equivalent is 1.4%, compared to a 0.08% when the wage offer is the one received by those with health at 90th percentile. When health is set at the 10th percentile (see table H.6 in External Appendix H), the negative contribution of these factors is much larger, again indicating substantial asymmetries.[21] Motivated by these findings, in Section 6 we investigate the welfare effect of relaxing individuals' time constraints.

4.2 The effect of health on inequality

Our measure of health allows us to compute the effects of health on inequality at a granular level. The findings from the preceding section underscore the significance of the model's nonlinearities and the interdependence among different channels when evaluating outcomes. Introducing a continuous measure of health enables us to show that different levels of health have different impacts both quantitatively and qualitatively, and affect both outcome levels and their variability.

We compute the effects of health on inequality by performing an exercise similar to the one performed to compute Table 4. Setting everyone to the same percentile of health (conditional on age) contributes to reducing variation because all individuals of the same age are assigned the same health level. However, this experiment is equivalent to imposing a health shock, and individual responses to these shocks may vary depending on their initial conditions, which may increase variation.

Table 5 shows that removing the effect of health decreases inequality when setting health at the 90th percentile: removing all health effects reduces the asset 80/20th percentile ratio by 1.15, or 34%. The standard deviation of assets also decreases substantially, and so does the inequality of cumulated earnings at 65. This effect is in line with the 30% reduction in the *variance* of log earnings at age 65 estimated

[21] We do not report the welfare effect when the mortality channel is switched off because in any counterfactual where mortality is changed, the number of years individuals stay alive changes, affecting the expected total utility at the start of life. Since utility is invariant up to an affine transformation, additional life years may increase or decrease utility depending on the sign of its yearly value.

Table 5: Measures of inequality

Effects removed	Assets		Cum. earnings	
	80/20pct	Std.	80/20pct.	Std. (log)
None (Baseline)	3.41	98	2.45	0.74
(a) Counterfactual health: 90th percentile				
All (relative to baseline)	-34%	-9%	-23%	-26%
Decomposition:				
Mortality	-0%	-0%	-1%	-1%
Time cost	-29	-8	-22	-22
Wages	-7	-1	-5	-7
Mortality+Time	-31	-8	-23	-23
Mortality+Wages	-7	-1	-6	-8
Time+Wages	-31	-8	-23	-25
(b) Counterfactual health: 10th percentile				
All (relative to baseline)	+68%	+1%	-2%	-3%

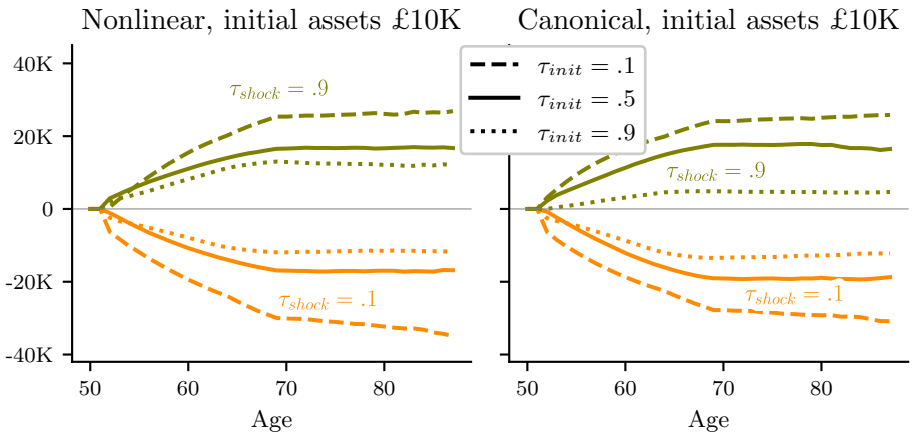
Note: The table reports average outcomes in the baseline model simulations (first row), and percent changes from the baseline after assigning everyone either the mortality rate, time cost of health, or wage offer computed at the indicated percentile of the age-specific health distribution. Assets and Cumulated earnings measured in £1000 averaged throughout the life cycle.

by Hosseini, Kopecky and Zhao (2024) for the US in a similar exercise. Even if the comparison is to be done with caution as the institutional contexts differ, it suggests that the inequality in earnings due to health is almost entirely generated at the end of the working career.[22] We also find that the time cost of bad health is the most important contributor to inequality generated by health.

Interestingly, indices of inequality either increase (Assets) or decrease by a small amount (Cumulated earnings) if health is set at the 10th percentile (Table 5 panel (b)). This is because bad health shocks have an especially adverse effect on poorer individuals, who are more likely to participate less, and accumulate fewer assets (see Section 5 for details); these factors contribute to the inequality increase.

[22]Their model differs from ours: they include disability insurance benefits in the choice set but do not model the extensive margin of labor supply.

Figure 7: Assets after different health shocks for the initially poor



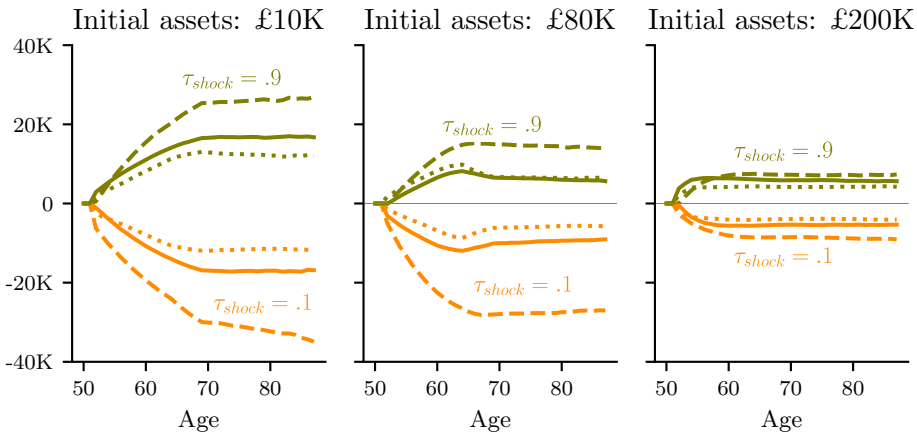
Note: Age profiles of the difference in assets between individuals subject to a permanent component of health shock τ_{shock} ($\tau_{shock} = 0.9$: green lines above zero and $\tau_{shock} = 0.1$ orange lines below zero) and individuals subject to $\tau_{shock} = 0.5$, starting from different initial levels of the permanent component (τ_{init}). Averages of 10,000 simulated histories per counterfactual. Individuals with £10,000 in initial assets. Nonlinear model (left panel) and canonical model (right panel) health dynamic.

