THE RICH LIVE LONGER: A MODEL OF INCOME AND HEALTH INEQUALITIES

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PABLO GARCIA-SANCHEZ AND OLIVIER PIERRARD

Abstract. Income and life expectancy are strongly correlated, yet the mechanisms underlying this relationship remain debated. This paper develops a structural model in which both variables are endogenous and jointly determined. Calibrated to U.S. data, the model replicates the income-longevity gradient and the distribution of age at death. It highlights the importance of both the health-to-income and income-to-health channels in accounting for these empirical patterns. Our model also offers a more cautious assessment of income redistribution policies than empirical studies, showing that redistribution can weaken incentives for preventive care and increase mortality risk. By contrast, lowering the price of preventive care through subsidies promotes better health and longer lives.

JEL Codes: C60, D15, H24, H51, I12, I14.

Keywords: Health, Income, Inequality, Redistribution, Subsidy.

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1. Introduction

The rich live longer. This strong correlation between wealth and life expectancy persists across time and countries (see e.g., Kitagawa and Hauser, 1973; Smith, 1998; Cutler et al., 2006, 2011). In the United States, for instance, 40-year-old women (men) in the top percentile of the income distribution can expect to live 15 (10) years longer than those in the bottom percentile (Chetty et al., 2016). Disparities in life expectancy are not confined to the extremes; they follow a smooth gradient across the income distribution.

Income and life expectancy are linked through multiple channels (Deaton, 2002; Chandra and Vogl, 2010; Cutler et al., 2011; Baker and Stabile, 2012). Income may affect health by enabling access to better medical care or nutrition, while health can affect income by shaping labor productivity and earnings. Both may also correlate with other factors, such as education or genetics, and in practice, all channels may operate simultaneously.

Against this backdrop, we present a simple yet quantitatively rich structural model in which income and life expectancy are endogenous and connected. We explore the mechanisms behind the income-longevity gradient and to assess government redistribution policies recently analyzed in statistical settings (see e.g., Himmelstein et al., 2024; Gugushvili and Wiborg, 2025). Our structural model offers a more cautious view of redistribution's impact on the distribution of age at death, as it accounts for how policy shapes individual incentives. By contrast, lowering the price of preventive care through subsidies yields more favorable longevity outcomes.

Our setup is a continuous-time life-cycle model with heterogeneous agents, in which both life expectancy and income are endogenous. As individuals age, they experience adverse health events that accumulate over time, collectively referred to as the health deficit. This deficit matters for three reasons. First, it generates medical costs, including long-term care, curative treatments, and palliative services. Second, it reduces labor productivity and wages. Third, it raises mortality risk: following empirical evidence in Scott (2023), individuals die not from age itself but from declining health, with the timing of death determined by their health deficit.

To counteract these adverse effects, individuals can slow the accumulation of health deficits by investing in preventive care: measures aimed at avoiding diseases and risk factors (e.g., vaccination, a healthy diet, or regular exercise) or detecting illnesses early (e.g., screenings). Our work is then part of the tradition began by the seminal contributions of Grossman

(1972) and Ehrlich and Becker (1972), which emphasize that health resembles an investment good whose evolution can be shaped through effort and resources.

Our framework features a *health-to-income channel*, where poorer health lowers wages, and an *income-to-health channel*, where higher earnings allow greater investment in preventive care. While both channels are recognized, most structural models focus on only one, typically the health-to-income channel (see Section 2). The model also includes agent heterogeneity; otherwise, all t-year-olds would earn the same income and share the same life expectancy, ruling out any income-longevity gradient. Specifically, individuals differ in the permanent component of labor productivity, capturing variation in work ethic, cognitive ability, or access to quality education and professional networks (see e.g., Becker, 1994, for evidence on the role of schooling and ability in generating life-cycle earnings differences).

To calibrate the model, we draw primarily on the 2022 National Health Interview Survey, the 2023 Medical Expenditure Panel Survey, and actuarial data from the 2022 Human Mortality Database. The model performs well in validation exercises, closely matching non-targeted patterns such as the distribution of age at death and the income-longevity gradient documented by Chetty et al. (2016): a 10% increase in income at middle age is associated with a gain in life expectancy of roughly six months.

Counterfactual analyses show that both the health-to-income and income-to-health channels matter for matching these patterns. Shutting down the health-to-income channel, so that earnings are independent of health, preserves the gradient but worsens the model's fit to the age-at-death distribution. Shutting down the income-to-health channel, so that health investment is independent of income, eliminates the gradient altogether.

Next, we contribute to the debate on the effects of wealth and income redistribution on mortality. As mentioned earlier, recent empirical studies reaffirm the strong link between wealth and longevity. Many of these studies conclude with a policy recommendation advocating redistribution, suggesting that reducing wealth inequality would lead to a more desirable distribution of age at death. For example, Himmelstein et al. (2024) argue that perfect wealth equality would increase US median longevity by 2.2 years. Our structural model offers a more cautious view. Redistribution weakens the link between health and disposable income, reducing the incentive to invest in preventive care and raising mortality risk. In our setup, redistribution policies actually *lower* median longevity. This highlights the need for caution when relying solely on statistical models, which are subject to the Lucas critique and may overstate the benefits of redistribution.

In addition, we show that the welfare consequences of income redistribution depend critically on which channel (income-to-health or health-to-income) is active. Ignoring one channel is not innocuous: if the health-to-income channel is shut down, redistribution no longer weakens

the link between health and disposable income (because there is none), and the predicted effects resemble those in Himmelstein et al. (2024). Conversely, if the income-to-health channel is shut down, redistribution reduces income inequality but leaves the age-at-death distribution unchanged, since it no longer responds to income. In both cases, redistribution is welfare improving. When both channels operate, however, the same redistribution lowers welfare.

Our model also indicates that lowering the price of preventive care through subsidies raises preventive investment and reduces mortality risk. The resulting shift in the age-at-death distribution closely mirrors what statistical models attribute to redistribution. For example, a 10% preventive care subsidy increases life expectancy at birth by nearly one year and yields a welfare gain of 1.7% relative to the baseline. This result is consistent with recent empirical evidence on the Affordable Care Act, which expanded access to affordable coverage and reduced mortality, especially for causes of death amenable to medical care (Sullivan et al., 2024; Borgschulte and Vogler, 2020; Miller et al., 2021).

Lastly, in one of three robustness checks, we extend the baseline model by introducing a risk-free asset that allows agents to transfer resources over time. Access to this asset affects longevity in much the same way as the preventive care subsidy. Both mechanisms shift resources from old to young in order to support greater preventive investment early in life. Hence, the model suggests that health subsidies could help offset the adverse effects of limited access to financial services, which remain significant in the United States, where roughly 40% of households live hand-to-mouth (Aguiar et al., 2024).

The remainder of the paper is organized as follows. Section 2 reviews the related literature. Section 3 presents the structural setup. Section 4 details the estimation and calibration procedure. Sections 5, 6, and 7 report the main quantitative results, while Section 8 tests their robustness. Section 9 concludes.

2. Literature review

2.1. **Empirical literature.** As mentioned earlier, there is a strong, persistent correlation between income and life expectancy, with richer individuals living significantly longer lives (Chetty et al., 2016). This gradient holds across countries and throughout the income distribution (Deaton, 2016). However, disentangling causality is difficult, as income and health influence each other through multiple channels, and shared determinants like education may confound the relationship (Deaton, 2002; Chandra and Vogl, 2010; Cutler et al., 2011; Baker and Stabile, 2012).

To illustrate, Panel A in Table 1 highlights the protective effects of parental income on self-reported health status later in life (a higher score indicates worse health, on a scale from

TABLE 1. On the links between wealth and income

Panel A	Family income from birth to age 16		
(Source: 2022 HRS)	Low	Medium	High
Self-reported health at (approx.) 60 years	3.3	3.0	2.7
Panel B	Difficulties with mobility, communication or cognition		
(Source: 2023 NHIS)	Onset before 22	Onset after 22	None
Prob. of below-poverty threshold	26.5	20.1	8.7
Panel C	Education Level		
(Source: 2023 NHIS)	Below college		Above college
Prob. of smoking regularly	11		7
Prob. of working last week	47.1		63.2

Notes. HRS refers to the Health and Retirement Study (over 2,300 survey respondents), and NHIS refers to the National Health Interview Survey (over 29,000 survey respondents). In Panel A, self-reported health is measured on a scale from 1 (excellent) to 5 (poor). Panel B reports the probability of being below the poverty threshold by age of disability onset. Panel C shows the probability of smoking regularly and the probability of employment in the past week, both by education level.

1 to 5) using data from the 2022 Health and Retirement Study (HRS). These figures echo Case and Paxson (2002), who study how parental behavior and socioeconomic status impact children's health, exploiting that child health is unlikely to influence family income. Their results align with other studies showing that children from low-income households tend to have lower birth weights, a higher likelihood of being born prematurely, and an increased risk of chronic health conditions as they grow older (Brooks-Gunn and Duncan, 1997; Newacheck and Halfon, 1998; Currie, 2009).

In turn, Panel B in Table 1 highlights the damaging effects of early-onset disabilities on economic well-being using data from the 2023 National Health Interview Survey (NHIS). Individuals whose difficulties with mobility, communication, or cognition began before age 22 are three times more likely to live below the poverty threshold later in life than those without such difficulties. This message echoes the literature showing that healthier children become wealthier adults. For example, Behrman and Rosenzweig (2004) and Black et al. (2007) show that lower birthweight babies face worse outcomes, both in the short run, in terms of one-year mortality rates, and in the longer run, in terms of labor market payoffs. Similarly, Almond (2006) uses the 1918 influenza pandemic as a natural experiment to reveal how improving fetal health can enhance the future human capital of these babies.

Lastly, Panel C highlights how third factors – education in this case – may influence both health and wealth, using data from the 2023 NHIS. The first row indicates that individuals with higher levels of education are less likely to smoke, consistent with Grimard and Parent (2007); de Walque (2010). Along the same lines, Lleras-Muney (2005) provides strong

empirical evidence of the significant causal impact of education on mortality. In addition, Mackenbach (2006) and Cutler et al. (2011) shows that more educated individuals in Europe and the United States report better health and face lower mortality risks. Glied and Lleras-Muney (2008), in turn, find that more educated individuals are better positioned to take advantage of medical breakthroughs, reducing their mortality risk for diseases with greater innovation. As for the second row of Panel C, it stresses that individuals with higher levels of education are more likely to be employed. This is a well-established fact: better-educated individuals earn higher wages, face lower unemployment rates, and hold more prestigious occupations than those with less education (see e.g. Card, 1999, and references therein).

