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, a pattern that cannot be accounted for by canonical models. We incorporate this health measure 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 persistent adverse economic effects that are more dramatic for poor individuals starting in bad health. Bad health shocks also increase the dispersion of asset accumulation within this category of individuals. A canonical model of health dynamics would not uncover these effects.

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

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1 Introduction

Are severe health shocks more persistent than milder shocks? If so, what are their effects on individuals' economic outcomes? To answer these questions, we introduce a complex measure of health and estimate its persistency using a non-parametric model with nonlinear dynamics. We incorporate this health measure into a life-cycle model of consumption, savings, and labor supply decisions to simulate the effects of various health shocks.

Several articles have studied the effects of health on life-cycle economic outcomes, helping our understanding of the complex interactions between health and economics. However, most of these studies posit a binary or 3-valued health measure evolving according to a first-order Markov process. Our analysis focuses on assessing the effects of especially severe and persistent health shocks, requiring a more detailed measure of health.[1]

We derive a continuous measure of health from several objective health indicators using a latent variable model in the spirit of Bound (1991).[2] We assume that this health measure evolves according to a complex, nonlinear dynamic process with a persistent and a transitory component. Drawing from Arellano et al. (2017), we assume the latter component evolves according to a non-parametric process with nonlinear persistence and age dependence. The persistence patterns resulting from this flexible approach could not be accounted for by a "canonical" first-order Markov process.

We incorporate this health measure into a standard life-cycle model where agents receive utility from consumption, leisure, and bequest of assets; they choose, every period, labor market participation, hours worked, and savings facing uncertainty over health, wages, and life expectancy. Health affects wages and life expectancy. Following the literature, health affects the time constaint, 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) panel of English households aged 50 and above. Focusing on English data allows us to simplify our analysis by excluding medical expenditure choices in the model (health care in the U.K. is publicly provided). We perform the estimation in two steps. First, we estimate the parameters of the health, wage, and life expectancy processes, which we assume

[1] See, for example, French (2005), De Nardi et al. (2010) Capatina (2015), Low and Pistaferri (2015), Ameriks et al. (2020), Blundell et al. (2021). An exception is De Nardi et al. (2017), modeling health with a second-order Markov process with age-dependent parameters, showing that health follows complex patterns of duration-dependence. See also the surveys De Nardi et al. (2016) and French and Jones (2017).

[2] See also Dal Bianco (2022). Hosseini et al. (2021a) develop a *frailty index as the cumulative sum of all adverse health indicators* to compare its dynamics with that of a self-reported health indicator, but do not incorporate it into a nonlinear process.

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 estimates of the nonlinear health process reveal that health indeed follows a complex, nonlinear dynamics. In particular, negative shocks are worse and much more persistent than good shocks when starting in bad health. When individuals start in good health instead, good shocks are only slightly more persistent. These asymmetries cannot be replicated with the canonical process by construction. They are also difficult to ascertain when health is a binary variable, or when it assumes few discrete values because they depend on the arbitrary health cutoffs used implicitly to discretize the health measure.

The moments we match to estimate the model are the age profiles of accumulated assets, of labor force participation by health quantiles, and of hours worked. Our estimates of the life-cycle model match these moments very well both when using the nonlinear model of health dynamics and when using the canonical model.

Using the estimated life-cycle model, we simulate counterfactual health shocks to understand the role and distributional consequences of bad health and its persistence. Specifically, we simulate individuals who start at the age of 51 with different levels of the persistent component of the health process (the 10th, 50th, and 90th percentile of its distribution). We subject the persistent component of health to a good, an intermediate, and a bad shock at the age of 52 (respectively, shocks that bring the persistent component of health to the 10th, 50th, and 90th percentile of its distribution).[3]

We find that the health asymmetries following different shocks in the nonlinear model produce considerable economic differences. Bad health shocks are worse and have more lasting effects when people start poor and in bad health than otherwise. For example, we find that for people 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 age 85 of £35,000 less than after a median shock. Starting from the median of the persistent component of health, a shock of similar magnitude would induce an accumulation of only £14,000 less than after a median shock. Starting from £80,000 in assets, the same difference reduces to no more than £6,000 and depends little on the initial level of health. We also find that the canonical model does not predict these large differences between shocks for poor people.

Using the estimated value function, we compute that the willingness to pay to avoid a bad shock is about £34,000 for people starting with £10,000 in assets at the

[3] Arellano et al. (2017) proposes a similar exercise when studying income dynamics. Health is reported only every two years in ELSA data, therefore we cannot impose the shock at the age of 50, the beginning of the age period we consider in our model.

age of 51, and does not depend much on the level of initial wealth: relative to their wealth, a bad shock is much costlier for the poor.

We find that bad shocks generate higher dispersion in accumulated assets among people that 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 the separate components of the model affected by health (wages, time endowment, and mortality) reveals that the effects of the three components are not additive (nonlinearities are important) and that the adverse effects of bad health are primarily due to its time cost.

Our results highlight the importance of accounting for a complex health measure, particularly when focusing on outcome inequality. We contribute to the literature first by proposing a continuous measure of health and showing that its evolution follows a complex, nonlinear process. Second, we show that using this measure of health, adverse shocks are costlier, especially for poorer individuals, and generate higher outcome dispersion.

The rest of the paper proceeds as follows. The next section describes our data. Section 3 describes the construction of our health measure and the estimation of the health process. Section 4 describes the life-cycle model and Section 5 its estimation. In Section 6 we simulate the effect of different health shocks on the level and the distribution of outcomes.

2 Data

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 health-related indicators covering the physical, mental, and cognitive health status of individuals.

We focus on male respondents. The final sample includes about 32,200 observations from 8,200 individuals. Table 1 reports 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 and wages decrease substantially with age, especially after the state pension age (65 for men in the period we consider). Total wealth increases with age as previously reported in the literature (see for example Blundell et al. (2016)).

Table 1: Descriptive statistics by age

	50-59	60-69	70-90
% working	79.5	40.7	6.8
annual hours worked	1926	1597	950
wages (£)	16775	12057	3576
wealth (1000 £)	128	169	193

Note: ELSA data, waves 1-7. 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 and is corrected for cohort effects (reference cohort 1946-1955 – see details in Appendix C).

3 Health measurement and dynamics

We construct a continuous index of health using a latent variable model.[4] True health ψ_{it} is a function of a set of objective health indicators Z_{it} and an error term ξ:

$$\psi_{it} = Z'_{it}\alpha + \xi_{it}$$

We observe self-reported health, h^b_{it}, a binary indicator equal to 1 if the individual declares to be in good, very good or excellent health, and 0 otherwise. We assume that h^b_{it} is determined by the latent variable h^*_{it}, equal to the sum of ψ_{it} and a reporting error μ_{it}:

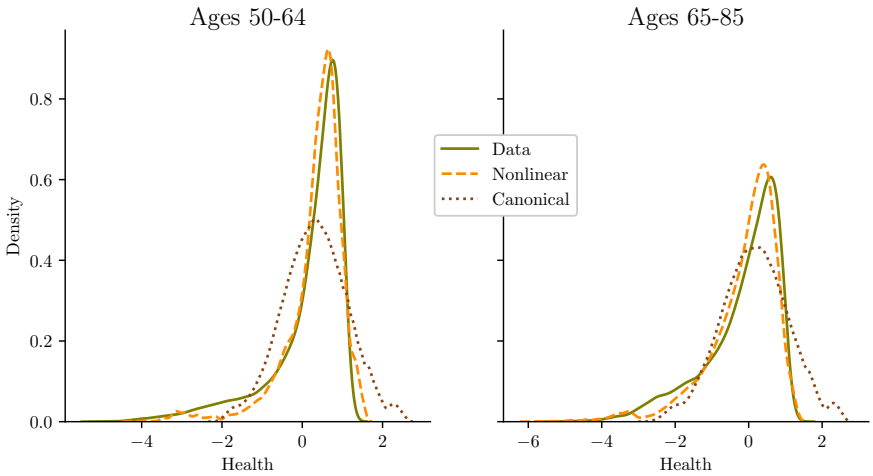
$$\begin{aligned} h^*_{it} &= \psi_{it} + \mu_{it} \\ &= Z'_{it}\alpha + (\xi_{it} + \mu_{it}) \\ &= Z'_{it}\alpha + u_{it} \end{aligned} \tag{1}$$

The individual reports $h^b_{it} = 1$ if $h^*_{it} > 0$. We also assume that ψ_{it} and μ_{it} are uncorrelated. The composite error term u_{it} reflects differences in reporting behavior across individuals. The health index is equal to the predicted value $h_{it} = \hat{h}^*_{it}$, according to (1). This procedure is analogous in spirit to using "objective" health information to instrument for the endogenous and error-ridden self-reported health measure (Bound et al. (1999)).