5 The effects of health shocks

To assess the economic effect of a health shock and evaluate the nonlinear health dynamic's impact, we simulate individuals' histories under the same conditions used to generate Figure 5. That is, we study individuals starting with the persistent component of the health process at the 10th, 50th, and 90th percentile of the health distribution at age 51 ($\tau_{init} = .1, .5, .9$), and we expose them to a bad, medium, and good shock that respectively place them at the 10th 50th, and 90th percentiles of the health distribution conditional on τ_{init}.

The asymmetries of the health shock dynamic in the nonlinear model have economic effects. Figure 7 reports the age profiles of cumulated assets in the Nonlinear and, for comparison, the Canonical model. In the Nonlinear model, when people start relatively poor, with £10,000 in assets at age 50, a bad shock induces an accumulation at the terminal age of about £35,000 less than after a median shock. This difference is much lower when starting at higher levels of health: £17,000 when $\tau_{init} = .5$ and less than £12,000 when $\tau_{init} = .9$. The gains after a good shock are smaller in magnitude, amounting at about £27,000, £17,000, and £12,000 respectively starting from

Figure 8: Assets after health shocks by initial wealth, nonlinear model



Note: Age profiles of the difference in assets between individuals subject to a permanent component of health shock τ_{shock} ($\tau_{shock} = 0.9$ green lines and $\tau_{shock} = 0.1$ orange lines) and individuals subject to $\tau_{shock} = 0.5$, starting from different initial levels of the permanent component (τ_{init}). Averages over 10,000 simulated histories per counterfactual using the Nonlinear model health dynamic, for individuals with three different values of initial assets

.

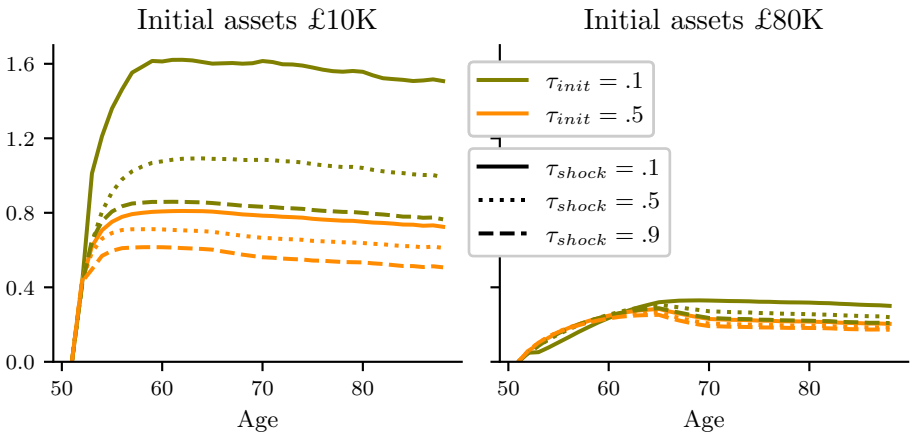
$\tau_{init} = .1, .5$, and $.9$.

The losses from bad shocks are smaller and less asymmetric in the canonical model. They range from £30,000 to £12,000 at age terminal age. Note that in the canonical model, even though the effect of shocks on health is theoretically identical across values of initial health, the effects of these shocks on asset accumulation are not symmetric because of the nonlinearities in the life-cycle model.

The economic effects of bad shocks are much weaker in absolute terms when people are wealthier, as illustrated in Figure 8, reporting results only from the Nonlinear model for individuals with three different values of initial assets. Differences in asset accumulation after a shock are much smaller when people start with higher wealth, indicating the potentially exacerbating effect of poor health on inequality.[23]

[23]Patterns for participation and hours worked are illustrated in External Appendix figures H.6(a) and H.6(b).

Figure 9: Assets, coefficient of variation following a shock



Note: Age profiles of the coefficients of variations of accumulated assets for different levels of τ_{init}, τ_{shock}, and initial assets. Average of 10,000 histories per simulation.

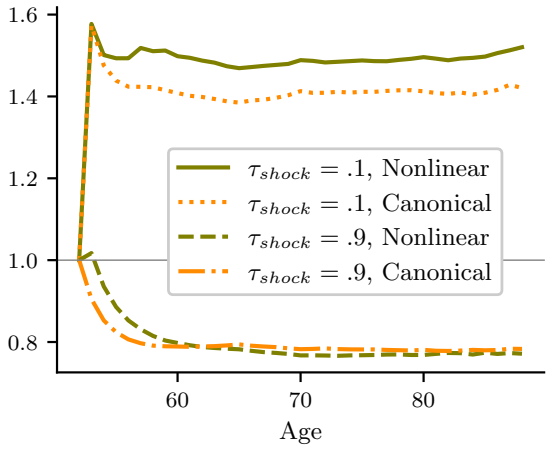
5.1 The effects of bad health shocks on inequality

The interpretation of the previous subsection's results is confirmed if we analyze the effect of the health shocks used to generate Figure 5 on inequality. Figure 9 displays the coefficient of variation of assets, by age, following permanent health shocks of three different intensities (marked by lines with different patterns) at the age of 52, and starting from a low or median level of the permanent component of the health shock (marked by lines of a different color).

The dispersion in assets across individuals is higher when starting from a lower initial level of the health shock and when starting with low wealth (solid green line in the left panel). For poorer individuals, dispersion increases dramatically after a bad shock, peaking above 1.6 before the age of 60, and remaining higher than 1.4. On the other hand, for individuals with the same initial wealth, dispersion is lower after a good shock (dashed line) than after a median shock (dotted line). This result follows because while the variance of assets is approximately constant after the shock, average assets decrease after a bad shock. Differences in dispersion are smaller for individuals starting with a median level of the health shock (yellow lines) and are negligible for initially wealthier individuals (right panel), and (not reported in the figure) are even smaller for people starting from an initial level of wealth of £200,000.

The persistence of bad shocks affects asset accumulation permanently, especially

Figure 10: Assets, coefficient of variation following a shock



Note: Age profiles of the ratio of the coefficients of variations of accumulated assets for levels of $\tau_{shock} = \{0.1, 0.9\}$, and the coefficient of variation of the same variable for $\tau_{shock} = 0.5$. Individuals starting with $\tau_{init} = 0.1$ and initial assets equal to £10,000. Average of 10,000 histories per simulation.

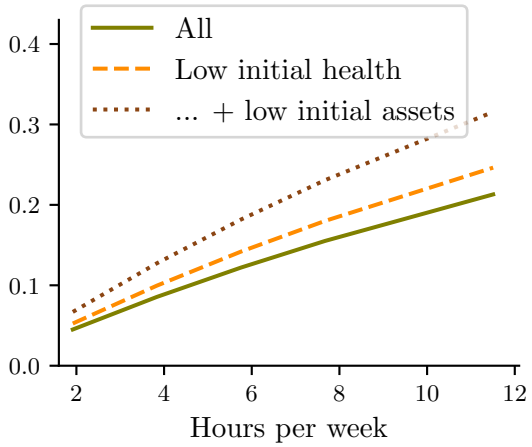
so for individuals starting with a low level of wealth. This evidence helps explain the differential effects of health we uncovered previously when discussing the decomposition results of Table 5. Bad health shocks affect inequality differently by wealth, contributing to increasing variation when health is reduced to the 10th percentile for all. Good health shocks do not affect inequality as differently; hence when all effects of health are computed at the 90th percentile, the equalizing-health effects dominate, and indices of inequality are lower than the baseline.