On the whole, reverse causation and omitted variable bias complicate identifying the causal links between health and wealth. Furthermore, these links may change with age (Smith, 1998; Cutler et al., 2011). Wealth might have the greatest impact in childhood when health levels and trajectories are set, while in adulthood, health might play a larger role in shaping wealth. Empirical evidence supports this view. On the one hand, studies using exogenous wealth and income shocks – such as recessions, lottery winnings, inheritances, and unexpected policy changes – find weak effects on adult health (see e.g., Ruhm, 2000, 2005; Kim and Ruhm, 2012; Apouey and Clark, 2015; Cesarini et al., 2016; Erixson, 2017). As discussed earlier, however, parental income seems to have a strong protective effect on children's health. On the other hand, among adults, the negative impact of poor health on wealth explains a significant part of the correlation between the two. For example, Smith (1998) estimates that a severe illness reduces total household wealth by about 8% on average. Furthermore, negative health shocks are strong predictors of retirement and reduced labor force participation (Smith, 2004, 2005; Case and Deaton, 2005).

Taken together, the evidence points to a bidirectional relationship between health and income. Structural models seeking to study health and longevity outcomes should therefore account for both channels rather than focusing on only one. Most theoretical setups, however, consider only one at a time, as we discuss next.

2.2. **Theoretical literature.** A first body of work assumes that health, and therefore survival probabilities, is exogenous, which prevents assessing the impact of policy on health dynamics (Pashchenko, 2025). For example, using an estimated life-cycle setup, De Nardi et al. (2024) show that losses from poor health are substantial and largely driven by factors determined early in life. Capatina (2015) highlights that poor health contributes to income inequality primarily through productivity losses and time costs, rather than higher medical expenditures. Similarly, Hosseini et al. (2025) argue that disability programs generate strong work disincentives, and are an important driver of the relationship between health

and earnings inequalities. Low and Pistaferri (2015) also study disability programs, weighing the trade-off between incentive costs and insurance benefits.

In contrast, Ozkan (2025) allow individuals to affect the distribution of health shocks by investing in preventive health capital, thereby shaping life expectancy. The author finds that public insurance, which covers large curative expenditures, can widen the life expectancy gap by reducing the poor's incentives to invest in preventive health. However, this study assumes that labor productivity, and thus income, is exogenous over the life cycle.

A few recent papers examine how health influences economic circumstances and vice versa. Mahler and Yum (2024) develop a heterogeneous-agent life-cycle model where health and wealth evolve endogenously. Using German microdata, they argue that if all individuals adopted the same age-specific health effort level, wealth inequality would decline by roughly 50%. In their framework, wealthier individuals invest more in health-promoting activities, leading to better health outcomes, higher labor income, and increased savings, a mechanism consistent with our setup. Also worth noting is Cole et al. (2018), who examine both the health-to-income and income-to-health channels. They analyze the trade-off between limiting the extent to which wages and insurance premiums can depend on a worker's health status and the resulting reduction in household incentives to maintain health. Their findings suggest that while health-related risks in labor and insurance markets justify strong social insurance (about 80% coverage), fully severing the link between wages and insurance premiums and health status is suboptimal, as the long-term adverse effects on health effort outweigh the short-term consumption insurance benefits.

These papers differ from ours in both their formulation and research focus. For example, we follow Dalgaard and Strulik (2014) and Hosseini et al. (2022) in measuring health through a frailty index and treating it as a continuous variable, rather than using a coarse discrete classification such as good or bad health. This approach is more realistic, as health changes gradually rather than in sudden jumps. It also helps align the model with the data, given the established empirical literature on constructing health deficit indices (see e.g., Mitnitski et al., 2002; Rockwood and Mitnitski, 2007). Our analysis further stresses the need to consider both the income-to-health and health-to-income channels when assessing government policy, also showing that redistribution policies analyzed in statistical models can lead to very different outcomes in a structural framework.

3. Baseline Continuous-time Life-cycle Model

This section introduces the baseline continuous-time life-cycle model. We focus on the stationary population distribution, in which a new cohort of size one is born at each instant. Agents differ in their permanent income component, giving rise to income heterogeneity within each age cohort. We now set out the optimization program of one agent.

3.1. **Setup.** Let $d(t)$ represent an individual's health deficit, which evolves according to the following law of motion

$$\dot{d}(t) = \gamma d(t) - \frac{Ah(t)^{\beta}}{\beta}, \tag{1}$$

$$d(0) = d_0, \tag{2}$$

where d_0 is a strictly positive parameter. Equation (1) captures a simple dynamic: as an individual ages, her health deteriorates, reflected by the accumulation of the health deficit at a rate $\gamma > 0$. However, preventive care, $h(t)$, slows down this process. In its most restrictive definition, preventive care includes activities aimed at preventing diseases and risk factors (e.g., vaccination) or early disease detection (e.g., screening). A broader definition might encompass lifestyle changes that promote better health, such as maintaining a healthy diet and engaging in regular exercise. The parameter $A > 0$ governs the effectiveness of preventive care, while $\beta \in (0, 1)$ introduces diminishing returns to scale, as in Dalgaard and Strulik (2014). This reflects the principle that as preventive care efforts increase, each additional intervention yields a smaller marginal improvement in health. For example, repeated screenings for the same cancer type within a short period provide little value added.

The individual's lifespan extends from 0 to T, where T is a random variable. The probability law governing T is defined by the hazard rate $\lambda(t, d(t)) \geq 0$, which is a C^1-function in both arguments.[1] When calibrating the model, we expect both first partial derivatives to be positive, as the likelihood of death increases with both age and the individual's health deficit (Scott, 2023). For example, in colorectal cancer, younger patients in the US tend to have better survival rates than older patients, even at the same stage of diagnosis (Cheng et al., 2021). Similarly, five-year net survival is highest in the youngest adults in the UK for nearly all cancers, with survival generally decreasing with increasing age (Office for National Statistics, 2013).

Given the probability law governing the time of death, the likelihood of being alive at age t is $\Lambda(t) = \mathrm{e}^{-\int_0^t \lambda(u,d(u))\mathrm{d}u}$, which starts at 1 at birth and decreases monotonically as the individual ages. We prevent unjustifiably long lifespans by imposing an exogenous maximum health deficit $\bar{d} > d_0$: if the individual reaches $\bar{d}$, she passes away immediately. Consequently, the maximum attainable age, $\bar{T}$, is implicitly determined by $d(\bar{T}) = \bar{d}$.

At each instant $t \in [0, T]$, the individual supplies one unit of labor inelastically, earning a wage $y(t, d(t)) > 0$, which is a C^1-function in both arguments (see Appendix A for a microfoundation). When calibrating the model, we expect the partial derivative of earnings with respect to the first argument to be positive, reflecting the general pattern of rising

[1] As a result, the cumulative probability function is $F(t) = 1 - \mathrm{e}^{-\int_0^t \lambda(u,d(u))\mathrm{d}u}$ and the probability density function is $f(t) = \partial F(t)/\partial t = \lambda(t, d(t))\,\mathrm{e}^{-\int_0^t \lambda(u,d(u))\mathrm{d}u}$. The hazard rate is $f(t)/(1 - F(t)) = \lambda(t, d(t))$.

earnings as individuals advance in their careers. In contrast, we expect the partial derivative with respect to the second argument to be negative, as poor health tends to lower income, due to factors such as involuntary unemployment, reduced productivity, or early retirement (see Section 2). As we shall see, these two forces together will generate the hump-shaped earnings profile observed in the data.

The individual's budget constraint is

$$c(t) + \theta(h(t) + Bd(t)) = y(t, d(t)), \tag{3}$$

where $c(t)$ represents consumption and $\theta > 0$ is the relative price of medical-related activities. The term B is a positive parameter that captures the monetary cost associated with the health deficit, $\theta B d(t)$. These costs represent pure expenditures with no direct effect on the health deficit and can be viewed as expenses related to long-term care (e.g., nursing, home care) as well as curative and palliative care.

The individual's expected lifetime utility is

$$\int_0^{\bar{T}} e^{-\rho t} \Lambda(t) \left[\ln c(t) + \alpha\right] \, \mathrm{d}t. \tag{4}$$

Here $\rho \geq 0$ is the discount rate and $\alpha \geq 0$ is a technical constraint ensuring that utility flows remain strictly positive over the life-cycle. Otherwise, the individual would prefer an earlier death, as continuing life would result in negative utility (see for instance Dragone and Strulik, 2020, for a similar discussion).[2]

The individual chooses sequences $\{c(t), h(t)\}_{t=0}^{\bar{T}}$ to maximize (4), subject to (1)-(3) and the endogenous hazard rate $\lambda(t, d(t))$.

3.2. **Solution.** We solve our stochastic control problem by reformulating it as an equivalent deterministic control problem (see Boukas et al., 1990, for mathematical proofs). As mentioned earlier, the probability that the individual is alive at time t is $\Lambda(t) = \mathrm{e}^{-\int_0^t \lambda(u,d(u))\mathrm{d}u}$. Hence, we have $\dot{\Lambda}(t) = -\lambda(t, d(t))\Lambda(t)$ with $\Lambda(0) = 1$, allowing us to write the Hamiltonian function

$$\begin{aligned} H(t) \;=\; & \mathrm{e}^{-\rho\, t}\, \Lambda(t)\, \left[\ln c(t) + \alpha\right] - \tilde{q}(t) \left[\gamma d(t) - \frac{A h(t)^{\beta}}{\beta}\right] \\ & -\tilde{p}(t)\lambda(t, d(t))\Lambda(t) + \tilde{\epsilon}(t) \left[y(t, d(t)) - c(t) - \theta(h(t) + Bd(t))\right]. \end{aligned}$$

Here $-\tilde{q}(t)$ is the shadow price of health deficit and measures the value of an infinitesimal increase in $d(t)$. Similarly, $\tilde{p}(t)$ is the shadow price of the probability of survival, better known as the value-of-life-saving (Schelling, 1968; Mishan, 1971). It measures the remaining lifetime utility along the optimal path from t to $\bar{T}$. Lastly, $\tilde{\epsilon}(t)$ measures the change in the

[2]We could also add a disutility related to deteriorating health. This would only provide one more incentive to invest in prevention, without changing our key insights.

optimal value of the utility function per unit of change in the budget constraint. Economic logic suggests that all three co-state variables $\{\tilde{q}(t), \tilde{p}(t), \tilde{\epsilon}(t)\}$ should be positive.