Z includes 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).

[4] See Bound et al. (1999), Bound (1991), Bound et al. (2010), J. S. Butler and Pincus (2010)

Figure 1: Health distribution: data versus model simulations



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

The solid green lines in Figure 1 illustrate that the distribution of the health index h is left-skewed.[5] Figure 2 illustrates the second and higher moments of the shock distribution conditional on previous health deciles and age.[6] The standard deviation (left panel) decreases as the previous level of health increases (from 0.8 to 0.3) and it increases with age, in particular, if previous health is above the median. Health shocks are negatively skewed (central panel), and the skewness decreases with the health level and is higher for older individuals. Finally, kurtosis (right panel) increases with the level of health and is higher for younger individuals. This evidence suggests that health is a highly nonlinear process with some age dependence and justifies the adoption of a flexible specification of the health process.

Following Arellano et al. (2017), we define the health process as the sum of a persistent and a transitory component:[7]

$$h_{it} = \eta_{it} + \epsilon_{it}, \quad i = 1, \ldots, N, \quad t = 1, \ldots, T \tag{2}$$

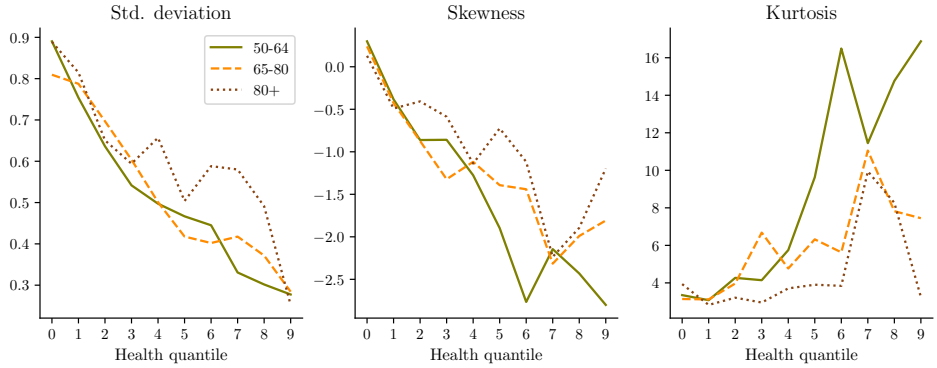
where η_{it} is the persistent component which follows a first-order Markov process, and ϵ_{it} is a transitory health shock with mean zero, independent of t and η. Note that from here on h_{it} denotes health residuals. We construct h_{it} as residuals from

[5]Its shape is consistent with the one obtained by Hosseini et al. (2021b) using as a health indicator the sum of adverse health indicators.

[6]Our health data is biennial, therefore previous health is 2 years earlier in these computations.

[7]Because our health data is biennial, for ease of notation in this section one time period is to be interpreted as two years. See Appendix A for a description of how we reconcile the biennial health process to the annual time span in the life-cycle model.

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



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. Define the τ-th quantile of η_{it} given $\eta_{i,t-1}$ as

$$\eta_{it} = Q_t(\eta_{i,t-1}, \tau) \text{ for } \tau \in (0,1). \tag{3}$$

The $\eta's$ evolve over time non-parametrically allowing for nonlinear persistence. A special case of (3) is $\eta_{it} = \rho\eta_{i,t-1} + \nu_{it}$, an AR(1) process.[8] We will refer to this case as the "canonical" model, whereas we call the more general case the "nonlinear" model.

A measure of persistence of η when hit by a shock u_{it} of rank q can be defined as:

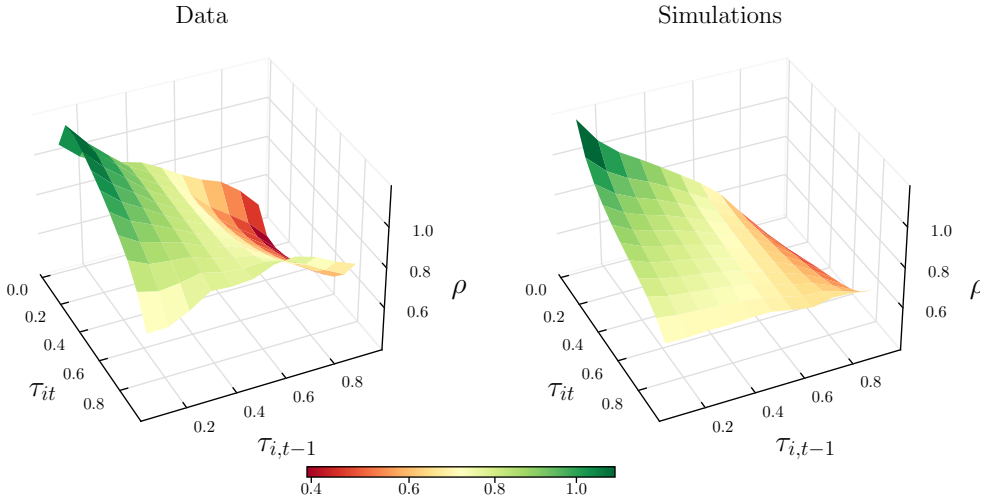
$$\rho_t(\eta_{i,t-1}, \tau) \equiv \frac{\partial Q_t(\eta_{i,t-1}, \tau)}{\partial \eta}. \tag{4}$$

In the canonical AR(1) model, ρ is constant, equal to the auto-regression parameter.

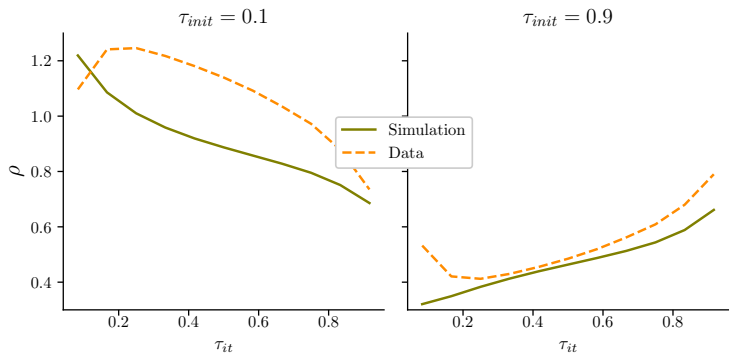
We use the quantile-based panel data method developed by Arellano et al. (2017) to estimate the nonlinear process. Figure 3 reports the average value of ρ_t as a function of current and previous health deciles, comparing values computed directly from the data with simulations from the model estimates. It shows that health persistence is high when both the current health shock and the previous health are low. Health persistence is instead low when the current health shock is high and previous health is low. In particular, in the left panel of Figure 3(b) we notice that for individuals with low health, bad shocks are much more persistent than high shocks. Instead, for individuals starting with high health (right panel), persistence is slightly increasing. The figure also shows that the nonlinearity of the health persistence is well reproduced by the model.