5.2 The canonical model does not uncover the same differential effects on inequality

Nonlinearities in the health process contribute to generating the asymmetric effects of good and bad shocks displayed in the left panel of Figure 9. In the nonlinear model, four years after a bad shock, the coefficient of variation of assets is about 50% larger than it is after a median shock and remains this high until the end of the life cycle. In the canonical model, variation increases, but not as dramatically, reaching about 40% of its level after a median shock by age 60 (see Figure 10).

For individuals starting off at age 50 with higher wealth, the two models perform

Figure 11: Consumption equivalent variation of time transfers



Note: Consumption equivalent keeping expected utility the same at age 50, relative to a counterfactual time transfer expressed in the horizontal axis. Dashed line: individuals with health below the 20th percentile. Dotted line: individuals with initial health below the 20th percentile and initial wealth and wealth below the 20th percentile

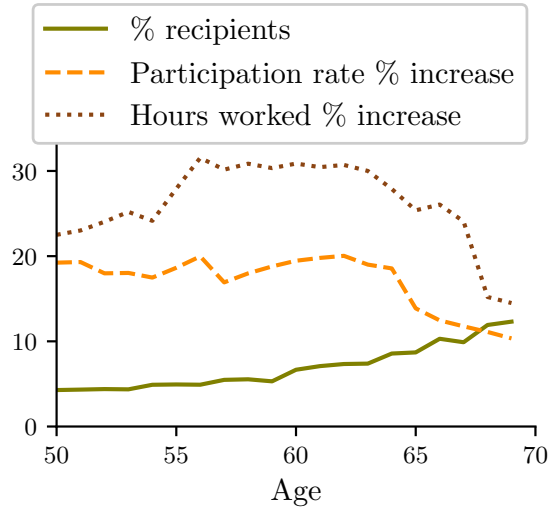
similarly. These results show that for poor individuals, the canonical model cannot generate the same dispersion of outcomes generated by health shocks.

6 Counterfactual: the provision of time to individuals in poor health

Our findings indicate that the availability of time is a significant factor that limits the well-being of individuals in poor health. Poor health hampers one's ability to work and enjoy leisure activities. While individuals can partially alleviate this constraint by purchasing home care or utilizing time-saving technology, governments can contribute by mandating the removal of architectural barriers, promoting work-from-home options, and investing in infrastructure to save time for people in poor health. While it is outside the scope of this paper to provide a full cost-benefit analysis of such initiatives, one way to measure the benefits of time-saving policies within the context of this model is to compute the consumption equivalent variation resulting from the relaxation of the time constraint.

In our counterfactuals, we relaxed the time constraint to individuals whose health

Figure 12: Age profiles of changes in participation with a 6-hour time transfer



Note: Labor market effects of transferring 6 hours of time to individuals in poor health. The yellow dashed line reports the percentage point increase of the participation rate among recipients. The Dotted brown line reports the percent increase in hours worked among working recipients who also worked in the baseline.

falls below the 20th percentile of the unconditional health distribution. The consumption equivalent variation of this time transfer is reported in Figure 11. Six additional hours of time per week has a non-negligible impact on their well-being, resulting in an increase of 0.12% in consumption equivalent terms, with a larger impact among the less healthy and wealthy.

The transferred time helps recipients to increase participation, hours worked, and leisure substantially. Most of the transfers occur when individuals are not of working age. Among individuals of working age, the transfer is received at least one year by about 30% individuals, and in 7% of individual-year observations of working age. With a transfer of 6 hours per week, the participation rate increases between 10 and 20 percentage points at every age (see Figure 12) among recipients, and hours increase on average 27% among people who also worked in the baseline. These numbers are large in part because the baseline participation and hours are small among low-health individuals. In the aggregate, participation increases by 1.5% and total hours worked by 0.9%.[24] Hours worked in years in which the transfer is received increase by 3.5 per

[24]There are also dynamic effects among the non-recipients, although negligible in size.

week, hence, a sizeable portion of the transfer is spent in leisure activities.

Individuals are willing to pay £90 per year for this time transfer.[25] Considering that the average wage of a social care worker (which could provide some time-saving labor for the recipient) is about £10 per hour in 2003[26], purchasing these services would cost about £3000 per year. The agents' willingness to pay to transfer £3000 to low-health individuals would be about £220 per year, indicating that individuals would prefer a cash transfer. However, a cash transfer policy would increase the employment rate more modestly, decrease hours worked among recipients, and decrease both participation and hours among non-recipients. In the aggregate, participation would decrease by 1.1% and total hours by 1%.

While these figures are somewhat speculative, primarily due to the lack of accounting for the potential negative impact on wages, prices, and profits resulting from mandates, as well as the increased taxation required to fund time-saving public investments, they affirm our finding that the time cost of poor health significantly affects individuals' well-being.

7 Conclusion

This paper shows that the health dynamic is highly nonlinear and that the quantile-based methodology developed by Arellano, Blundell and Bonhomme (2017) can fit the health process' main features. Incorporating the estimated health process into a life-cycle model reveals substantial heterogeneity in the response to severe health shocks by initial health and wealth. A canonical model of health dynamics that fits the life-cycle profiles of assets, labor force participation, and hours worked equally well generates different economic effects of health shocks, especially for poor individuals subject to a bad shock. Our research reveals the importance of accounting for a flexible health dynamic, especially when investigating the distributional effects of health shocks on poor individuals.

Our simulations highlight the time cost imposed by bad health as the most significant factor affecting sick individuals' welfare. This result suggests the importance of policies to relieve individual health-related time constraints, such as facilitating work

[25] We compute this figure as the lump-sum tax applied to *all* citizens in the sample that equalizes the average expected utility at age 50 with the same figure in the baseline.

[26] See https://www.communitycare.co.uk/2003/12/04/social-care-wages-in-england/, last accessed: 9/15/2023.

from home, providing home care, and removing architectural barriers.

Because of computational limitations, we did not consider extensions of our model that may appear natural and important, such as the joint nonlinearity of the health and income processes, the endogeneity of the health process, spousal income, the joint decision of work when living in a couple, and private health expenditures. These are important factors that we leave to future research. Nonetheless, we believe our results highlight the importance of adopting a detailed measure of health, with a nonlinear dynamic, when trying to assess economic outcomes and their distribution.