Applying the maximum principle to $H(t)$ yields

$$\begin{cases} H_h = 0\,, & H_c = 0\,, & H_{\tilde{\epsilon}} = 0\,, \\ H_d = \dot{\tilde{q}}(t)\,, & H_\Lambda = -\dot{\tilde{p}}(t)\,, & H_{-\tilde{q}} = \dot{d}(t)\,, \quad H_{\tilde{p}} = \dot{\Lambda}(t)\,. \end{cases}$$

These necessary optimality conditions are standard in deterministic control theory. Furthermore, the concavity of the utility function ensures that these necessary conditions are also sufficient. Let $q(t) \coloneqq \mathrm{e}^{\rho\, t}\, \tilde{q}(t)/\Lambda(t)$, $\epsilon(t) \coloneqq \mathrm{e}^{\rho\, t}\, \tilde{\epsilon}(t)/\Lambda(t)$ and $p(t) \coloneqq \mathrm{e}^{\rho\, t}\, \tilde{p}(t)$. The optimal control system must thus solve the following system of nonlinear differential equations

$$\begin{cases} \dot{d}(t) = \gamma d(t) - \dfrac{A h(t)^\beta}{\beta}\,, & \text{(5a)} \\ \dot{\Lambda}(t) = -\lambda(t, d(t))\Lambda(t), & \text{(5b)} \\ \dot{q}(t) = \left[\rho + \lambda(t, d(t)) - \gamma\right] q(t) - \lambda_d(t, d(t)) p(t) + \epsilon(t)\left[y_d(t, d(t)) - \theta B\right]\,, & \text{(5c)} \\ \dot{p}(t) = \left[\rho + \lambda(t, d(t))\right] p(t) - \left[\ln c(t) + \alpha\right]\,, & \text{(5d)} \end{cases}$$

together with the three intratemporal conditions

$$\begin{cases} c(t) = \dfrac{1}{\epsilon(t)}\,, & \text{(6a)} \\ h(t) = \left(\dfrac{A q(t)}{\theta \epsilon(t)}\right)^{\frac{1}{1-\beta}}\,, & \text{(6b)} \\ y(t, d(t)) = c(t) + \theta(h(t) + B d(t))\,. & \text{(6c)} \end{cases}$$

Solving the above system of differential equations requires a set of boundary conditions, which in our setup are naturally given by

$$d(0) = d_0, \qquad \Lambda(0) = 1, \qquad d(\bar{T}) = \bar{d}\,, \qquad p(\bar{T}) = 0.$$

The first three conditions have been previously introduced. As for the last condition, it ensures that the remaining lifetime utility at the maximum attainable age $\bar{T}$ is zero, which must be the case since the objective function does not include any bequest terms. Lastly, since $\bar{T}$ is free, it must be endogenously determined by $H(\bar{T}) = 0$ (see e.g., Seierstad, 2009, for a formal derivation).

There is no closed-form solution to this nonlinear boundary value problem. Therefore, we solve it numerically using the collocation method proposed by Shampine et al. (2003).

4. Mapping the Baseline Model to the Data

We select parameter values to align the model with key observations from the US in 2022 and 2023. The parameters are divided into three groups: those determined a priori, $\{\alpha, \beta, \theta\}$;

TABLE 2. Model parametrization

Parameter	Value	Description	Parameter	Value	Description
Parameters chosen a priori					
α	3	Constant utility flow	θ	1	Relative price of care
β	0.5	Decreasing returns to h			
Parameters estimated from the data					
λ_0	-0.08	Hazard rate: scaling	λ_1	0.02	Hazard rate: sensitivity age
λ_2	3.6	Hazard rate: sensitivity health	ρ	1	Discount rate
d_0	0.02	Initial deficit	$\bar{d}$	0.31	Maximum deficit
μ_0	3	Income: scaling	μ_1	1	Income: sensitivity age
μ_2	-20	Income: sensitivity health	γ	0.75	Natural deficit growth
Parameters calibrated within the model					
A	0.007	Efficiency of prevention	B	1.87	Cost of deficit

Notes. $t = 0$ in the model corresponds to the age of 20 years, and $t = \bar{T}$ corresponds to the age of 105 years. Since our calibration implies that $\bar{T} = 4.3$, one unit of time in the model corresponds to $(105 - 20)/4.3 \approx 20$ years.

those estimated directly from data, $\{\rho, d_0, \bar{d}, \gamma, \lambda(\cdot,\cdot), y(\cdot,\cdot)\}$; and those calibrated within the model to minimize the distance between data targets and model outcomes, $\{A, B\}$. Table 2 summarizes the calibration. Importantly, most parameters in the second and third groups are calibrated to match statistics of the median agent, defined in our model as the agent with the median level of permanent income.

4.1. **Parameters chosen a priori.** We set the scaling parameter α, governing the constant flow of utility, to 3. This choice ensures positive utility flows everywhere, thereby ruling out any preference-for-death scenario. Next, we set the parameter β, governing diminishing returns to preventive care, to 0.5. Consequently, given $q(t)$ and $d(t)$, $\epsilon(t)$ becomes the unique positive solution to the quadratic equation derived by substituting equations (6a) and (6b) into (6c). Lastly, we normalize the relative price of medical care, θ, to 1.

4.2. **Parameters estimated from the data.**

4.2.1. *Discount rate, ρ.* We think of $t = 0$ as the age at which an individual reaches adulthood, setting its empirical counterpart to age 20; and we assume an annual time discount factor of 0.95. In our continuous time model, if one period corresponds to x years and the discount rate is ρ, we then must have the relationship

$$0.95^x = \mathrm{e}^{-\rho} \ .$$

We here choose to normalize $\rho = 1$, which implies that $x = -1/\ln(0.95) \approx 20$ years, i.e. one period in our model corresponds to 20 years. Based on the 2022 actuarial life table from the Human Mortality Database, only 0.2% of individuals live to age 105, so we set 105 as

the empirical counterpart for the model's maximum admissible age, $\bar{T}$.[3] Therefore, in model units, $\bar{T} = (105 - 20)/20 \approx 4.3$. However, $\bar{T}$ is endogenous and hence determined as part of the model's solution rather than set as a fixed parameter. Thus, we retain $\bar{T} = 4.3$ as a condition to match when setting the third subset of parameters.

4.2.2. *Health deficit bounds,* $\{d_0, \bar{d}\}$. We follow Mitnitski et al. (2002) and Hosseini et al. (2022) and measure an individual's health status as her cumulative number of health problems. The index, here termed the health deficit, is defined as the ratio of a person's accumulated health issues to the total number of conditions considered.

To construct the health deficit index used throughout our paper, we use data from the 2023 National Health Interview Survey (NHIS), which provides a sample of over 29,000 individuals after restricting ages to 18 to 85. We consider 17 health conditions, all requiring a medical diagnosis to prevent variations in pain thresholds from affecting the index: hypertension, high cholesterol, coronary heart disease, angina pectoris, myocardial infarction, stroke, asthma, cancer, diabetes, chronic obstructive pulmonary disease, arthritis, dementia, hepatitis, epilepsy, Crohn's disease, ulcerative colitis, and psoriasis. Following the cited studies, we weight all health conditions equally, so incurring one additional health condition increases one's deficit by 1/17 or 6%.

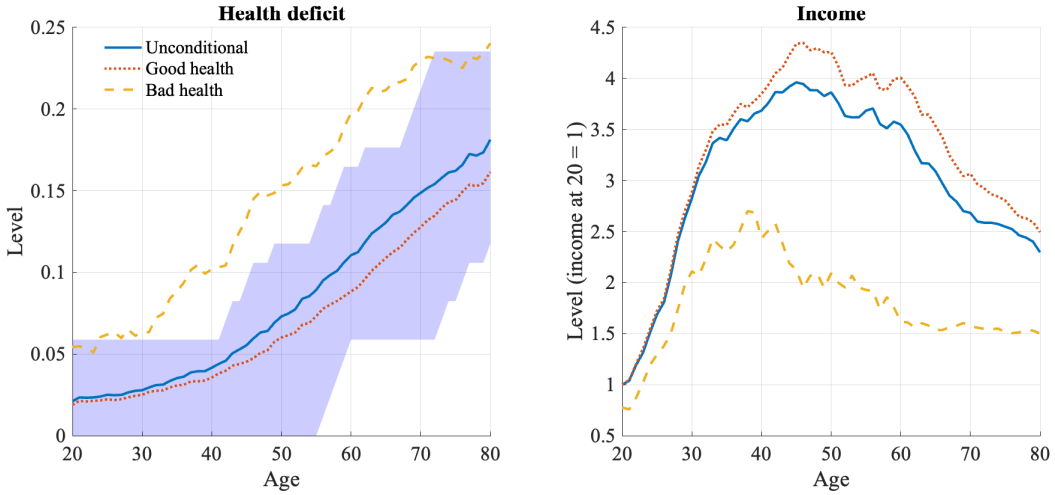
After computing the health deficit for each individual in our sample, we group individuals by age and calculate the mean health deficit for the average person within each age range. To smooth out any abrupt spikes, we apply a four-year backward moving average. The solid blue line in the left panel of Figure 1 plots the resulting index, which is a convex function of age, as found in Mitnitski et al. (2002) and Hosseini et al. (2022). This index grows at an average annual rate of roughly 3%, consistent with figures documented in studies from Australia, Canada, Sweden, and the United States (Rockwood and Mitnitski, 2007). For future reference, the red dotted line shows the mean health deficit for individuals reporting good health, and the dashed yellow line for those reporting bad health.[4] As expected, self-reported health aligns closely with our index: individuals reporting bad health have a significantly higher health deficit throughout the life cycle than those reporting good health.

As mentioned earlier, we think of $t = 0$ as the age at which an individual reaches adulthood (20 years). Therefore, we set d_0 to 0.02, matching the mean health deficit for individuals aged 20 in the data. Calibrating $\bar{d}$, representing the mean health deficit at age 105, is more

[3]For context, 2% of individuals reach the age of 100.

[4]The NHIS asks individuals to rank their health from excellent to poor. We classify those reporting excellent, very good, or good health as being in good health and those reporting fair or poor health as being in bad health.