[8] see Blundell et al. (2021), Hosseini et al. (2021b) and Dal Bianco (2022).

Figure 3: Health's nonlinear persistence



(a) Average persistence, nonlinear model vs data



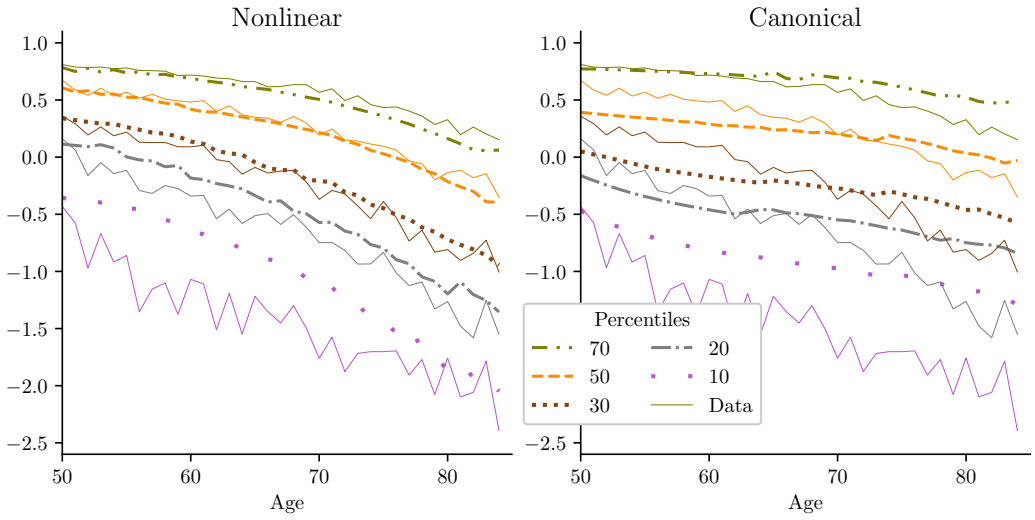
(b) Average persistence by initial decile of health

Estimates of the average derivative of the quantile function (4), computed directly from data, and from simulations of the nonlinear model estimates (adding the predictions of the persistent and transitory component). Panels 3(b) illustrate sections of figures in panels 3(a) taken at the 1th and 10th deciles of τ_{init}.

As a term of comparison, we estimate the canonical model. Figure 1 shows that while the nonlinear model reproduces the health index distribution over time quite well, unlike the canonical model.

Figure 4 illustrates the fit of the two models with respect to the health index at different percentiles of the health distribution over the life cycle. The canonical model understates health for younger individuals and overstates it for older individuals. The nonlinear model performs significantly better over the entire life cycle and at different quantiles of the health distribution.

Figure 4: Health by percentile



Note: Health at different quantiles of the health distribution indicated in the legend. The thin solid lines represent values of the data index computed according to (1); the other lines represent averages from 15,000 simulated histories

4 The life-cycle model

Because of data limitations, we model the behavior of males who enter the model at age 50 ($t = 1$), may work until 70 but start receiving pension income at 65 (the state pension age in the UK in the period covered by our data). 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, $b(a_t)$, with a_t denoting assets at time t.

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

$$U(c_t, l_t) = \frac{1}{1-\nu}(c_t^{\gamma} l_t^{1-\gamma})^{1-\nu} \tag{5}$$

where ν is the relative risk aversion parameter and γ the consumption weight.

Upon death, the agent also receives utility from bequests a according to:

$$b(a) = \phi_B \frac{(a+K)^{(1-\nu)\gamma}}{1-\nu}. \tag{6}$$

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 E for details). The time constraint is:

$$l_t = L - s_t - \phi_h(h_t) \tag{7}$$

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.[9] Health uncertainty affects utility through leisure, the wage offer (see below), and the survival probability.

The earnings process has a deterministic component, $\omega_e(h_t, t)$, which depends on health and age. 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.

$$\begin{aligned} \log e_t = \; & \omega_e(h, age_t) + \psi_t \\ & \psi_t = \vartheta_t + \upsilon_t \\ & \vartheta_t = \rho_e \vartheta_{t-1} + \nu_t^e, \quad \nu_t^e \sim N(0, \sigma^2_{\nu^e}). \end{aligned} \tag{8}$$

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.

The budget constraint is therefore

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

Where hours worked s_t are constrained to be zero after the age of 70, tax are taxes, r is the exogenous interest rate on accumulated assets and tr_t are transfers received in such a way that consumption c_t is greater or equal to the consumption floor $\underline{c}$, plus, starting from age 65, income from pension wealth p_t, annuitized at rate r_p. Finally, $\phi_w(\cdot)$ denotes a monetary cost of working.[10] Each agent is endowed with initial pension wealth p, to which he contributes a fraction c_p of his annual earnings.

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, $\pi^{t+1} = \pi(h_t, t)$. We set the terminal age $T = 85$. Time is discounted at factor β. With these assumptions, preferences can be expressed using a recursive formulation with state-space be $X_t = \{a_t, e_t, h_t, p_t\}$, which we use to compute the numeric solution to the optimization problem. The expression for the Bellman equation is in Appendix D.

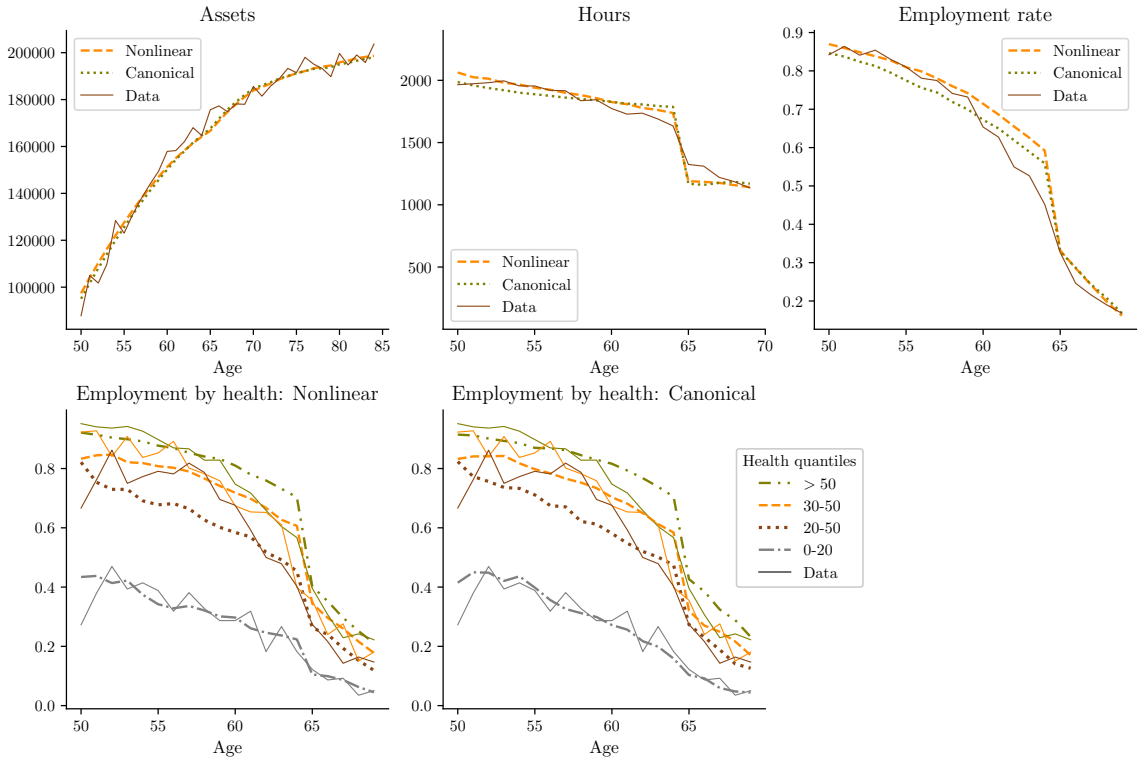
5 Estimation

We estimate the life-cycle model with both the nonlinear health dynamics and, for comparison, the canonical model dynamics. We adopt a Simulated Method of Mo-

[9] De Nardi et al. (2020) use this procedure to incorporate the estimates of a nonlinear model of *earnings* dynamics into a life-cycle model. Additional details are in Appendix A.