A Appendix: model and estimation details

Following De Nardi, Fella and Paz-Pardo (2020), after estimating the nonlinear health process using Arellano, Blundell and Bonhomme (2017)'s procedure, we simulate health histories and discretize the persistent and transitory components of health at each age and then compute the transition matrices for the persistent component. See the External Appendix for additional details and descriptions of the estimation of the canonical health and income processes, mortality rates, and wealth profiles.

To define the Bellman Equation, let the state space be $X_t = \{a_t, e_t, h_t, p_t\}$. The dynamic programming problem for workers is to maximize over hours worked and next period's assets the Bellman equation:

$$
\begin{aligned}
V(X_t) &= \max_{a_{t+1}, s_t} \Bigg\{ U(c_t, l_t) + \beta \pi^{t+1} \iint_{e_{t+1}, h_{t+1}} V(X_{t+1}|X_t) dF(X_{t+1}|X_t) \qquad \text{(A.1)} \\
&\quad + \beta(1-\pi^{t+1}) b(a_{t+1}) \Bigg\} \\
&\quad \text{s.t.:} \\
a_{t+1} &= (1+r)\cdot a_t + s_t \cdot e_t \cdot (1 - c_p \mathbb{1}(t<15)) - tax_t + tr_t - c_t - \phi_w(\mathbb{1}(s_t>0, t<15)) \qquad \text{(A.2)} \\
l_t &= L - s_t - \phi_h(h_t) \\
tax_t &= f(s_t \cdot e_t \cdot (1 - c_p \mathbb{1}(t<15)) + r_p \cdot p_t \mathbb{1}(t \geq 15), r \cdot a_t) \\
tr_t &= \max\left(0, \underline{c} - (a_t + r_p \cdot p_t \mathbb{1}(t \geq 15) + s_t \cdot e_t \cdot (1 - c_p \mathbb{1}(t<15)) + r \cdot a_t - tax_t)\right) \\
a_t &\geq 0
\end{aligned}
$$

Transfers tr_t are computed so that $c_t \geq \underline{c}, \forall t$. States h_t and e_t evolve according

to the processes specified in Appendices C and E, with earnings e_t equal to zero if $t \geq 21$ (after age 69). Assets a_t evolve according to constraint (A.2), and pension wealth increases in each period by a fraction c_p of earned income, such that $p_{t+1} = p_t + e_t \times c_p \mathbb{1}(t < 15)$, up to state pension age.

The time cost of health $\phi_h(h_t)$ is a 5-knots spline defined between $\underline{h}$ and $\hat{h}$, the minimum and maximum value of health in our simulated sample, and with knots $\underline{h}, h_{20}, h_{30}, h_{50}, \hat{h}$, where the three intermediate knots are the 20th, 30th, and 50th percentile of the simulated health distribution (respectively, -.697, -.279, .218). We constrain $\phi_h(\hat{h}) = 0$. Table 2 reports the values of ϕ_h at the remaining four knots.

The cost of work is a linear function

$$\phi_w(\mathbb{1}(s_t > 0, t)) = \begin{cases} \phi_w^1 + \phi_w^2 \cdot t, & \text{if } t < 15 \\ \phi_w^1 + \phi_w^2 \cdot t + \phi^3 \cdot s_t & \text{if } t >= 15 \text{ and } s_t > 1250 \end{cases}$$

The extra cost suffered by older workers working full-time is identified by the sudden drop in participation and hours worked occuring at the age of 65 ($t = 15$).

To estimate the model, we simulate 30,000 histories and minimize the sum of the squared difference between simulated moments and moments from data. We target 135 averages: accumulated assets by age (35 moments), hours worked if working, by age (20 moments), and labor force participation by age and 4 health quantiles (below the 20th percentile, between the 20th and the 30th percentiles, between the 30th and the 50th percentiles, and above the 50th percentile) (80 moments).

We discretize the state space of assets, state pension, wages, and transitory and permanent components of health, respectively using 30, 6, 5, 5, and 19 (24 in the canonical model) gridpoints. Grid points for assets and for the transitory and permanent components of health are more finely discretized for lower values.

To minimize the loss function, we adopt a combination of simulated annealing and downhill simplex procedures, starting from several initial values of the parameter vector and stopping when the routine converges to a parameter vector equal to the initial starting vector. We also constrained $K > 0$ and $\phi_h(\cdot) < L$.