FIGURE 1. Health deficit and income as a function of age



Notes. In the left panel, the health deficit is the ratio of accumulated health issues to the total conditions considered. The blue lines show the mean health deficit for the full sample, the red dotted line for individuals self-reporting good health, and the dashed yellow line for those self-reporting bad health. The shaded area indicates the 25th and 75th percentiles. These health deficit indexes are based on a sample of over 29,000 individuals from the 2023 NHIS. The right panel follows the same color code and is based on a sample of over 22,000 individuals from the 2022 MEPS.

challenging, as our data only includes individuals up to age 80. To overcome this problem, we follow Mitnitski et al. (2002) and fit the exponential regression

$$d(t) = \theta_0 + \theta_1 e^{\theta_2 t}, \quad t \in [20, 80],$$

yielding $\theta_0^* \approx -0.1$, $\theta_1^* \approx 0.1$, and $\theta_2^* \approx 0.02$. All coefficients are statistically different from zero at the 5% level. The fit is strong, with a root mean square error below 0.01, so we set $\bar{d} = \theta_0^* + \theta_1^* e^{\theta_2^* 105} \approx 0.31$.

4.2.3. *Natural growth of health deficits, γ.* As mentioned earlier, parameter γ represents the natural growth rate of the health deficit – its rate of increase in the absence of medical care (see equation 1). Identifying γ from the data is challenging, so we take a holistic approach, which requires a brief detour.

At the turn of the 20th century, the health care industry was not the economic behemoth it is today. In 1900, medical care accounted for just 2.5% of total GDP and employed roughly one in a hundred workers (see Table 3). By contrast, it now makes up nearly one-fifth of total GDP and employs almost one in ten workers. Over this period, medical advances and new therapeutics have reduced mortality and improved human well-being (Preston, 1975; Cutler et al., 2006), marking a departure from an era when doctors had limited training, the causes of diseases were poorly understood, and hospitals were often places where people went to die (Catillon et al., 2018). Against this backdrop, assuming that in 1900 the share of personal

TABLE 3. Age at death distribution and health industry indicators for 1900 and 2022

	Health industry (in %)		Age at death distribution (in years)		
	Expenditures to GDP	Health to total employment	Mean	Median	90th percentile
1900	2.5	1.2	48	57	82
2022	17.3	9.3	78	82	94

Notes. Data on the age-at-death distribution in 1900 are from Bell and Miller (2005), while data for 2022 are from the HMD. Information on the health industry in 1900 is from Catillon et al. (2018). Health expenditures as a share of GDP in 2022 come from the National Health Expenditure Fact Sheet (2022), and data on health care employment in 2022 are from the U.S. Bureau of Labor Statistics.

income spent on medical care (h), the efficiency of medical care (A), or both were almost nil seems reasonable (see Dalgaard and Strulik, 2014, for a similar logic). This assumption implies that in 1900, health deficits evolved by $\dot{d}(t) = \gamma d(t)$, yielding $d(t) = d_0 e^{\gamma t}$.

In words, the evolution of the health deficit in 1900 depended solely on d_0 and γ. We view these as biological parameters – fundamental to the human body and unaffected by socioeconomic conditions of the time. One might argue that nutrition, public health, or environmental factors could influence them, but the magnitude and direction of such effects are unclear. Given this uncertainty – which lies beyond the scope of this paper – we assume these parameters have remained constant over the past 125 years.

What has certainly changed over time is the distribution of age at death (see e.g. Lutz and Kebede, 2018, and references therein). Life at the turn of the 20th century was much shorter than it is today: life expectancy has increased by more than 60%, with the 90th (50th) percentile of the age-at-death distribution rising by 12 (25) years (see Table 3). Against this backdrop, assuming that the maximum admissible age in 1900 (identified in subsection 4.2.1 with the 99.8th percentile of the distribution) was 10 years lower than in 2022 seems reasonable. This assumption implies that in 1900, the maximum admissible age was 95 years, which in our model units translates to $\bar{T}^{1900} = (95 - 20) \times 4.3/(105 - 20) \approx 3.8$.

In sum, we characterize the dynamics of the health deficit in 1900 by

$$\begin{aligned} d(t) &= d_0 e^{\gamma t}, \\ d(\bar{T}^{1900}) &= \bar{d}, \end{aligned}$$

yielding

$$\gamma = \frac{1}{\bar{T}^{1900}} \ln \frac{\bar{d}}{d_0} \approx 0.75.$$

That is, the natural growth rate of the health deficit depends on the maximum admissible age in 1900, as well as on d_0 and $\bar{d}$. In line with the discussion above, we assume that the

maximum number of health disorders the human body can sustain, $\bar{d}$, has remained constant over the past 125 years.

4.2.4. *Income process,* $y(t, d(t))$. Using data from the 2022 Medical Expenditure Panel Survey (MEPS), which includes over 22,000 individuals, the solid blue line in the right panel of Figure 1 shows average income by age, normalized to 1 at age 20. Income follows a hump-shaped pattern, rising early in life, peaking in middle age, and declining as individuals reduce work hours or retire.

In turn, the dotted red and yellow lines show income by age for individuals self-reporting good and bad health, respectively.[5] Consistent with Section 2, individuals in bad health earn significantly less than those in good health. The gap widens with age, peaks around 50 (with income nearly twice as high for those in good health), and then narrows slightly.

We specify the functional form for $y(t, d(t))$ as

$$y(t, d(t)) = \mu_0 + \mu_1 t + \mu_2 d(t), \tag{7}$$

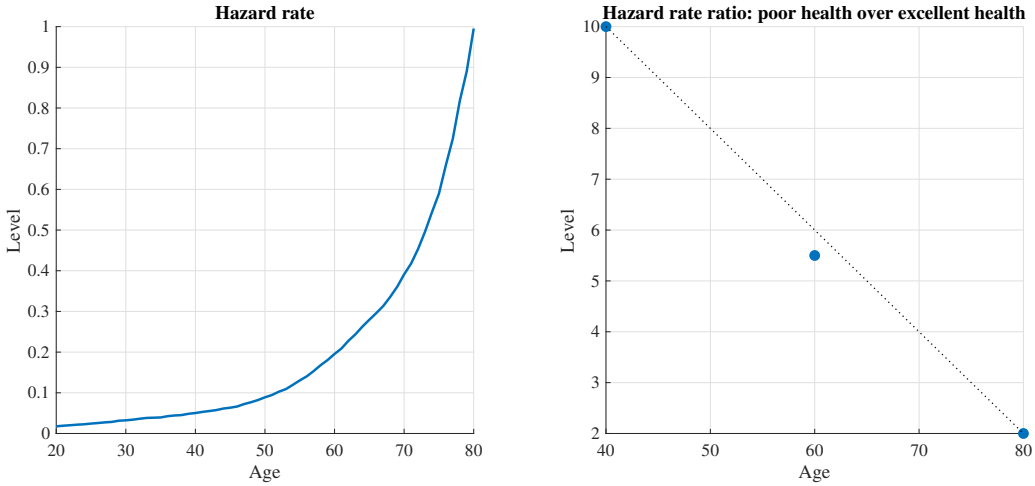
which, despite its simplicity, help us match the two empirical facts just described: (i) income follows a hump-shaped trajectory over age, and (ii) poor health reduces income.[6] The coefficients μ_i are estimated by ordinary least squares (OLS). Figure 1 plots health deficits and incomes by age for individuals in good and bad health. To estimate the equation, we pool observations from both groups and run OLS, obtaining $\mu_0^* \approx 3$, $\mu_1^* \approx 1$, and $\mu_2^* \approx -20$. All coefficients are statistically different from zero at the 5% level. The fit is satisfactory ($R^2 \approx 0.7$), and the signs of the partial derivatives are as expected: $y_t(t, d(t)) > 0$ and $y_d(t, d(t)) < 0$.

Conditional on age and health, agents differ in their permanent income component, μ_0, reflecting variations in productivity, work ethic, or access to education and professional networks. In the data, young individuals' income distribution has a long right tail, but extreme values pose numerical challenges: solving our model requires a nonlinear boundary value problem with an endogenous maximum admissible time, which does not always yield a solution. To address this, we assume individual i draws μ_0 from a uniform distribution, $\mu_0 \sim \mathcal{U}(\mu_0^* \pm \epsilon_y)$. We set $\epsilon_y = 0.4$, the largest range that permits a solution. This choice ensures that asymmetries in the resulting age-at-death distribution stem from endogenous model mechanisms rather than imposed assumptions on initial income.

[5]The MEPS asks individuals to rank their health from excellent to poor, just like the NHIS. We classify individuals using the procedure described earlier.

[6]See Low and Pistaferri (2015) and De Nardi et al. (2024) for similar assumptions about how health status affects income. In Section 8, we moreover test two alternative representation: one with a quadratic term in t, and one with the payment of a PAYG pension after a given retirement age. We show that it does not modify our main results.

FIGURE 2. Mortality risk



Notes. The left panel displays the unconditional hazard rate constructed from the 2022 HMD. The right panel shows the ratio of the hazard rate for those in poor health to the hazard rate for those in excellent health, approximated from the data in Cho et al. (2022).

4.2.5. *Hazard rate,* $\lambda(t, d(t))$. Section 3 defined $\Lambda(t)$ as the probability that an individual is alive at time t. For an individual of age t, the probability of dying between age t and age $t + \mathrm{d}t$ is then given by

$$z(t) = \frac{\Lambda(t) - \Lambda(t + \mathrm{d}t)}{\Lambda(t)}.$$

Furthermore, this probability of death $z(t)$ relates to the hazard rate $\lambda(t)$ as

$$\lambda(t) = \frac{-\dot{\Lambda}(t)}{\Lambda(t)} \approx \frac{-(\Lambda(t + \mathrm{d}t) - \Lambda(t))}{\mathrm{d}t\ \Lambda(t)} = \frac{z(t)}{\mathrm{d}t}\,.$$

The 2022 Human Mortality Database (HMD) provides the probability of death for individuals aged 0 to 110 in the coming year. This means it provides $z(t)$ when $\mathrm{d}t$ represents one year, which in the model corresponds to $\mathrm{d}t = \frac{4.3}{85} \approx 0.05$, since $t = 4.3$ in the model represents a period of 85 years (i.e., 105 - 20 = 85). Therefore, we recover the hazard rate in the US in 2022 using the formula $\lambda(t) = z(t)/0.05$. The left panel in Figure 2 shows the results. As expected, the hazard rate rises monotonically with age, rising slowly at first before accelerating around 60 years.

The hazard rate constructed here is unconditional; it does not account for an individual's health. However, as Cho et al. (2022) document, individuals aged 40 years reporting poor health have a hazard rate roughly 10 times higher than those in excellent health, whereas

it is roughly 5.5 times higher at age 60 and 2 times higher at age 80 (see blue dots in right panel of Figure 2).