[10] 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.

Figure 5: Estimation fit

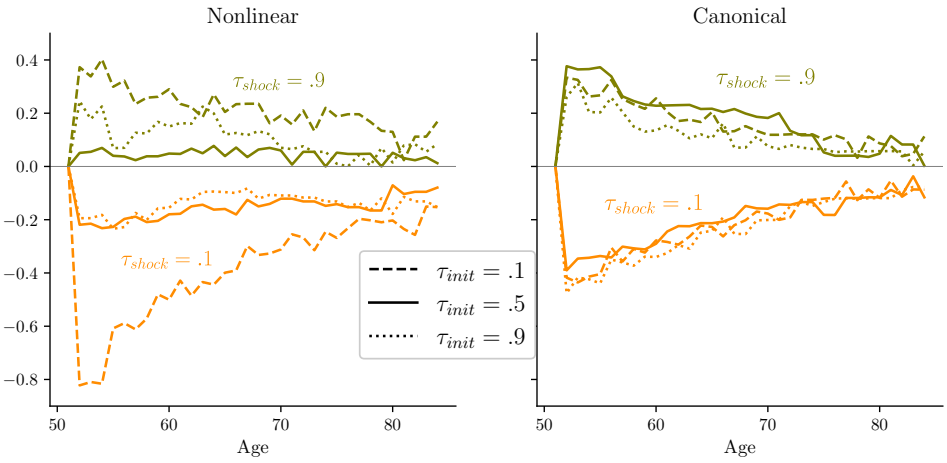


Note: Age profiles of average assets, employment rate, and average hours worked conditional on being employed at the estimated parameters. Simulations of 15,000 histories, nonlinear and canonical models. The thin solid lines represent the age profiles in the data.

ments, matching the age profiles of average assets, hours worked, and participation by quantile of the health distribution (using thresholds at percentiles 20, 30, and 50). The estimated parameters are in Appendix Table E.3.

Both the nonlinear and the canonical models match the life-cycle profiles of assets, participation, and hours worked well (see figures 5). The fit of the two models is very similar, despite the models reproducing the health patterns differently, as shown in Section 3. In particular, the model fits participation by health for people in low health very well. Our parsimonious specification, a linear function of the cost of work by age, cannot fully account for the steep decline in participation between ages 60 and 65. In simulations using different parameters, we found that parameters matching a faster decline for people in good health would generate very low employment for people in bad health. A psychic benefit from retiring starting at age 60 (justified by legislation allowing the withdrawal of private pension funds) may help improve matching these moments. Since the model performs fairly well overall, we chose not to add ad-hoc parameters to this purpose.

Figure 6: Health after different shocks



Note: 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 5,000 simulated histories.

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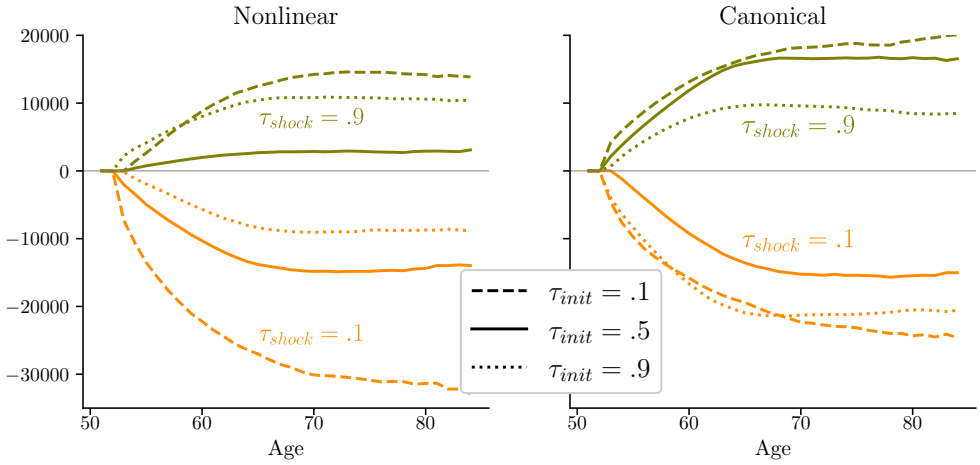
6 The effects of health shocks

To assess the economic effect of a health shock and evaluate the impact of the nonlinear health dynamics, we simulate histories of 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 three different shocks at age 52: a bad, medium, and good shock that bring them, respectively to the 10th 50th, and 90th percentiles of the health distribution conditional on τ_{init}. The resulting health dynamics of the good and bad shock, relative to the medium shock, are shown in Figure 6.

By construction, the canonical model results in symmetric patterns of health after the good and bad shock, identical in magnitude with respect to τ_{init}. The discrepancies from this theoretical prediction in the right panel of Figure 6 (the three lines of each shock level should overlap) are due to approximation errors from the discretization of the health variable.

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 6 shows that a bad shock is worse and more persistent, relative to a median shock, when starting in bad health than when starting with medium or high health. Good shocks lead to higher levels of health when starting in bad health (relative to a median shock), but their magnitude is

Figure 7: Assets after different health shocks



Note: The figures illustrate the 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 5,000 simulated histories of individuals starting at age 51 with £10,000 in assets.

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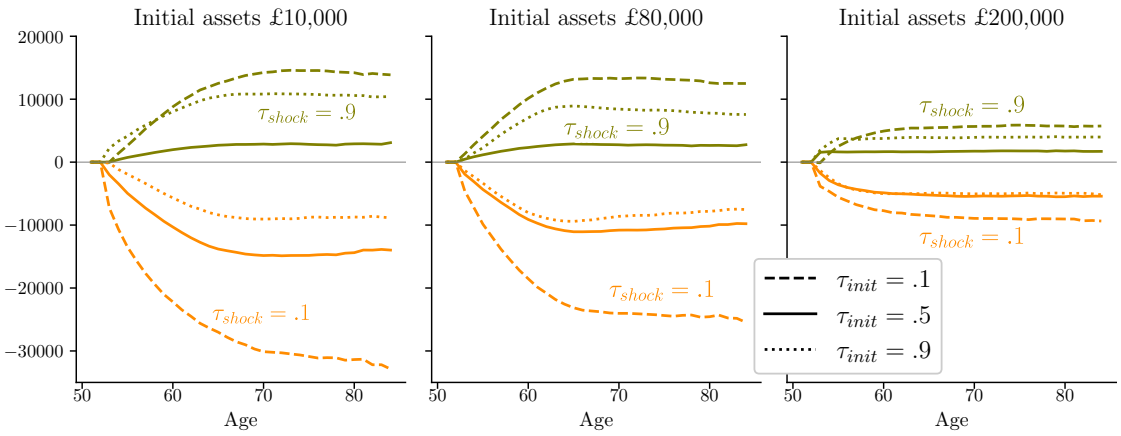
quantitatively smaller, in absolute value, than bad shocks.