External appendix

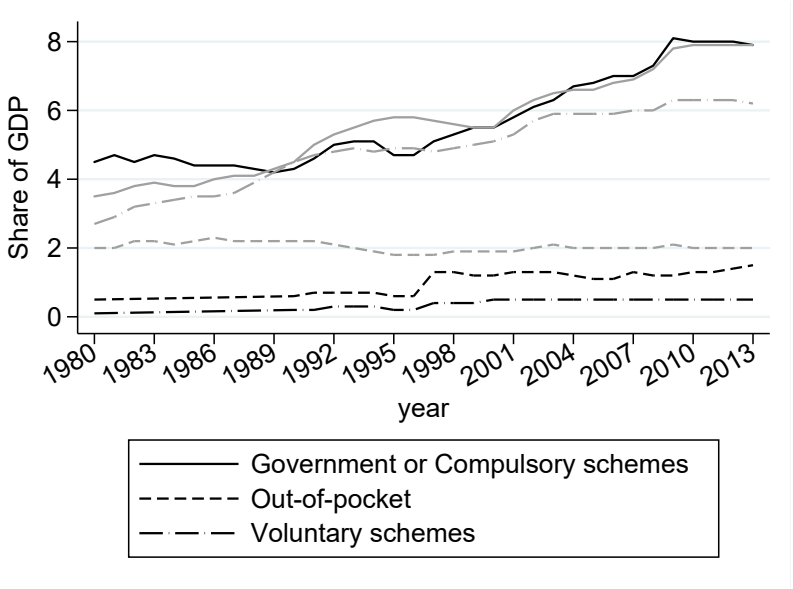
B Data

Table B.1: Health measures descriptive statistics

	Age			
	50-59	60-69	70-90	All
Self-reported health measures:				
self-reported health (1 for excellent , 5 for poor health)	2.42	2.59	2.78	2.61
health limits activities (1 for never, 4 for often)	0.83	1.01	1.34	1.06
health limits work (Yes/No)	0.20	0.26	0.36	0.27
eyesight (from 1=excellent to 6=blind)	2.35	2.38	2.58	2.44
hearing (from 1=excellent to 5=poor)	2.51	2.75	3.05	2.78
ADL/IADL: difficulty				
walking 100 yards	0.053	0.076	0.11	0.082
sitting 2 hours	0.11	0.11	0.099	0.11
getting up from chair after sitting long periods	0.14	0.19	0.23	0.19
climbing several flights stairs without resting	0.15	0.21	0.36	0.24
climbing one flight stairs without resting	0.054	0.074	0.12	0.082
stopping, kneeling or crouching	0.22	0.27	0.38	0.29
reaching or extending arms above shoulder level	0.067	0.076	0.086	0.076
pulling or pushing large objects	0.072	0.086	0.12	0.092
lifting or carrying weights over 10 pounds	0.080	0.11	0.15	0.11
picking up 5p coin from table	0.024	0.033	0.049	0.036
dressing, including putting on shoes and socks	0.079	0.11	0.15	0.11
walking across a room	0.013	0.011	0.015	0.013
bathing or showering	0.039	0.047	0.084	0.057
eating, such as cutting up food	0.0085	0.0086	0.013	0.010
getting in and out of bed	0.040	0.040	0.040	0.040
using the toilet, including getting up or down	0.014	0.016	0.020	0.017
using map to figure out how to get around strange place	0.013	0.015	0.023	0.017
preparing a hot meal	0.017	0.014	0.023	0.018
shopping for groceries	0.035	0.034	0.046	0.038
making telephone calls	0.011	0.013	0.033	0.019
with communication (wave 4 onwards)	0.068	0.079	0.12	0.091
managing money, eg paying bills,keeping track expenses	0.018	0.013	0.020	0.017

depression: CES-D questions answered yes (from 1 to 8)	1.15	1.02	1.07	1.07
disgnosed conditions:				
angina	0.028	0.065	0.12	0.072
heart attack	0.030	0.065	0.11	0.070
congestive heart failure	0.0025	0.0043	0.0064	0.0045
heart murmur	0.019	0.021	0.042	0.027
abnormal heart rhythm	0.043	0.067	0.11	0.074
stroke	0.012	0.029	0.063	0.035
high blood pressure or hypertension	0.28	0.36	0.44	0.37
diabetes or high blood sugar	0.078	0.11	0.14	0.11
chronic lung disease such as chronic bronchitis or emphysema	0.028	0.050	0.062	0.048
asthma	0.079	0.097	0.092	0.090
arthritis (including osteoarthritis , or rheumatism)	0.19	0.28	0.33	0.27
osteoporosis	0.013	0.018	0.025	0.019
cancer or a malignant tumour	0.015	0.033	0.061	0.037
Parkinson's disease	0.0018	0.0050	0.011	0.0061
any emotional, nervous or psychiatric problems	0.091	0.073	0.029	0.064
Alzheimer's disease	0	0.00034	0.0024	0.00092
dementia (serious memory impairment)	0.0046	0.0041	0.012	0.0067
eye problems:				
glaucoma or suspected glaucoma	0.024	0.043	0.096	0.055
diabetic eye disease	0.0085	0.013	0.014	0.012
macular degeneration	0.0069	0.0077	0.025	0.013
cataracts	0.027	0.062	0.20	0.096
incontinence	0.037	0.067	0.11	0.072
BMI	28.5	28.4	27.6	28.1
grip stength (measure of sarcopenia)	45.5	41.3	34.7	40.3
Number of observations	18980			

Figure B.1: Health care expenditure as share of GDP.



Note: Health care expenditure as share of GDP divided by government/compulsory schemes, household out-of-pocket payments, voluntary healthcare payment schemes. Black lines = UK. Grey lines = US. Source: OECD statistics.

C Health processes

C.1 Nonlinear health process

Following De Nardi, Fella and Paz-Pardo (2020), after estimating the health process using Arellano, Blundell and Bonhomme's procedure, we simulate many health histories and discretize the persistent and transitory components of health at each age in N dimensional grids. We then compute the transition matrices from t to $t+2$ for the persistent component (recall that our health data is biennial).[27]

To reconcile this biennial health process with the annual time span in the life-cycle model, we assume that health remains constant in the period we do not observe, such that the transition matrix is the identity matrix from t_o to t_{o+1}, o being an odd number, and the matrix computed in the simulated data from t_{o+1} to t_{o+2}.[28]

[27]Because of inconsistencies in the measurements of BMI from wave 7 onwards, the health measure and health dynamics are estimated using the first six waves only. We also estimate the death probabilities using six waves as ELSA data are linked to national death registers up to wave 6.

[28]We experimented with splitting the transition by computing the matrix T_1 satisfying $T_2 = T_1 \times T_1$, where T_2 is the matrix resulting from our simulations. The procedure is computationally

Table C.2: Canonical health process parameter estimates

Random component		
ρ	0.953	(0.015)
σ_ν^2	0.082	(0.020)
σ_ϵ^2	0.138	(0.026)
σ_0^2	0.459	(0.048)

Note: Standard errors in parentheses.

C.2 Canonical health process

To estimate the canonical health process, we first obtain health residuals by regressing the health index on a set of demographics, which includes a third-order polynomial in age, year of birth, education, and an indicator for having a partner.

We assume that health residuals are the sum of a persistent and a transitory component, as in (3). The persistent component follows an AR(1) process, $\eta_t = \rho\eta_{t-1} + \nu_t^H$, with $\nu_t^H \sim N(0, \sigma_\nu^2)$. The transitory component ϵ_t is an iid shock $\sim N(0, \sigma_\epsilon^2)$.

The three parameters of the random component of the health process (σ_ϵ^2, σ_ν^2 and ρ) plus the initial variance at age 50 (σ_0^2) are identified by the variances and covariances of the health residuals h_{it}. The initial period variance ($t = 0$), the following periods variances ($t = 1, \ldots, T$) and the lag ℓ covariances are equal to

$$
\begin{aligned}
Var(h_{i0}) &= \sigma_0^2 + \sigma_\epsilon^2 \\
Var(h_{it}) &= \rho^{2t}\sigma_0^2 + \frac{1-\rho^{2t}}{1+\rho^2}\sigma_\nu^2 + \sigma_\epsilon^2 \\
E(h_{it}h_{it-\ell}) &= \rho^\ell \left(\rho^{2(t-\ell)}\sigma_0^2 + \frac{1-\rho^{2(t-\ell)}}{1+\rho^2}\sigma_\nu^2 \right)
\end{aligned}
$$

Identification requires at least three periods of data. Note that, given the biennial nature of ELSA data we consider lags ℓ that are multiple of 2 up to lag 8. Table C.2 reports the estimates.

intensive and does not always produce a real-valued solution.

D Survival process

To compute mortality rates we discretize health in four quantiles defined by the 20, 30, and 50th percentile cutoffs. We assume that mortality risks perceived by the individuals are consistent with the life tables, and rescale estimated mortality in each health-age group in order to match the life tables' mortality rates. In this way, we use the hetogeneity by health in mortality obtained from the data, but anchor the aggregate mortality to match the cross-sectional life tables.