We specify the functional form for $\lambda(t, d(t))$ as

$$\lambda(t, d(t)) = \lambda_0 + \lambda_1 e^t + \lambda_2 d(t), \tag{8}$$

which, although highly stylized, helps us match the two empirical facts just described: (i) the hazard rate rises exponentially with age, and (ii) poor health raises the likelihood of death. We estimate the λ_i coefficients as follows. From the left panel of Figure 1, we retrieve the unconditional health deficit at ages 20 and 80, denoted by $d_u(20)$ and $d_u(80)$. The figure also reports the health deficits for individuals in good and bad health, classified as those reporting excellent, very good, or good health and those reporting fair or poor health, respectively. However, to align with Cho et al. (2022), we now focus on individuals reporting excellent or poor health. Using our dataset, we retrieve their health deficits at age 80, denoted by $d_e(80) \approx 0.1$ and $d_p(80) \approx 0.27$. Lastly, from the left panel of Figure 2, we obtain the unconditional hazard rate at ages 20 and 80, denoted by $\lambda_u(20)$ and $\lambda_u(80)$. The coefficients λ are determined by solving the linear system of equations.

$$\begin{aligned}
\lambda_0 + \lambda_1 + \lambda_2 d_u(20) &= \lambda_u(20), \\
\lambda_0 + \lambda_1 e^{(80-20)\times 4.3/85} + \lambda_2 d_u(80) &= \lambda_u(80), \\
\lambda_0 + \lambda_1 e^{(80-20)\times 4.3/85} + \lambda_2 d_p(80) &= 2\left[\lambda_0 + \lambda_1 e^{(80-20)\times 4.3/85} + \lambda_2 d_e(80)\right].
\end{aligned}$$

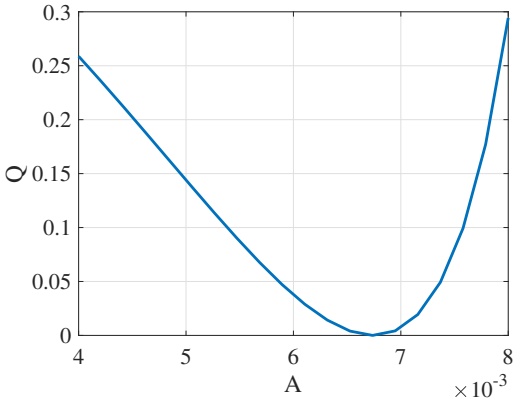
In words, the first and second equations represent the unconditional hazard rate at ages 20 and 80, respectively, while the third equation requires that at age 80, the hazard rate for an individual in poor health be twice that of an individual in excellent health. The resulting parameter values are $\lambda_0 \approx -0.08$, $\lambda_1 \approx 0.02$ and $\lambda_2 \approx 3.8$. As expected, $\lambda_t(t, d(t))$ and $\lambda_d(t, d(t))$ are positive. Moreover, the functional form implies that at age 60, individuals in poor health face a hazard rate about four times higher than those in excellent health, somewhat below the 5.5-fold difference reported by Cho et al. (2022).

4.3. **Parameters calibrated within the model.**

4.3.1. *Monetary cost of health deficit, B.* Data from the 2022 MEPS show that median medical expenditures as a share of income remain relatively stable at around 3–4%, before rising sharply after age 60 and reaching about 20% by age 80. Extrapolating the series with a quadratic time trend suggests that by age 105, medical expenditures amount to roughly 50% of income. This steep increase is consistent with De Nardi et al. (2016), who find that medical expenses more than double between ages 70 and 90 and that average expenditures for Americans aged 65 and older are 2.6 times the national average.

In our setup, medical expenditures are divided between preventive care, h, and the monetary cost of health deficit, Bd. Recall that the latter can be viewed as long-term care expenditures,

FIGURE 3. Grid search and model non-targeted moments



	Data	Model
Mean age at death	77	76
Dispersion age at death	17	19
Skewness age at death	-1.5	-0.5
$P(T > 75 \mid T > 50)$	0.7	0.6

Notes. The left panel shows the objective function $Q(A)$ as a function of A, while the right panel compares the model-implied distribution of age at death with the 2022 US data from actuarial tables in the HMD.

as well as curative or palliative care. Importantly, at the maximum admissible age, $\bar{T}$, there is no incentive to spend on preventive care, as death is imminent, and all medical expenditures are the monetary cost of health deficit.[7] In addition, at that moment, the individual's health deficit is $\bar{d}$ (see boundary conditions in subsection 3.1). Therefore, at time $\bar{T}$, the ratio medical expenditures to income equals $\frac{\theta B \bar{d}}{y(\bar{T},\bar{d})} = 0.5$. Since we consider 105 years as the maximum admissible age, we set

$$B = \frac{y(\bar{T}, \bar{d} | \mu_0 = \mu_0^*)}{2\theta \bar{d}} \approx 1.9,$$

where $y(\bar{T}, \bar{d} \mid \mu_0 = \mu_0^*)$ denotes the income at the maximum admissible age of the median agent.

4.3.2. *Efficiency of preventive care, A.* We calibrate the remaining parameter, A, using a simple grid search over a predefined range. The goal is to ensure that, for the median agent ($\mu_0 = \mu_0^*$), the condition $\bar{T} = 4.36$ (see Subsection 4.2.1) holds. Our grid search method then minimises the objective function

$$Q(A) = (\bar{T}(A) - 4.36)^2,$$

for the median agent. The left panel in Figure 3 plots the objective function against A, showing a distinct U-shape. The local minimum occurs at $A = 0.0067$, which we set as our baseline calibration for A.

[7]Our model's implication that, at the end of life, most medical expenditures are not related to preventive care is both intuitive and consistent with De Nardi et al. (2016), who argue that the majority of the increase in medical expenditures between ages 70 and 90 is driven by nursing home spending.

4.4. **Assessment.** To evaluate the model's performance on non-targeted criteria, we first examine key statistics of the stationary age-at-death distribution.[8] These statistics include the first three moments of the age-at-death distribution and the probability of surviving to age 75 conditional on being alive at 50. We compute these moments using a Monte Carlo simulation with 500,000 agents, each drawing a permanent income component $\mu_0 \sim \mathcal{U}(2.6, 3.4)$. As shown in the right panel of Figure 3, the model reproduces the age-at-death distribution well, although it underestimates the absolute value of the skewness.

We now study life-cycle dynamics from birth until the maximum lifespan, $\bar{T}$. The solid blue lines in Figure 4 trace the outcomes for agents with the median permanent income component, while the shaded areas span those between the 5th and 95th percentiles. For comparison, the dotted red lines depict the observed data. The top-left panel shows a health deficit that rises monotonically and convexly with age, consistent with the data. The model also generates a survival probability $\Lambda(t)$ that closely matches the observed pattern. Nevertheless, it underestimates mortality at very old ages, which accounts for the less pronounced negative skewness of the age-at-death distribution (right panel of Figure 3).

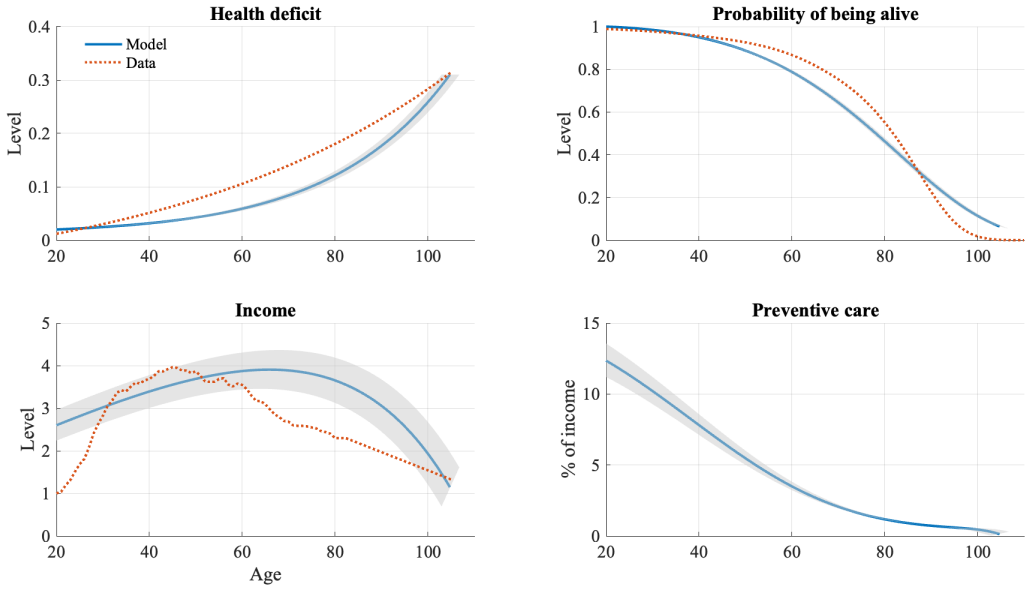
Turning to the bottom-left panel, income follows a hump-shaped pattern, similar to the data, although it peaks later in the model. As explained earlier, these inverted-U dynamics arise from two competing forces. On one hand, higher health deficits reduce income, capturing more complex dynamics such as involuntary unemployment, limitations on the type of work one can perform, or lower productivity. On the other hand, income increases with age, reflecting the general trend of rising earnings as individuals advance in their careers. Together, these forces result in income dynamics that are qualitatively close to the observed pattern.

In addition, the bottom-right panel shows the share of income spent on preventive care. In the model, preventive care declines with age because the health deficit is a stock that affects the remainder of an individual's life. A unit of preventive care at time t provides benefits over $[t, T]$, so as t approaches T, the available time to enjoy these benefits shortens, reducing the incentive to invest in preventive care. To our knowledge, no direct empirical counterpart of this series exists. However, data from the National Ambulatory Medical Care Survey indicate that preventive care as the primary reason for a visit declines with age. For instance, in 2019, preventive visits accounted for 40% of visits for children under 18, 22% for adults aged 18–64, and 13% for adults aged 65 and over (Ashman et al., 2023).

Lastly, we compare the share of income spent on medical care across the entire population in both the data and the model. Using data from the 2022 MEPS and the 2022 HMD, we find that the share of income spent on medical care across the entire US population was 6.5% in

[8]Recall that at each instant a new cohort of size 1 is born, resulting in a stationary population distribution.