6.1 The nonlinear and canonical models produce different economic effects

These asymmetries have economic effects, as shown in Figure 7. When people start relatively poor, with £10,000 in assets at age 50, a bad shock induces an accumulation at the age of 85 of nearly £33,000 less than after a median shock. The difference in accumulation after a bad shock relative to a median shock is 43% this amount when starting at higher levels of health: £14,000 when $\tau_{init} = .5$ and less than £9,000 when $\tau_{init} = .9$ The gains after a good shock are smaller in magnitude, amounting at about £14,000, £3,000, and £10,000 respectively starting from $\tau_{init} = .1, .5$, and .9.

The right panel of Figure 7 illustrates that the gains and losses from good and bad shocks are smaller and more symmetric in the canonical model. The gains at age 85 range from £8,000 to £20,000 and the losses from £15,000 to £24,000. Even though the effect of shocks on health is theoretically identical across values of initial health, as illustrated in Figure 6, the effects of these shocks on asset accumulation are not the same because of the nonlinearities in the life-cycle model.

Figure 8: Assets after health shocks, starting from different wealth levels



Note: The figures illustrate, for different values of initial assets in the canonical model of health dynamics, the age profiles of the difference in assets 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.

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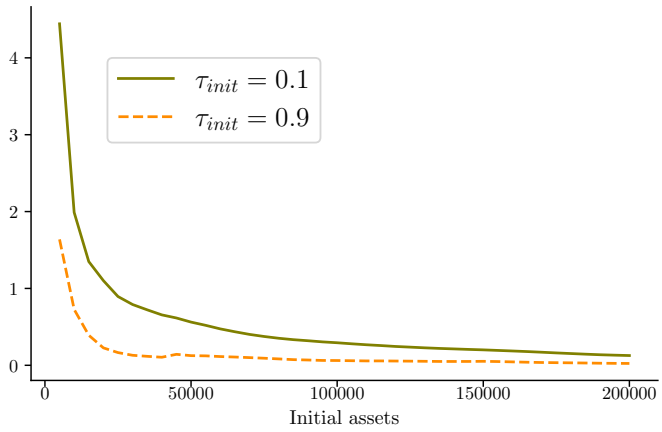
6.2 Bad health shocks are relatively worse for the poor

Focusing on the nonlinear process, we found that the economic effects of bad shocks are much stronger in absolute terms when people start off poor, as illustrated in Figure 8. The left panel reproduces the left panel of Figure 7; the other panels report the simulations of individuals subject to the same health shocks, but with assets equal to £80,000 (middle panel), and £200,000 (right panel) at age 50. While the patterns of asset accumulation depend on the value of initial health across wealth levels, they are smaller in magnitude when people start with higher wealth. Patterns for participation and hours worked are illustrated in Appendix figures F.1(a) and F.1(b).

The more damaging effect of a bad health shock suffered by poorer individuals is better measured by the willingness to pay to avoid the health shock. We computed what wealth transfer would make an individual indifferent between being subject to a bad shock ($\tau_shock = 0.1$). For someone starting in relatively bad health ($\tau_{init} = .1$) and £10,000 in assets, such value at age 50 is £20,000, and increases slightly for wealthier individuals, reaching £30,000 for individuals starting with over £115,000 in assets. The same figure is never above £8,200 for people starting in good health. Figure 9 illustrates these figures as a fraction of initial assets, highlighting the importance of health shocks for poorer individuals, and people in bad health.

Figure 9: Willingness to pay to avoid a bad shock (fraction of initial assets)



The figure reports the asset transfer, as a fraction of initial assets, that would make a person indifferent between being subject to $\tau_{shock} = .1$ at age 52, starting from different level of the permanent component of health τ_{init}, and receiving the transfer, or not being subject to the shock.

6.3 The sources of health effects

In the model, health affects economic outcomes through three channels: mortality, the availability of time for leisure and work, and the wage process. To ascertain the relative importance of these channels we turn them off by assigning to all individuals the mortality rate, the cost of bad health, and wages equal to the values these variables assume at a given quantile of the health distribution, conditional on age.

Changing the effects of health for a majority of individuals generates both income effects (higher income and time available), and substitution effects (higher hourly wages), as well as dynamic effects due to changes in mortality and uncertainty about future health. Obviously, removing the effect of health has very different effects on economic outcomes depending on the reference health level, as reported in Table 2.[11] When the effects of health through all three channels are removed, average assets are 8 percent higher if health were equal for everyone to the 75th percentile of its age-conditional distribution, but 10 percent lower if health were set at the 25th percentile. Employment, hours, and income increase as well in the first scenario, and decrease in the second.

The decomposition of these effects reveals that when people are assigned health at the 75th percentile, Mortality alone accounts for 19% of the +8% total effect on assets at the time of death.[12] This effect is driven by the need to finance a

[11] We report additional tables with decompositions of health levels set at the 25th, 50th and 90th percentile in Appendix F.

[12] 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. The main differences

Table 2: Decomposition of the effects of health on outcomes

Effects removed	Assets	Income	Empl.	Hours
None (baseline)	158	11.2	0.65	1848
(a) Counterfactual health: 75th percentile				
All (relative to baseline)	+8.0%	+11.1%	+10.7%	+6.3%
Decomposition:				
Mortality	+0.9%	-0.3%	-0.2%	+0.2%
Time cost	+4.6	+8.2	+8.4	+7.0
Wages	+1.4	+2.3	+3.1	-1.2
Mortality+Time	+4.7	+7.1	+8.2	+6.3
Mortality+Wages	+2.4	+2.0	+2.9	-1.1
Time+Wages	+7.6	+12.2	+10.7	+7.0
(b) Counterfactual health: 50th percentile				
All (relative to baseline)	-2.6%	-1.9%	+4.2%	-5.8%
(c) Counterfactual health: 25th percentile				
All (relative to baseline)	-10.3%	-9.9%	-1.0%	-11.2%

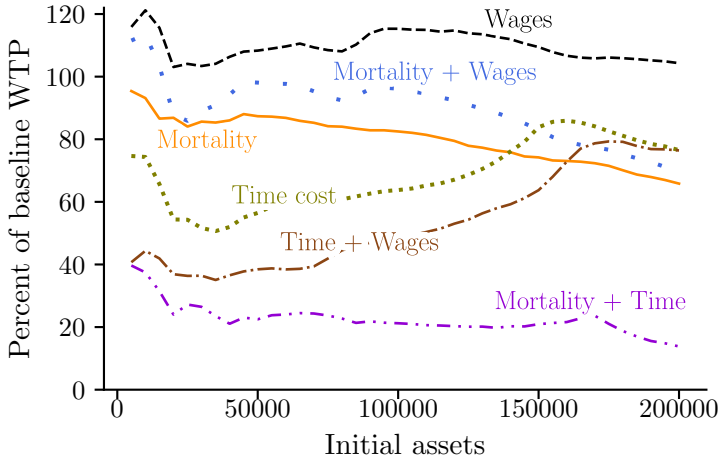
Note: The table reports average outcomes in the baseline model simulations (first row), and percent changes from the baseline after assigning everyone either the mortality rate, time cost of health, or wage offer computed at the percentile of the age-specific health distribution indicated at the top of each panel. Assets and (disposable) Income in thousand pounds averaged throughout the life cycle. Employment rate averaged from age 50 to 69. Hours are annual, conditional on employment.

longer expected lifetime. The time cost is the factor contributing the most to all outcomes. When health increases, the reduced time cost shifts the static budget constraint outwards, inducing an increase in leisure, hours worked, and participation, making it less costly to save. The income effect induced by higher wages is different from the one induced by lower time cost because time available does not change. As a consequence hours worked actually slightly decrease (the substitution effect dominates), and higher income and employment generate a more moderate effect on accumulated assets.