ELSA data are linked to administrative death records which allow to know the exact year of death of any individual (including attriters) up until February 2013. Therefore, we can estimate yearly death probabilities. The steps followed are reported below.

1. We estimate the probability of being of health level i ($\hat{Pr}(H_t = i)$) and of dying by $t+1$ conditional on health level i ($\hat{Pr}(death^D_{t+1}|H_t = i)$) using all observations for male respondents. To control for cohort effects, we estimate these probabilities using fixed-effect regressions. When we predict from the estimated regressions, we set the fixed effect equal to the average fixed effect for those born between 1948 and 1952;

2. the probability of dying by $t+1$ at each age t is given by:

$$\hat{Pr}(death^D_{t+1}) = \sum_{i=1}^{4} \hat{Pr}(H_t = i) * \hat{Pr}(death^D_{t+1}|H_t = i); \tag{D.3}$$

3. we compare the estimated probability with the life tables for each age t:

$$\frac{\hat{Pr}(death^{LT}_{t+1})}{\hat{Pr}(death^D_{t+1})} = \alpha_t$$

4. we rescale each conditional probability in such a way that the unconditional probability matches the life tables:

$$\hat{Pr}(death^{LT}_{t+1}) = \sum_{i=1}^{4} \hat{Pr}(H_t = i) * \hat{Pr}(death^C_{t+1}|H_t = i)$$

with $\hat{Pr}(death^C_{t+1}|H_t = i) = \alpha_t * \hat{Pr}(death^D_{t+1}|H_t = i)$.

The procedure adopted is the same proposed by Dal Bianco (2023), see External Appendix B.3 of that paper for a detailed description of the derivation.

E Earnings process

The earnings process has a deterministic component, $\omega_e(h_t, t)$, which depends on a second order polynomial in health and a second order polynomial in age. We additionally control for being in a couple, level of education and cohort. We use both singles and indiviuals having a partner to increase the sample size. In the model solution and simulation, we fixed the deterministic component to be representative of respondents born between 1948 and 1952, having a partner, and who left school between compulsory school leaving age and age 18.

The composite error term ψ_t is the sum of a persistent (ϑ_t) and a transitory (υ_t) component. Persistence in wages is captured by an AR(1) process ϑ_t.

$$\begin{aligned} \log e_t = \; & \omega_e(h, age_t) + \psi_t \\ & \psi_t = \vartheta_t + \upsilon_t \\ & \vartheta_t = \rho_e \vartheta_{t-1} + \nu_t^e, \quad \nu_t^e \sim N(0, \sigma^2_{\nu^e}). \end{aligned} \tag{E.4}$$

We assume that at time $t-1$ the individual knows ϑ_{t-1}, but he only knows the distribution of the innovations ν_t^e. We further assume that υ_t captures measurement error only.

Table E.3 reports the estimated parameters of the earnings process stochastic component. To identify the parameters we use the same moments used to estimate the canonical health process (see External Appendix C).

Table E.3: Earnings process parameter estimates

Random component		
ρ_e	0.897	(0.053)
$\sigma^2_{\nu^e}$	0.035	(0.021)
σ^2_{υ}	0.226	(0.031)
σ^2_0	0.152	(0.040)

F Moment profiles

Wealth profile

Our measure of wealth includes both housing and non-housing wealth. Blundell et al. (2016) report real house prices in England from 2002 to 2013 and document a 40% increase between 2002 and 2004, the first two waves of ELSA. We assume that the house price increase and the resulting wealth increase for homeowners do not affect individual decisions in terms of consumption, retirement, and labor market participation. Therefore, we strip out house price changes by dividing net primary housing wealth by the house price index, using as reference year 2004, and we assume a price increase equal to the real rate of return on other financial assets. The corrected net primary housing wealth is added up to net non-housing wealth and used to estimate the wealth profile.

To correct for cohort effects, we regress wealth a_{it}, on an individual specific effect f_i, a polynomial in age and unemployment rate U_t, proxying for aggregate time effects.

$$a_{it} = f_i + \sum_{n=1}^{S} \pi_n age_{it}^n + \pi_U U_t + u_{it} \tag{F.5}$$

This specification allows the estimation of age parameters accounting for individual fixed effects and time effects.

The estimated fixed effects $\hat{f}_i$ are regressed on a set of ten-year cohort dummies, this allows to compute the conditional expectation of $\hat{f}_i$ for a specific cohort of individuals, $E[\hat{f}_i|cohort = c]$. We then simulate from the estimated model fixing unemployment rate at 4.9% and the individual fixed effect with the average fixed effect for the cohort of interest. Specifically, we replace f_i with $\tilde{f}_i = \hat{f}_i - E[\hat{f}_i|cohort_i] + E[\hat{f}_i|cohort = c]$. The reference cohort c includes individuals born between 1948 and 1952.

Hours and participation by health profiles

Equations similar to F.5 are used to derive hours and participations profiles net of cohort effects, and in particular representative of the cohort 1948-1952.

G Tax function

The tax function reproduces the one that applied in 2003/04 in the UK. The tax unit in the UK system is the individual. The income tax schedule is based on three bands. The tax base includes earnings, pensions and interest income net of personal tax-free allowances. The main tax allowances are listed in Table G.5.

For those aged less than SPA, National Insurance payments are levied on earnings between a lower limit (£4,628) and the upper earnings limit (UEL £30,940) at a rate of 11%. Those having gross earning below the lower limit do not pay social insurance contributions, whereas those with earnings above UEL are subject to a rate of 1%.