FIGURE 4. Non-targeted life-cycle paths: data and model



Notes. Solid blue lines trace the outcomes for agents with the median permanent income component, while the shaded areas span those between the 5th and 95th percentiles. Dotted red lines depict the observed data. Refer to the corresponding subsection above for details on data sources. Since the time series for the health deficit and income extend only to 80 years in the data, we extrapolate them to 105 years using polynomial fits.

2022. In the model, the corresponding figure is 9%, which remains within a plausible range.[9]

5. Income-longevity gradient

The rich live longer. This section assesses whether our baseline model can match this income-longevity gradient in a quantitatively meaningful way. Unlike most models, our setup allows health and income to affect each other, so we also examine the effect of each channel separately by shutting them down one at a time.

5.1. **Income-longevity gradient in the baseline model.** Figure 5 plots the relationship between income and life expectancy at age 50. We simulate 500,000 agents, each drawing $\mu_0 \sim \mathcal{U}(2.6, 3.4)$ as described in Subsection 4.2.4. Among those alive at age 50, we sort

[9]The empirical share of 6.5% is considerably lower than the ratio of total health expenditures to GDP, 17.6%, reported in the 2023 National Health Expenditure Fact Sheet. This difference likely arises because survey data focus on individuals' direct expenditures on healthcare services, whereas the GDP-based measure includes broader spending such as government public health expenditures, hospital funding, and other indirect costs.

individuals into 25 income groups, compute life expectancy as the mean age at death within each group, and report the results as the solid blue line.

The curve is upward sloping: higher earners enjoy longer lives. This reflects the interaction of several mechanisms. First, there is a direct effect: higher income allows greater investment in preventive care, which lowers health deficits and, in turn, reduces mortality risk.[10] This direct channel is reinforced by indirect effects that compound over the life cycle: lower health deficits reduce medical expenditures Bd and increase income, which further supports preventive care. In addition, lower health deficits decrease the effective discount rate (see subsection 3.2), making individuals more patient, a pattern recently documented by De Nardi et al. (2024). More patient individuals invest more in preventive care, which slows the accumulation of health deficits and further boosts income.

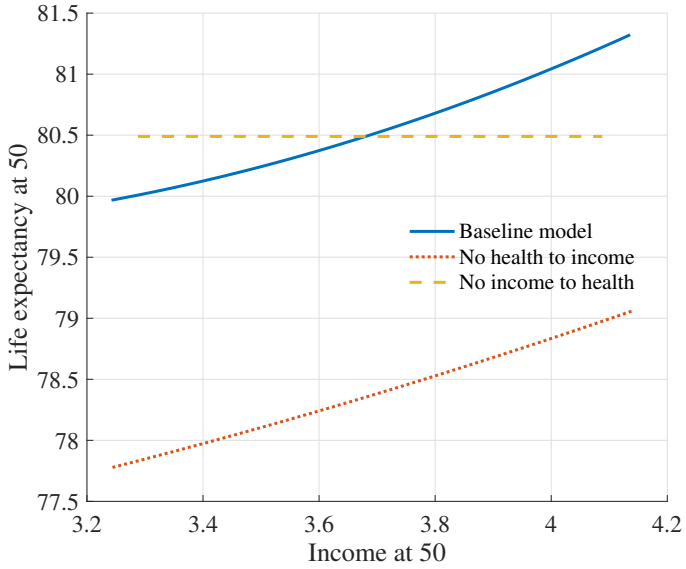
From a quantitative perspective, the blue line implies that a 10% increase in income at age 50 is associated with a gain in life expectancy of about six months. This matches the empirical estimates of Chetty et al. (2016), who find that in 2014, middle-aged American men in the second quartile of the income distribution (mean \$47k) had an expected age at death of roughly 81 years, compared with about 85 years for those in the third quartile (mean \$83k). Assuming a linear relationship between income and life expectancy – an assumption supported by further evidence in their study, and one that our model also reproduces – a 10% rise in income corresponds to an increase in life expectancy of about six months. In addition, the location of the curve is consistent with the data: according to the 2022 HMD, life expectancy at age 50 was about 80.5 years, which is what the model delivers.

5.2. **Income-longevity gradient with channels shut down.** Unlike much of the literature, our model features two channels: a *health-to-income channel*, since agents in poor health earn lower wages; and an *income-to-health channel*, since higher earners devote more resources to preventive care. Let us then assess how the income-longevity gradient changes when we shut down each channel in turn.

To shut down the health-to-income channel, we assume that income depends only on age and not on health. Specifically, we let income $y(t)$ follow a second-order polynomial in age. Because individuals differ in their permanent income component, the polynomial coefficients

[10]Recent empirical evidence supports this mechanism. For example, Kurani et al. (2020) show that individuals living in the least deprived census block groups in Minnesota, Iowa, and Wisconsin were about twice as likely to complete recommended cancer screenings compared to those in the most deprived groups. Bauer et al. (2022) report similar findings across the US, with counties of higher social vulnerability showing significantly lower odds of receiving recommended cancer screenings. French et al. (2019) further document that lower-income households purchase less healthful foods than higher-income households, even after adjusting for education, marital status, and race.

FIGURE 5. Income-longevity gradient at age 50



Notes. Link between income and life expectancy at age 50, based on a Monte Carlo simulation with 500,000 agents

vary across agents. We estimate them separately so that each individual's income at every age coincides with the level they had in the baseline model.

To shut down the income-to-health channel, we assume that preventive care depends only on age and not on income. In this case, we let preventive care $h(t)$ follow a fourth-order polynomial in age and choose the coefficients so that preventive care at each age matches the path of the median agent in the baseline model. As a result, all individuals within a cohort invest the same amount in preventive care, irrespective of their income.

The dotted red line in Figure 5 shows the income-longevity gradient at age 50 when the health-to-income channel is shut down. The curve remains upward sloping, since high earners still invest more in preventive care and thus live longer. But it shifts downward, because removing the effect of health on income reduces incentives to invest in preventive care, increasing mortality risk across the population. Quantitatively, life expectancy at 50 for the median agent falls to 78.4 years, compared with 80.5 in the baseline model and in the data.

In turn, the dashed yellow line shows the gradient when the income-to-health channel is shut down. The curve flattens, since all agents now consume the same amount of preventive care regardless of their income. As a result, life expectancy is identical across the population and coincides with that of the median agent in the baseline model, 80.5 years. These results

thus resemble the standard Yaari (1965) setup, where differences in health or income do not generate a longevity gradient.

In sum, both channels affect either the level or the slope of the income-longevity gradient, hinting at the relevance of considering both when evaluating the effects of government policy. We turn to this next.

6. Income redistribution

Recent empirical studies have reaffirmed the strong correlation between wealth and longevity (see e.g., Himmelstein et al., 2024; Machado et al., 2025; Gugushvili and Wiborg, 2025). It is rare for these studies not to conclude with a policy recommendation advocating wealth redistribution, suggesting that reducing wealth inequality would lead to more equal longevity outcomes and increase population longevity. This section casts doubt on this type of claim by studying how redistribution policies shape mortality patterns and welfare in our theoretical setup.

6.1. **Setup.** Let the government provide lump-sum transfers ω. To balance its budget, it finances these expenditures through a proportional earnings tax τ. These two instruments apply uniformly to all individuals, regardless of age, income, or health. As a result, individuals' budget constraints (3) now become

$$c(t) + \theta\left[h(t) + Bd(t)\right] = (1-\tau)y(t, d(t)) + \omega. \tag{9}$$

From this point on, we refer to $y(t, d(t))$ as earnings, and to $(1-\tau)y(t, d(t)) + \omega$ as total net income or disposable income. Up to now, we have used these terms interchangeably, since they were identical in the absence of government intervention. This equivalence no longer holds once policy is introduced.

Any government action redistributes resources both within and across cohorts. Across cohorts, redistribution occurs because earnings follow a hump-shaped pattern – rising early in life, peaking in middle age, and declining with age. As a result, a proportional income tax shifts resources from middle-aged individuals to both younger and older individuals. Within cohorts, redistribution occurs as wealthier individuals contribute more to government revenues, supporting lower-income individuals through transfers. To compute the government budget constraint, we aggregate over all individuals. Each individual is characterized by age t, and initial permanent income component μ_0. Any individual variable $x \in \{c, h, d, y, \lambda, \Lambda\}$ can then be expressed as $x(t, \mu_0)$ and aggregated as

$$X = \underbrace{\int_{\mu_0^*-\epsilon_y}^{\mu_0^*+\epsilon_y} \underbrace{\int_0^{\bar{T}(\mu_0)} x(t,\mu_0)\Lambda(t,\mu_0)\,\mathrm{d}t\,\mathcal{U}_{\left[\mu_0^*\pm\epsilon_y\right]}}_{\text{Age dimension}}\,\mathrm{d}\mu_0}_{\text{Income dimension}}. \tag{10}$$

Here $\mathcal{U}_{[\mu_0^* \pm \epsilon_y]}$ is the uniform distribution governing μ_0, while $\bar{T}(\mu_0)$ is the maximum admissible age of an individual with permanent income component μ_0. Aggregating the linear equation equation (9) yields the government budget constraint

$$\omega P = \tau Y, \tag{11}$$

where P is the size of the population, obtained from equation (10) with 1, the size of each newborn generation, instead of $x(t, \mu_0)$. Therefore, the left-hand side represents government expenditures and the right-hand side represents total revenues. Given any lump-sum transfer ω, the government chooses τ to satisfy equation (11).

6.2. **Redistribution in the baseline model.** Let us set lump-sum transfers at $\omega = 1.7$, which requires a round 50% income tax ($\tau = 0.50$) to balance the government budget. The logic of the insights that follow applies to any calibration. The first two rows of Panel A in Table 4 report how the first two moments of the age-at-death and disposable income distributions respond to income redistribution. The table also reports welfare across the population, computed as

$$W = \int_{\mu_0^* - \epsilon_y}^{\mu_0^* + \epsilon_y} \int_0^{\bar{T}(\mu_0)} \Lambda(t, \mu_0) \left[\ln c(t, \mu_0) + \alpha\right] \, \mathrm{d}t \, \mathcal{U}_{[\mu_0^* \pm \epsilon_y]} \, \mathrm{d}\mu_0.$$

This welfare measure corresponds to that of a benevolent central planner who weights all individuals equally, regardless of income or age.

Three insights emerge from the first two rows of Panel A First, income redistribution *lowers* average longevity. It weakens the link between health and disposable income – an income tax of 100% would remove it entirely. This reduces the incentive to invest in preventive care, raising mortality risk.[11] This result stands in sharp contrast to recent work based on statistical models, which finds that perfect wealth equality would raise US median longevity by 2.2 years (Himmelstein et al., 2024). Yet statistical models are subject to the Lucas critique: they rely on historical patterns that may no longer hold once policy changes alter individual incentives. This distinction is crucial here, since redistribution alters incentives and choices, and with them, life expectancy. Our structural model, immune to this critique, shows that the consequences of redistribution for longevity can be starkly different.