The table also reveals that the effect of the three components is not additive, a result of the model's nonlinearities. The effect on assets of mortality and time cost combined is less than the sum of the two effects: the increase in assets induced by the reduced time cost is enough to fund the increased lifespan. When mortality does not

between her model to ours are: (1) health takes only three values, that evolve with a Markov process without a transitory component; (2) the model is estimated using data from the U.S., (3) modeling the U.S. market, personal health expenditures are included as a choice variable.

Figure 10: Components of willingness to pay to avoid a bad health shock



Note: The figure reports the willingness to pay (as a percentage of the baseline willingness to pay) to avoid a bad health shock $\tau_{shock} = 0.1$ at age 52, for individuals starting from $\tau_{init} = 0.1$ with assets in the horizontal axis, after removing the effect of health on mortality, time available, wages, and their combinations. Health is removed by computing the value of the corresponding variable(s) at the 75th percentile of the health distribution conditional on age

change, instead, the effect of time cost and wages combined is *more* than the sum of the single effects when health increases, when health is set at the 75th percentile.

We perform the same decomposition on the willingness to pay to avoid a bad health shock. In Figure 10 we report the willingness to pay for a bad shock (as a percent of the baseline willingness to pay) after removing the effect of health on each channel, or their combination. To remove the effect of health, we assign individuals the value each variable would take at the 75th percentile of health (conditional on age) as in panel (a) of Table 2. We consider a health shock $\tau_{shock} = .1$ at age 52, starting from $\tau_{init} = 0.1$.

Interestingly, the results depend on the value of the initial assets of the individual. The time cost of bad health is again the most important factor for most initial asset levels, by itself accounting for between 50 and 80% of the baseline willingness to pay. The effect of health on mortality has a small impact only for the poorest individuals; for the highest wealth individuals, removing this effect would decrease the willingness to pay by over 30%. This is because bequest motives become more important at a higher level of wealth, and are negligible at low wealth. The effects of health on mortality and time endowment combined account for about 80% of the baseline willingness to pay to avoid the shock throughout most of the initial assets range we considered. Removing the effect of health on wages increases the willingness to pay to avoid a bad shock, as indicated by the dashed line being above 100%. This is because in this experiment we are assigning a majority of individuals a higher

hourly wage than under the baseline (equal to the hourly wage offered to those at the 75th percentile of health). Following a bad shock participation and hours worked decline, hence compared to the baseline, a bad shock results in a higher income loss. The effect of wages complements the effect of mortality, since adding the effects of mortality and wages generates a willingness to pay closer to the baseline than mortality alone. Instead, a comparison of the dash-dotted brown line with the dotted green line reveals that adding the effect of wages to the effect of the time endowment reduces the willingness to pay, meaning that the substitution effect dominates the income effect when the individual has more hours to allocate and a higher wage offer.

The results from the decompositions performed in this section suggest that policies aimed at reducing the time cost of bad health, such as incentivizing remote work, or providing home-care support, would be especially effective in improving the welfare of those in bad health.

6.4 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 results from the previous section reveal the importance of the model's nonlinearities and interdependency between channels for assessing outcomes; accounting for a continuous measure of health changes results quantitatively and qualitatively. In this section, we show that different levels of health affect not only outcome levels, but also their variation.

6.4.1 Removing the effects of health

We first analyze the same decomposition we used to generate Table 2, reporting the results in Table 3. Our baseline model can reproduce the reported measures of inequality from the data fairly well. Removing all effects of health by setting mortality, the time cost of health, and wages at the level they have when health is equal to the 75th percentile by age reduces the 80/20th percentile ratio by 0.42, or 11%, the 50/20th percentile ratio by 0.14 (6%), the Gini coefficient of the asset distribution by 0.05 (11%). The standard deviation of assets increases slightly.

A decomposition reveals that the effect of health on Mortality is the most significant factor contributing to asset inequality in terms of the Gini coefficient and 50/20 percentile ratio, whereas the Time cost of bad health is the most important contributor to inequality according to the 80/20th percentile ratio.

Interestingly, all indices of inequality increase if health is set at the 25th percentile of the distribution, except for the standard deviation which decreases slightly. The reason is that we are imposing a negative health shock on a majority of individuals, which, as highlighted earlier in Figure 8, affects the poor more than the rich. These negative shocks are also larger in magnitude than the positive shocks. While setting everyone to the same percentile of health level contributes to reducing variation, the differential effect of the shock by wealth is larger in magnitude.

Table 3: The effects of health on assets inequality

Effects removed	Percentile ratio 80/20	Percentile ratio 50/20	Gini	St. dev.
None (Data)	4.04	2.27	0.41	134
None (Baseline)	3.70	2.42	0.49	109
(a) Counterfactual health: 75th percentile				
All (relative to baseline)	-11%	-6%	-11%	+1%
Decomposition:				
Mortality	-4%	-3%	-7%	-1%
Time cost	-6	-3	-2	+2
Wages	-3	-2	-1	+0
Mortality+Time	-6	-3	-9	+0
Mortality+Wages	-6	-5	-8	-1
Time+Wages	-11	-6	-4	+2
(b) Counterfactual health: 25th percentile				
All (relative to baseline)	+18%	+10%	+8%	-3%

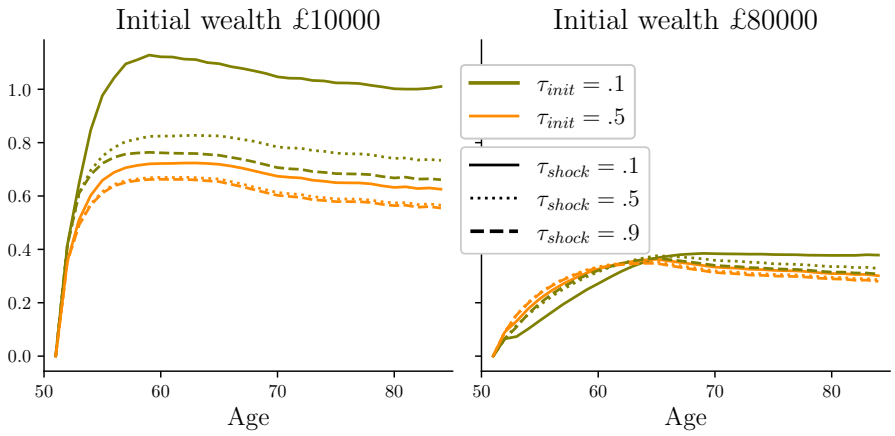
The table reports measures of assets inequality in the data and the baseline model simulations (first two rows) and percent changes from the baseline (following rows) after assigning everyone either the mortality rate, time cost of health, or wage offer computed at the percentile of the age-specific health distribution indicated at the top of each panel. Standard deviation measured in thousand pounds.

6.4.2 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 8 on inequality. Figure 11 displays the coefficient of variation of assets, by age, for people starting at age 51 with two initial levels of wealth, 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 1.4 before the age of 60, and remaining higher than 1.2. On the other hand, for individuals with the same initial wealth, dispersion is lower after a good shock than after a median shock. 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 negligible for initially wealthier individuals

Figure 11: Assets, coefficient of variation following a shock



The Figure reports the age profiles of the coefficients of variations of accumulated assets for different levels of τ_{init}, τ_{shock}, and initial assets. Average of 5,000 histories per simulation.

(right panel), and (not reported in the figure) are even smaller for people starting from an initial level of wealth of £200,000.