Table G.4: Income tax schedule

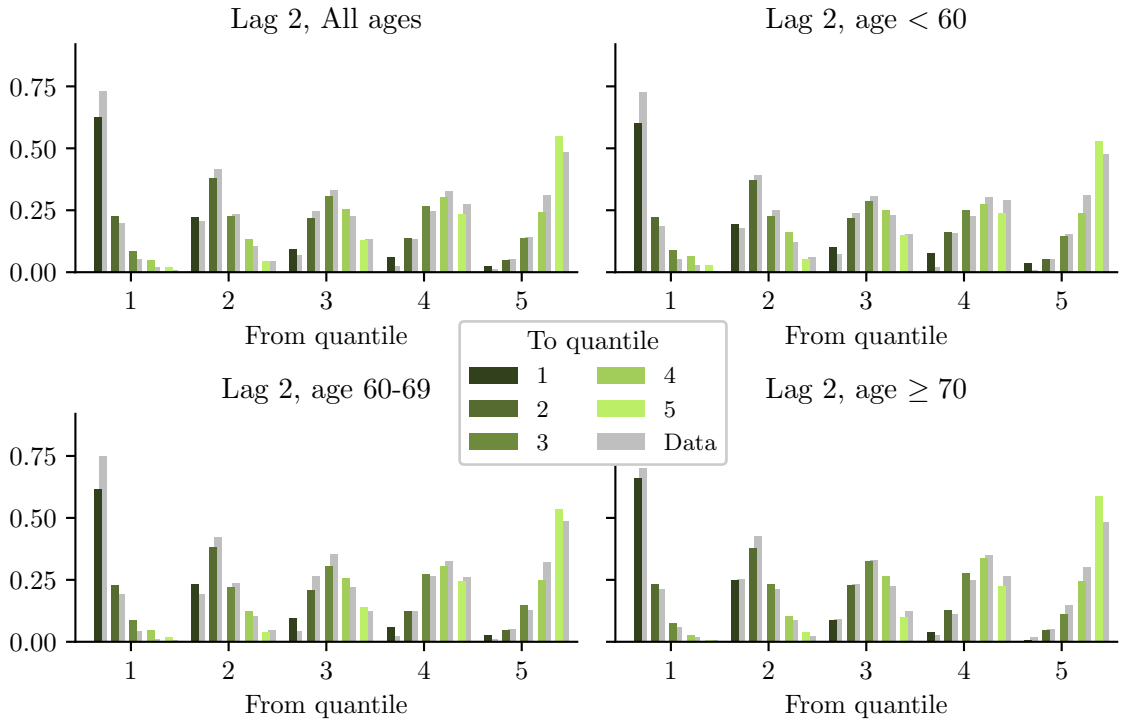
Band	Rate on earned income	Rate on investment income
0-1960	0.1	0.2
1961-30500	0.22	0.2
30501-	0.4	0.4

Table G.5: Personal tax allowances and credits

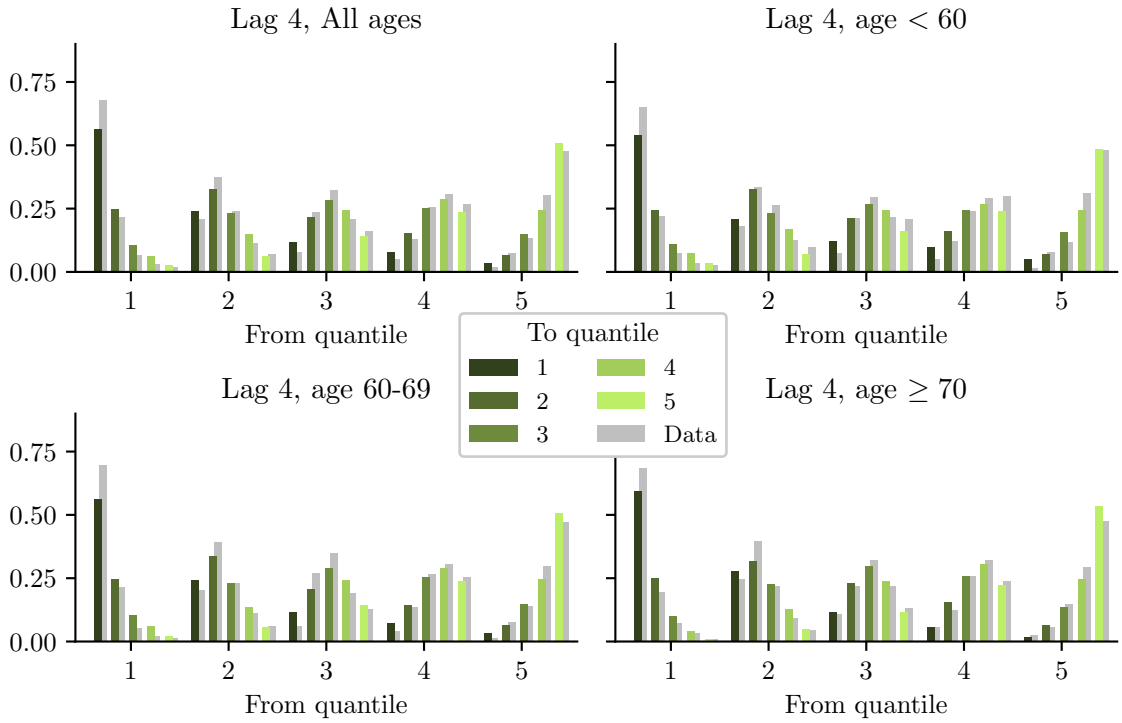
Allowance/credit	Amount per year (£)
Single personal allowance: all individuals	£4,615
Age allowance: Age 65-74	£6,610 reduced to £4,615 (50% of income over £18,300)
Age allowance: Age 75+	£6,720 reduced to £4,615 (50% of income over £18,300)
Married Couples age allowance: Age 65-74	£5,565 reduced to £0 (50% of income over £18,300, less any reduction to personal age allowance)
Married Couples age allowance: Age 75+	£5,635 reduced to £0 (50% of income over £18,300, less any reduction to personal age allowance)

Figure H.3: Health transition probabilities

(a) 2 years lag



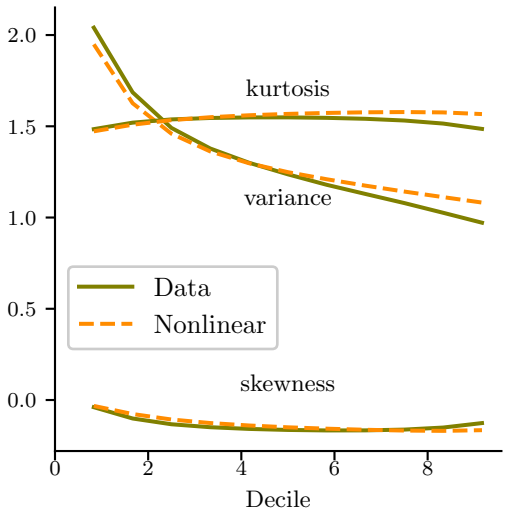
(b) 4 years lag



*Note:*Probabilities of health transitioning by quantile of initial health and quantile of health after n years, simulation (shaded green histograms) and data (grey histograms. Panel (a): 2-year lags; Panel(b): 4-year lags)

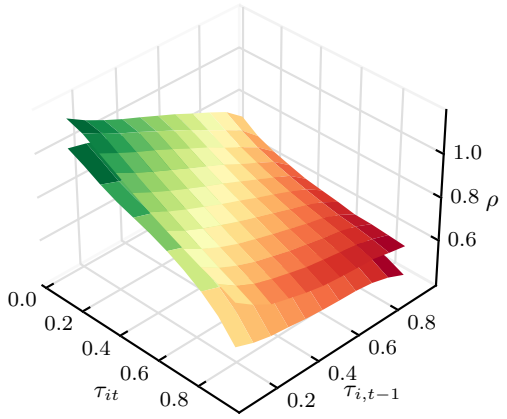
H Additional results

Figure H.2: Fit of conditional dispersion, skewness and kurtosis.