Second, income redistribution reduces disparities in life expectancy, lowering the standard deviation of age at death by nearly five months. High earners cut preventive care more sharply, as both incentives and disposable income fall. For poorer individuals, higher transfers partly

[11] This mechanism mirrors the one described in the previous section when we studied the effect of shutting down the health-to-income channel on the income-longevity gradient.

TABLE 4. The effects of government intervention

	Age at death		Disposable income		Δ Welfare (%)
	Mean	Stand. Dev.	Mean	Stand. Dev	
Panel A: baseline model					
No policy	76.0	19.4	3.49	0.52	-
Income redistribution	75.0	19.0	3.33	0.24	-1.72
Subsidy to prevention	76.9	19.6	3.46	0.54	1.66
Panel B: no health-to-income					
No policy	73.5	18.7	3.43	0.50	-
Income redistribution	73.8	18.7	3.43	0.25	0.87
Panel C: no income-to-health					
No policy	75.9	19.3	3.41	0.52	-
Income redistribution	75.9	19.3	3.41	0.26	0.84

Notes. No policy refers to the baseline scenario with $\omega = \tau = 0$. The income redistribution scenario sets $\omega = 1.7$, with the income tax τ equal to 0.50 in Panel A, 0.48 in Panel B, and 0.49 in Panel C. Disposable income is defined as $(1-\tau)y(t, d(t)) + \omega$. The last column reports the welfare gain or loss (in percent) of the income redistribution scenario relative to the no policy scenario.

offset weaker incentives, limiting their decline in preventive care. That redistribution reduces disparities in longevity is consistent with Himmelstein et al. (2024).

Third, income redistribution lowers average disposable income. As noted earlier, reduced preventive care accelerates health deficit accumulation, which in turn reduces earnings across the entire population. It also decreases inequality in disposable income by construction. Qualitatively, this aligns with recent evidence suggesting that a $1000 monthly universal basic income in the U.S. would reduce employment and output, but lower disposable income inequality (Luduvice, 2024).

These insights highlight that the common belief that redistribution will lead to a healthier population and longer lives need not hold. When income relates to health – as it almost certainly does – redistribution can weaken the incentives to invest in health, which in turn shapes the distribution of age at death. Our analysis also points to a tradeoff between reducing disparities in income and longevity and raising their averages. To examine this, the last column of the table reports the welfare gain or loss (in percent) from income redistribution relative to the no-policy baseline. Redistribution lowers welfare by 1.7%, indicating that the costs of lower averages outweigh the benefits of reduced disparities.

6.3. **Redistribution with channels shut down.** Let us now illustrate why accounting for both the health-to-income and income-to-health channels matters when evaluating government intervention. We first shut down the health-to-income channel, so that income depends

only on age and not on health (see subsection 5.2). With lump-sum transfers set at $\omega = 1.7$ as before, the required income tax to balance the government budget is now $\tau = 0.48$.

In Panel B of Table 4, average longevity *increases* under income redistribution. Without the health-to-income channel, redistribution no longer weakens the link between health and income – because there is none. As a result, the incentives to invest in preventive care (i.e. longer lives and lower monetary costs of health deficits) remain unaffected. Redistribution now simply moves resources from high earners to low earners, allowing the latter to invest more in their health. Overall, this raises mean longevity by almost four months.

This pattern is closer to the results suggested by the statistical models discussed earlier. This is not surprising: without the health-to-income channel, we create a data generating process in which individuals no longer face incentives linking current health choices to future earnings. In this case, statistical models that abstract from agents' optimization produce predictions that align more closely with the outcome.

All other statistics in Panel B behave as expected. Mean income remains unchanged, since it now depends only on age and not on health, while income inequality is cut in half. More importantly, income redistribution now raises welfare by 0.9%. This is unsurprising: without the health-to-income channel, there is no tradeoff between reducing disparities and raising averages.

We now shut down the income-to-health channel, so that preventive care depends only on age and not on income. As explained above, in this scenario $h(t)$ follows a polynomial in age, with coefficients chosen so that preventive care at each age matches the path of the median agent in the baseline model without policy. With lump-sum transfers set at $\omega = 1.7$, the required income tax to balance the government budget is $\tau = 0.49$.

As noted earlier, this version of the model resembles a standard Yaari (1965) setup, where mortality is an exogenous random variable. Moreover, the deterministic preventive care path renders average disposable income independent of policy. As a result, income redistribution only reduces disposable income inequality, which fully accounts for the welfare gains reported in the last column of Panel C.

All told, two insights emerge. First, the effects of income redistribution on longevity in our structural model differ sharply from those suggested by recent statistical analyses. Second, it is important to account for both the income-to-health and health-to-income channels when evaluating government policy. In our example, shutting down either channel generates welfare gains from redistribution. Yet in the full model, where both channels are active, redistribution lowers welfare.

7. Subsidy to preventive care

A natural follow-up question is which government intervention in our structural setup could shape agents' incentives to extend longevity in the way redistribution appears to do in statistical models. The most plausible candidate is a policy that lowers the relative price of preventive care, thereby encouraging its use and fostering a healthier population – in other words, a subsidy to preventive care.

7.1. **Setup.** Let the government reimburse a share s_h of preventive care investment. As before, expenditures are financed through a proportional earnings tax τ. The individual budget constraint becomes

$$c(t) + \theta\left[(1 - s_h)h(t) + Bd(t)\right] = (1 - \tau)y(t, d(t)).$$

Applying the same procedure as in the previous section yields the government budget constraint

$$s_h \theta H = \tau Y,$$

where H denotes total preventive care across the population. For any given subsidy s_h, the government sets τ to balance its budget.

7.2. **Numerical illustration.** Let us consider a 10% preventive care subsidy in the baseline model, requiring a 0.7% income tax to balance the government's budget.[12] The third row of Panel A in Table 4 shows a positive impact on the age-at-death distribution: the mean rises, while the standard deviation increases only slightly, lowering the coefficient of variation. By reducing the cost of preventive care, subsidies make it more attractive, prompting individuals to allocate a larger share of their income to it. This slows the accumulation of health deficits across the population and reduces mortality risk.

Qualitatively, this finding aligns with recent empirical evidence on the Affordable Care Act, which expanded access to affordable coverage (Sullivan et al., 2024). This expansion reduced mortality, with the strongest effects for causes of death amenable to medical care (Borgschulte and Vogler, 2020; Miller et al., 2021).

Turning to the impact on disposable income, two opposing forces are at play. On one hand, a healthier population earns higher wages, which raises disposable income. On the other hand, the government levies taxes on earnings to finance the subsidy. Overall, these two effects nearly offset each other, leaving both the mean and the standard deviation of the disposable income distribution essentially unchanged. Lastly, with improved mortality outcomes and an

[12]The degree of government intervention differs between the health subsidy and income redistribution scenarios. This does not affect our analysis, as the focus lies on their qualitative implications.

almost unchanged disposable income, the preventive care subsidy raises welfare by roughly 1.7%.

In sum, in our structural model, which captures how agents adjust their behavior in response to changes in the environment, lowering the relative price of preventive care generates shifts in mortality patterns similar to those recently attributed to redistribution measures in statistical models (see e.g., Himmelstein et al., 2024; Gugushvili and Wiborg, 2025).

8. Robustness and sensitivity analyzes

We now explore the robustness of our results to alternative strategies for mapping the model to the data and to model extensions. In subsection 8.1, we introduce a risk-free asset, allowing agents to transfer resources safely over time. In subsection 8.2, we incorporate retirement, with individuals receiving a pension financed through a pay-as-you-go system. Lastly, in subsection 8.3, we study alternative specifications for the income process $y(t, d(t))$.

8.1. **Risk-free asset.** Our baseline model abstracts from saving instruments for mathematical tractability. This assumption may not be as restrictive as it appears: many households hold little wealth. For instance, Aguiar et al. (2024) find that 40% of US households live hand-to-mouth, and that this behaviour persists over time, with many remaining so for years. Nonetheless, we now relax the no-savings assumption by introducing a risk-free asset that allows agents to transfer resources safely over time. Appendix B provides technical details; here we discuss the key insights.

Let us consider a small open economy in which foreign agents meet domestic saving demand at a fixed interest rate r. We set this rate equal to the discount rate, $r = \rho$, as is standard in most macroeconomic models. As a result, individuals optimally smooth consumption over their life cycle, achieving constant utility flows.

In addition, individuals borrow early in life against future earnings to finance higher preventive care. Greater investment in preventive care improves their health trajectory, leading to higher future earnings, lower medical costs, and reduced mortality risk across the population. As a result, the mean age at death is nearly 10 months higher than in the baseline model (76.8 versus 76 years), while the standard deviation remains virtually unchanged at 19.4 years.

Access to a risk-free asset then affects longevity in much the same way as the preventive care subsidy examined in Subsection 7. Both mechanisms transfer resources from the old to the young – through borrowing against future earnings in one case and taxation in the other – to support higher preventive care early in life. In this sense, the model suggests that health

subsidies could help offset the adverse effects of borrowing constraints faced by a significant share of the US population, particularly among younger adults.[13] Lastly, allowing for savings does not alter our main conclusions about income redistribution, since the key mechanism at play, namely a weaker incentive for prevention, remains the same.

8.2. **Pay-As-You-Go (PAYG) pension scheme.** In the baseline model, individuals supply one unit of labor inelastically throughout their lives and earn a real wage equal to their productivity. In reality, most people retire before death; in the US, for example, the effective retirement age is around 65 years. To capture this, we now assume that individuals work inelastically until a fixed retirement age and then receive a constant pension, independent of their past earnings. Pensions are financed by a proportional tax on labor income.

As before, we report technical details in Appendix C and focus here on the key insights. Unsurprisingly, the PAYG scheme affects mortality as income redistribution does. Both mechanisms weaken the link between health and earnings: through retirement in the PAYG scheme and through taxation in income redistribution. This weaker link reduces incentives for preventive care during youth, increasing mortality risk later in life. For instance, assuming a retirement age of 65 years and a replacement rate of 55% for the median agent lowers the mean age at death by over two years compared with the baseline model (73.8 vs 76 years).