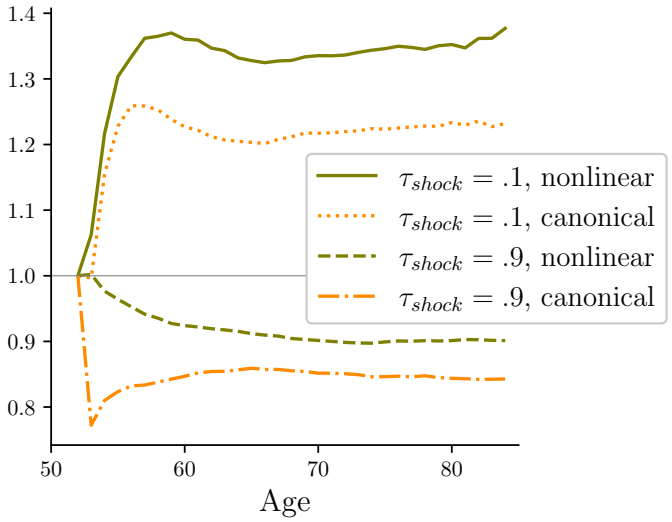
The persistency of bad shocks affects asset accumulation permanently, especially so for individuals starting with a low level of wealth. This evidence helps explain the differential effects of health we uncovered previously when discussing the decomposition results of Table 3. Bad health shocks affect inequality differently by wealth, contributing to increasing variation when health is reduced to the 25th percentile for all. Good health shocks do not affect inequality as differently, hence when all effects of health are computed at the 75th percentile, the equalizing-health effects dominate, and indices of inequality are lower than the baseline.

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

Notice, in the left panel of Figure 11, the asymmetric effects of good and bad shocks, with respect to the median shock. Nonlinearities in the health process contribute to emphasizing this asymmetry, as illustrated in Figure 12, which compares the two models focusing on individuals starting with £10,000 in assets, plotting the coefficients of variation following good and bad shocks, as a fraction of the same measure following a median shock.

The solid green line shows that in the nonlinear model, 4 years after a bad shock the coefficient of variation gets over 35% larger than after a median shock. In the canonical model, variation increases, but not as dramatically (dotted yellow line). Following a good shock, variation decreases *more* in the canonical model, relatively to following a median shock.

Figure 12: Assets, coefficient of variation following good and bad shocks, relative to a median shock



The Figure plots the 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 15,000 histories per simulation.

Not shown in the figures, for individuals starting off at age 50 with higher wealth the two models perform similarly. These results show that for poor individuals, the canonical model is not capable of generating the same dispersion of outcomes generated by health shocks.

7 Conclusion

This paper shows that the health dynamics is highly nonlinear and that the quantile-based methodology developed by Arellano et al. (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 complex measure of health, 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 of the importance

of policies aimed at relieving individual time pressure, such as facilitating work from home, or providing home care.

A Appendix: health processes

A.1 Nonlinear health process

Following De Nardi et al. (2020), after estimating the health process using Arellano, Blundell and Bonhomme's procedure, we simulate a large number of 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).[13]

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}.[14]

Selection into mortality in the original data and the discretization of the process lead to underestimation of health in simulated health trajectories, after death probabilities conditional on health are applied to the process. Therefore, we apply an iterative procedure and rescale the original data at each age in such a way that median simulated health conditional on survival coicides with median health in the data.

A.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 (2). 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

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

[14] 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 intensive and does not always have a real-valued solution.

Table A.1: Canonical health process parameter estimates

Random component		
ρ	0.953	(0.016)
σ^2_ν	0.084	(0.023)
σ^2_ϵ	0.137	(0.028)
σ^2_0	0.450	(0.051)

Table B.2: Earnings process parameter estimates

Random component		
ρ_e	0.896	(0.054)
$\sigma^2_{\nu^e}$	0.034	(0.021)
σ^2_υ	0.226	(0.031)
σ^2_0	0.148	(0.039)

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 A.1 reports the estimates.

A.3 Mortality

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.

B Appendix: earnings process

Table B.2 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 Appendix A).

C Appendix: 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.

$$a_{it} = f_i + \sum_{n=1}^{S} \pi_n age_{it}^n + \pi_U U_t + u_{it} \tag{10}$$

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 1946 and 1955.

D Appendix: 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:

$$
\begin{aligned}
V(X_t) &= \max_{a_{t+1},s_t} \Bigg\{ U(c_t,l_t) + \beta\pi^{t+1} \iint_{e_{t+1},h_{t+1}} V(X_{t+1}|X_t) dF(X_{t+1}|X_t) \qquad (11) \\
&\quad + \beta(1-\pi^{t+1}) b(a_{t+1}) \Bigg\} \\
&\quad \text{subject to:} \\
a_{t+1} &= a_t + s_t \cdot e_t \cdot (1 - c_p \mathbb{1}(t<15)) + r \cdot a_t - tax_t + tr_t - c_t - \phi_w(\mathbb{1}(s_t>0,t)) \qquad (12) \\
l_t &= L - s_t - \phi_h(h_t) \\
tax_t &= f(s_t \cdot e_t \cdot (1 - c_p \mathbb{1}(t<15)) + r_p \cdot p_t \mathbb{1}(t \geq 15), r \cdot a_t) \\
tr_t &= \max\left(0, \underline{c} - (a_t + r_p \cdot p_t \mathbb{1}(t \geq 15) + s_t \cdot e_t \cdot (1 - c_p \mathbb{1}(t<15)) + r \cdot a_t - tax_t)\right)
\end{aligned}
$$

The value function (11) is also subject to $a_t > 0$. Transfers tr_t are computed so that $c_t \geq \underline{c}, \forall t$. States h_t, e_t evolve according to the processes specified in Appendices A and B, with earnings $e_t = 0$ if $t \geq 21$ (after age 69). Assets a_t evolves according to constraint (12), and pension wealth $p_{t+1} = p_t + e_t(1 - c_p \mathbb{1}(t<15))$.

E Appendix: functional form specification and estimates

We model the time cost of health $\phi_h(h_t)$ as 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, -.633, -.170, .340). We constrain $\phi_h(\hat{h}) = 0$. Table E.3 reports the value of ϕ_h at the remaining four knots. The cost of work is a linear function

$$
\phi_w(\mathbb{1}(s_t>0,t)) = \begin{cases} \phi_w^1 + \phi_w^2 \cdot t, & \text{if } t < 65 \\ \phi_w^1 + \phi_w^2 \cdot t + \phi^3 \cdot s_t & \text{if } t >= 65 \text{ and } s_t > 1250 \end{cases}
$$

The extra cost suffered by older workers working part-time is identified by the sudden drop in participation and hours worked occuring at the age of 65.

To estimate the model, we simulate 15,000 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.

Table E.3: Life-cycle model parameter estimates

Calibrated

Time endowment	L	4880
Consumption floor	$\underline{c}$	1660
Risk aversion	ν	4
Pension annuity rate	r_p	0.0378
Pension contribution rate	c_p	0.06
Discount factor	β	0.9756

Estimated

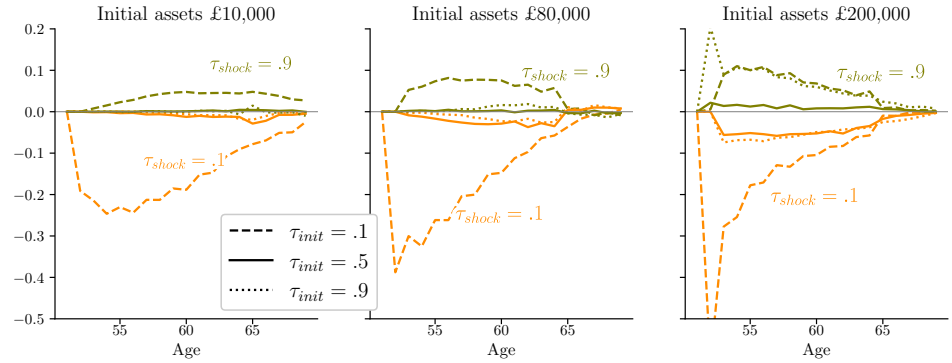
Parameter		Nonlinear	Canonical
Consumption weight	γ	0.378	0.378
Bequest	ϕ_B	0.042	0.067
	K	533219	522361
Cost of health	$\phi_h(h_{50})$	1401.9	1455.7
	$\phi_h(h_{30})$	1409.0	1461.3
	$\phi_h(h_{20})$	2312.5	2390.2
	$\phi_h(\underline{h})$	4879.0	4879.1
Cost of work	ϕ_w^1	3585.0	3518.9
	ϕ_w^2	32.8	45.5
	ϕ_w^3	2.8	3.2

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. In our loss minimization routine we also constrained $K > 0$ and $\phi_h(\cdot) < L$.