Note: Conditional dispersion $\sigma(h,\tau) = Q(\tau|h_{i,t-1}=h) - Q(1-\tau|h_{i,t-1}=h)$, for $\tau = 11/12$. Conditional skewness $sk(h,\tau) = \frac{Q_t(h_{i,t-1},\tau)+Q_t(h_{i,t-1},1-\tau)-2Q_t(h_{i,t-1},1/2)}{Q_t(h_{i,t-1},\tau)-Q_t(h_{i,t-1},1-\tau)}$, for $\tau \in (1/2,1)$. Conditional kurtosis, for some $\alpha < 1-\tau$, $kur_t(h_{i,t-1},\tau,\alpha) = \frac{Q_t(h_{i,t-1},1-\alpha)-Q_t(h_{i,t-1},\alpha)}{Q_t(h_{i,t-1},\tau)-Q_t(h_{i,t-1},1-\tau)}$.

Figure H.4: Simulated persistence confidence intervals



Note: Nonparametric bootstrapped (500 replications) 95% confidence intervals of nonlinear persistence by quantile of previous health and quantile of current shock

Figure H.5: Earnings, model fit

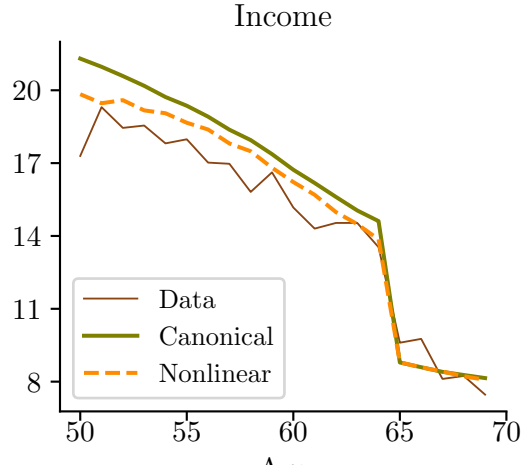
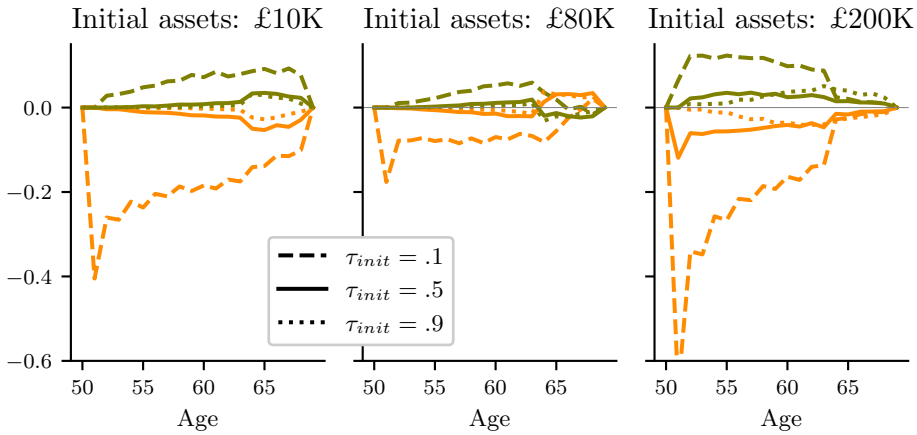
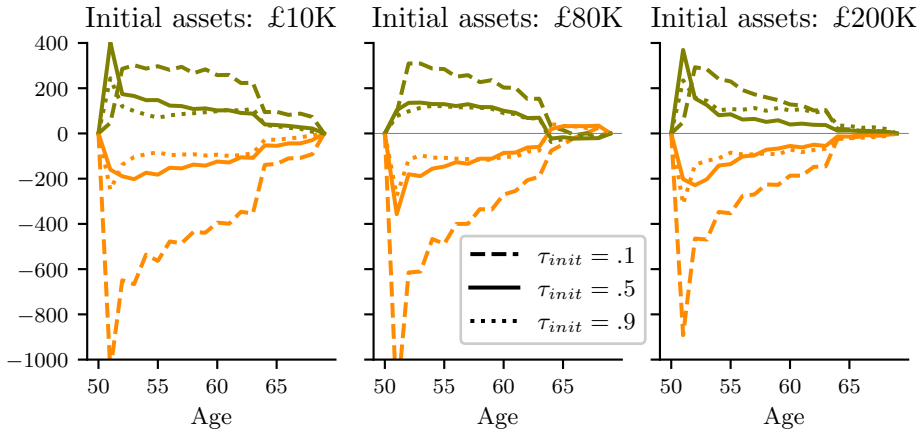


Figure H.6: Effects of health shocks, from different wealth levels

(a) Labor force participation



(b) Hours worked



Note: The figures illustrate, for different values of initial assets in the nonlinear model of health dynamics, the age profiles of the difference in labor force participation and hours worked between individuals subject to a permanent component of health shock τ_{shock} and individuals subject to $\tau_{shock} = 0.5$, starting from different initial levels of the permanent component (τ_{init}). Averages over 5,000 simulated histories of individuals starting at age 50 with the same level of assets indicated at the top of each panel.

.

Table H.6: The effect of health and its components

(a) Health: 10th percentile

Effects removed	Assets	Earnings	Empl.	Hours	CEV
All (relative to baseline)	-21.1%	-8.2%	-16.5%	-22.0%	–
Decomposition:					
Mortality	-1.7%	-0.2%	+0.1%	+1.0%	–
Time cost	-17.0	-6.5	-15.1	-21.3	-9.37
Wages	-3.8	-1.2	-0.9	-0.8	-1.35
Time+Wages	-20.5	-8.0	-17.5	-22.3	-10.61
Mortality+Time	-17.5	-6.6	-14.2	-20.6	–
Mortality+Wages	-5.4	-1.4	-0.9	+0.2	–

(b) Health: 50th percentile

Effects removed	Assets	Earnings	Empl.	Hours	CEV
All (relative to baseline)	+1.8%	+1.2%	+4.9%	-3.3%	–
Decomposition:					
Mortality	-0.7%	+0.1%	-0.4%	-0.3%	–
Time cost	+1.1	+0.9	+4.8	-3.0	-2.57
Wages	-0.1	+0.3	+1.2	-0.5	-0.50
Time+Wages	+1.7	+1.0	+5.0	-2.8	-3.05
Mortality+Time	+1.0	+1.1	+4.8	-3.4	–
Mortality+Wages	-0.7	+0.4	+0.8	-0.8	–

Note: Each panel reports percent changes from the baseline after assigning everyone either the mortality rate, time cost of health, or wage offer computed at the indicated percentile of the age-specific health distribution. Assets measured in £1000 averaged throughout the life cycle. Earnings are the log of cumulated earnings at 65. Employment rate and hours averaged from age 50 to 69. Hours are annual, conditional on employment.

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