Two remarks are in order before concluding. First, if pensions were tied to past earnings rather than being constant, the PAYG setup would move closer to the baseline model, since retirement income would then indirectly depend on past health choices. Second, the PAYG scheme does not alter our main conclusions regarding government intervention.

8.3. **Alternative specification for income process.** Our baseline setup assumes that the decline in income from a certain age onward is entirely due to worsening health (see equation (7)). In reality, part of this decline may reflect age-related factors unrelated to health, such as reducing work hours to care for family or lower work motivation. Thus, the baseline model represents one end of the spectrum. At the other end, when we shut

[13] In addition to the previously mentioned statistic that 40% of households are hand-to-mouth, data from the 2023 Survey of Household Economics and Decisionmaking (SHED) conducted by the Federal Reserve Board indicate that younger adults face greater difficulties accessing financial services. They are more likely to be unbanked, meaning they do not hold a checking, savings, or money market account, and less likely to have a credit card. They also experience worse credit outcomes, with higher rates of denial and credit rationing.

down the health-to-income channel, we assume that the decline in income is entirely age-driven. Understanding the model's behavior at both ends already provides insight into its performance in intermediate cases.

The question then becomes: if both forces operated simultaneously – worsening health and age-related factors reducing earnings later in life – would the outcome resemble the baseline setup or the scenario without the health-to-income channel? To explore this, we slightly modify the functional form for income, setting

$$y(t, d(t)) = \chi_0 + \chi_1 t + \chi_2 t^2 + \chi_3 d(t).$$

As in subsection 4.2.4, the coefficients χ_i are estimated by OLS using the data in Figure 1. We obtain $\chi_0 \approx 1.7$, $\chi_1 \approx 2.3$, $\chi_2 \approx -0.3$ and $\chi_3 \approx -19$. The coefficient on the health deficit variable, while slightly smaller than in the baseline calibration (≈ -20), remains large. This suggests that a model in which both age and health contribute to lower earnings later in life would still behave similarly to the baseline setup.

A point here deserves further comment. Since age and the health deficit are correlated, one might raise multicollinearity concerns. To assess this, we compute the variance inflation factor for each regressor. For the health deficit, this factor is 2.7, which is moderate, suggesting that the coefficient on the health deficit remains interpretable and its estimated magnitude should not be substantially distorted. To provide additional robustness, we also estimate the coefficients χ_i using ridge regression, a standard approach to address multicollinearity.[14] The coefficient on the health deficit remains large: under a ridge penalty of 1, it is very close to the OLS estimate; a penalty of 5 reduces it by about 3 units (from 19 to 16); and even with a high penalty of 10, the coefficient decreases by only 5.4 units. In all cases, the estimated effect of the health deficit remains strong.

9. Concluding remarks

Our model provides a simple yet quantitatively rich framework that reproduces the income-longevity gradient documented by Chetty et al. (2016). It also calls into question the view, based on statistical models, that redistributing income or wealth would necessarily extend life expectancy across the population. Because our structural setup captures how government intervention shapes incentives and choices, it avoids the limitations of purely statistical approaches subject to the Lucas critique. The analysis shows that redistribution can, in fact, worsen mortality outcomes by weakening the link between health and disposable income, thereby reducing incentives to invest in preventive care. Statistical models may thus offer an overly optimistic view of redistribution's effects. By contrast, our results indicate that

[14] Ridge regression is a regularization technique that involves penalizing high-value coefficients, thereby decreasing the impact of multicollinear predictors on the model's output.

lowering the price of preventive care through subsidies encourages higher preventive investment and reduces mortality risk. The resulting shift in the age-at-death distribution closely mirrors that which statistical models attribute to redistribution.

For mathematical tractability, our setup relies on several simplifying assumptions; relaxing two of them offers promising directions for future research. First, the model abstracts from morbidity risk: the probability that individuals develop severe diseases that sharply raise medical costs and reduce labor productivity. This could be incorporated by introducing a hazard rate governing the onset of such conditions, dependent on both age and the health deficit. Accounting for morbidity risk would naturally introduce a role for health insurance markets, providing a richer framework to study the income-health gradient and the effects of policy. Second, the model assumes that agents supply one unit of labor inelastically. Allowing labor supply to be endogenous would open new channels through which government policy affects the macroeconomy. Policies that extend life expectancy, such as preventive care subsidies, could increase labor supply among older individuals by lengthening their planning horizon. This would, in turn, generate general equilibrium effects, as changes in labor supply influence government revenues and the tax rate required to balance the budget.

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Appendix A. Representative firm

We have a distribution of individuals differing in

- age t,
- permanent component of productivity $\mu_0 \sim \mathcal{U}(\hat{\mu}_0 - \epsilon_y, \hat{\mu}_0 + \epsilon_y)$.

Each individual is characterized by (t, μ_0), and supplies one unit of labor with labor productivity given by $y(t, d(t, \mu_0))$.

For each type of individual (t, μ_0), a representative firm demands $l^d(t, \mu_0)$ units of labor and pays a unit wage of $w(t, \mu_0)$. The firm runs a constant return to scale technology with labor as the only input, and can perfectly substitute between each type of labor. Its maximization program is

$$\begin{aligned} \max_{l^d(t,\mu_0)} \quad & \Big\{ \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_0^{\bar{T}(\mu_0)} y(t, d(t, \mu_0))\, l^d(t, \mu_0)\, \mathrm{d}t \ \tfrac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0 \\ - \quad & \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_0^{\bar{T}(\mu_0)} w(t, \mu_0)\, l^d(t, \mu_0)\, \mathrm{d}t \ \tfrac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0 \Big\}, \end{aligned}$$

where $\bar{T}(\mu_0)$ is the maximum admissible age for an individual with μ_0. It is implicitly given by $d(\bar{T}, \mu_0) = \bar{d}$. The solution to the maximization problem is $w(t, \mu_0) = y(t, d(t, \mu_0))$ and the profit of the firm is zero.

Equilibrium on the competitive labor market requires that for each type (t, μ_0) of individuals, labor demand is equal to labor supply

$$l^d(t, \mu_0) = 1 \times \Lambda(t, \mu_0)\,.$$

The right-hand side implies that each individual supplies one unit of labor and that there is a mass $\Lambda(t, \mu_0)$ of individuals of type (t, μ_0), which is given by

$$\Lambda(t, \mu_0) = \mathrm{e}^{-\int_0^t \lambda(u, d(u, \mu_0))\mathrm{d}u}\,.$$

Here $\lambda(t, d(t, \mu_0))$ is the hazard rate, as defined in equation (8). Finally, total production is

$$Y = \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_0^{\bar{T}(\mu_0)} y(t, d(t, \mu_0))\, \Lambda(t, \mu_0)\, \mathrm{d}t\ \frac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0\,.$$

Appendix B. Model with a risk-free asset

This Appendix extends the baseline model with a risk-free asset that allows agents to transfer resources safely over time. For simplicity, we consider a small open economy where foreign agents meet domestic savings demand at a fixed interest rate r.

Denote by $s(t)$ an agent's asset holdings at age t. Assuming no assets at birth, $s(t)$ follows

$$\dot{s}(t) = i(t)\,, \tag{12}$$

$$s(0) = 0\,. \tag{13}$$

Here $i(t)$ is positive if the individual saves and negative if she borrows. The budget constraint (3) becomes

$$c(t) + i(t) + \theta(h(t) + Bd(t)) = y(t, d(t)) + rs(t) + z(t)\,.$$

Due to the uncertainty surrounding the agent's age at death, she might pass away with positive asset holdings ($s(T) > 0$) or debts ($s(T) < 0$). To tackle this issue, we introduce an annuity market as in Yaari (1965) or Blanchard (1985). More precisely, an insurance firm pays $z(s(t))$ to the individual at each instant. In return, the firm collects all assets when the individual dies. The running profit of this insurance firm is then

$$\pi(t) = \lambda(d(t))s(t) - z(t)\,.$$

Free entry in the insurance market ensures zero profits, and hence, $z(t) = \lambda(d(t))s(t)$. The individual's budget constraint becomes

$$c(t) + i(t) + \theta(h(t) + Bd(t)) = y(t, d(t)) + (r + \lambda(d(t)))\, s(t)\,. \tag{14}$$

Lastly, if the agent reaches the maximum lifespan, her assets must be zero, $s(\bar{T}) = 0$. This holds because we abstract from warm-glow preferences, where agents derive utility from the warm glow of their bequests to newborns. For brevity, we do not derive the optimality conditions for the extended model, as they result from the same methodology used in the baseline case.

Appendix C. Model with PAYG pension scheme

This Appendix extends the baseline model with a PAYG pension scheme. Let us assume that individuals supply one unit of labor inelastically until age $\hat{t}$ and none afterward. Labor earnings until age $\hat{t}$ is $y(t, d(t))$, as specified by equation (7). Once retired, individuals receive a pension $k > 0$, which, for simplicity, is independent of past labor earnings. Total pre-tax income then evolves by

$$y^T(t, d(t)) = \begin{cases} y(t, d(t)), & \text{if } t < \hat{t} \\ k, & \text{if } t \geq \hat{t}. \end{cases}$$

Letting $\hat{t} \to \bar{T}$ restores the baseline model.[15] The government budget constraint is

$$kP_{>\hat{t}} = \tau Y_{<\hat{t}}, \tag{15}$$

[15]This labor earnings profile introduces a discontinuity at age $\hat{t}$, which is not compatible with our optimal control framework, as it requires differentiability everywhere. To address this, we approximate this step function using a logistic function with a high steepness parameter. This ensures smooth transitions while closely mimicking the intended time profile. Visually, the logistic approximation is almost indistinguishable from the original step function, preserving the intended economic effects without introducing mathematical inconsistencies.

where $P_{>\hat{t}}$ denotes the population above age $\hat{t}$, and $Y_{<\hat{t}}$ represents the total labor earnings of individuals below that age. This formulation corresponds to a PAYG pension system, in which current workers' contributions finance the benefits received by current retirees.

The numerical example in the main text sets the effective retirement age to 65 years, roughly in line with US observations, corresponding to $\hat{t} = (65 - 20) \times 4.3/85$. It also sets $k = 1.7$, which requires a labor income tax rate of 17% to balance the government budget. The resulting pension replacement rate, defined as $\frac{k}{(1-\tau)y(\hat{t},d(\hat{t}))}$, is 55% for the median agent. This aligns with empirical evidence showing a net pension replacement rate in the US in 2022 slightly above 50%.[16]

[16]Organisation for Economic Co-operation and Development, OECD Data Archive, Net Pension Replacement Rates.

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