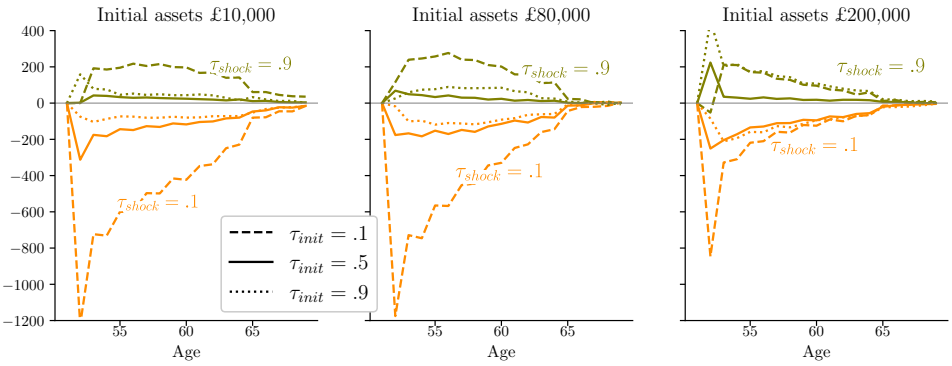
F Other results

Figure F.1: 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 canonical 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.

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Table F.4: The effect of health and its components

(a) Health: 25th percentile

Effects removed	Assets	Income	Empl.	Hours
All (relative to baseline)	-10.3%	-9.9%	-1.0%	-11.2%
Decomposition:				
Mortality	-0.9%	-0.2%	-0.3%	+0.1%
Time cost	-8.1	-7.5	+0.5	-11.1
Wages	-2.7	-3.3	-0.7	-0.8
Mortality+Time	-8.6	-7.6	+0.3	-10.9
Mortality+Wages	-3.5	-3.5	-0.9	-0.7
Time+Wages	-9.9	-9.9	-0.9	-11.4

(b) Health: 50th percentile

Effects removed	Assets	Income	Empl.	Hours
All (relative to baseline)	-2.6%	-1.9%	+4.2%	-5.8%
Decomposition:				
Mortality	+0.2%	-0.2%	-0.3%	+0.2%
Time cost	-2.9	-2.2	+3.3	-5.3
Wages	-0.6	-0.4	+1.3	-1.0
Mortality+Time	-3.2	-2.7	+3.0	-5.6
Mortality+Wages	-0.4	-0.5	+1.1	-0.8
Time+Wages	-2.6	-1.6	+4.3	-5.5

(c) Health: 90th percentile

Effects removed	Assets	Income	Empl.	Hours
All (relative to baseline)	+15.9%	+21.0%	+15.9%	+13.1%
Decomposition:				
Mortality	+1.4%	-0.1%	-0.1%	+0.4%
Time cost	+9.4	+14.7	+12.6	+13.6
Wages	+3.9	+5.6	+4.8	-1.5
Mortality+Time	+9.9	+13.3	+12.3	+13.0
Mortality+Wages	+5.5	+5.3	+4.8	-1.2
Time+Wages	+14.9	+22.5	+16.2	+13.6

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 percentile of the age-specific health distribution indicated at the top of each panel. Assets and (disposable) Income in thousand pounds averaged throughout the life cycle. Employment rate averaged from age 50 to 69. Hours are annual, conditional on employment.

References

Ameriks, John, Joseph Briggs, Andrew Caplin, Matthew D. Shapiro, and Christopher Tonetti, "Long-Term-Care Utility and Late-in-Life Saving," *Journal of Political Economy*, 2020, *128* (6), 2375–2451. (Cited on page 2)

Arellano, Manuel, Richard Blundell, and Stéphane Bonhomme, "Earnings and Consumption Dynamics: A Nonlinear Panel Data Framework," *Econometrica*, 2017, *85* (3), 693–734. (Cited on pages 2, 3, 6, 7, 21, and 22)

Blundell, Richard, Jack Britton, Monica Costa Dias, and Eric French, "The impact of health on labor supply near retirement," *Journal of Human Resources*, 2021, pp. 1217–9240R4. (Cited on pages 2 and 7)

Blundell, Richard W, Jack Britton, Monica Costa Dias, and Eric French, "The dynamic effects of health on the employment of older workers," Michigan Retirement Research Center Research Paper 2016. (Cited on pages 4 and 24)

Bound, John, "Self-reported versus objective measures of health in retirement models," *Journal of Human Resources*, 1991, *26* (1), 106–138. (Cited on pages 2 and 5)

_ , Michael Schoenbaum, Todd R. Stinebrickner, and Timothy Waidmann, "The dynamic effects of health on the labor force transitions of older workers," *Labour Economics*, 1999, *6*, 179–202. (Cited on page 5)

_ , Todd Stinebrickner, and Timothy Waidmann, "Health, economic resources and the work decisions of older men," *Journal of Econometrics*, 2010, *156* (1), 106–129. (Cited on page 5)

Burkhauser, Jean M. Mitchell J. S. Butler Richard V. and Theodore P. Pincus, "Measurement error in self-reported health variables.," *Review of Economics and Statistics*, 2010, *69* (4), 644–650. (Cited on page 5)

Capatina, Elena, "Life-cycle effects of health risk," *Journal of Monetary Economics*, 2015, *74*, 67–88. (Cited on pages 2 and 15)

Dal Bianco, Chiara, "Disability Insurance and the Effects of Return-to-work Policies," Working paper, Università di Padova 2022. (Cited on pages 2 and 7)

De Nardi, Mariacristina, Eric French, and John B Jones, "Why do the elderly save? The role of medical expenses," *Journal of Political Economy*, 2010, *118* (1), 39–75. (Cited on page 2)

_ , Svetlana Pashchenko, and Ponpoje Porapakkarm, "The lifetime costs of bad health," National Bureau of Economic Research working paper #w23963 2017. (Cited on page 2)

French, Eric, "The effects of health, wealth, and wages on labour supply and retirement behaviour," *The Review of Economic Studies*, 2005, *72* (2), 395–427. (Cited on pages 2 and 10)

_ and John Bailey Jones, "Health, health insurance, and retirement: a survey,"

Annual Review of Economics, 2017, *9*, 383–409. (Cited on page 2)

Hosseini, Roozbeh, Karen A. Kopecky, and Kai Zhao, "The evolution of health over the life cycle," *Review of Economic Dynamics*, 2021. (Cited on page 2)

_ , Karen A Kopecky, and Kai Zhao, "The evolution of health over the life cycle," *Review of Economic Dynamics*, 2021. (Cited on pages 6 and 7)

Low, Hamish and Luigi Pistaferri, "Disability insurance and the dynamics of the incentive insurance trade-off," *American Economic Review*, 2015, *105* (10), 2986–3029. (Cited on page 2)

Nardi, Mariacristina De, Eric French, and John Bailey Jones, "Savings after retirement: A survey," *Annual Review of Economics*, 2016, *8*, 177–204. (Cited on page 2)

_ , Giulio Fella, and Gonzalo Paz-Pardo, "Nonlinear household earnings dynamics, self-insurance, and welfare," *Journal of the European Economic Association*, 2020, *18* (2), 890–926. (Cited on pages 10 and 22)