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WHEN HEALTH AFFECTS INCOME (AND VICE-VERSA): POLICY TRANSMISSION IN A HETEROGENEOUS AGENT LIFE-CYCLE MODEL

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Abstract. Both health and wealth are distributed heterogeneously across the population. These two dimensions are empirically linked by a robust positive correlation between income and life expectancy. Yet the mechanisms underlying this link and the implications for economic policy remain incompletely understood. This paper develops a life-cycle model with heterogeneous agents to explore the bidirectional relationship between income and health: higher income enables greater health investment, while better health enhances productivity and therefore earnings. We calibrate the model to U.S. data, capturing key empirical aspects of the distribution of income, health and age-at-death. We show that the income-to-health channel is more important early in life, while the health-to-income channel dominates at older ages. We then use this framework to evaluate policies aimed at redistribution or health. We find that income redistribution, while reducing inequality, weakens individuals' incentives to invest in health, lowering both average life expectancy and aggregate income. In contrast, health subsidies enhance health, raising both longevity and economic output, without reducing income inequality.

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

Keywords: Health, Income, Heterogeneity, Life-cycle model, Policies.

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Résumé non technique

L'évaluation stratégique 2025 de la BCE considère le vieillissement démographique comme un défi pour la conduite de la politique monétaire. En effet, le vieillissement a des effets sur la productivité, la croissance économique, les finances publiques et l'inflation. Ce papier se concentre sur le lien entre richesse et espérance de vie, qui est observable dans tous les pays et qui reste stable dans le temps. La richesse et la santé sont liées de plusieurs façons. La richesse peut améliorer la santé, par exemple en facilitant l'accès aux soins ou à une meilleure alimentation. Inversement, la santé influence la capacité à travailler et donc à gagner sa vie. D'autres facteurs, comme l'éducation ou les gènes, peuvent aussi jouer un rôle à la fois sur la santé et les revenus.

Mieux comprendre les liens entre revenus et santé est important pour évaluer correctement l'impact des impôts et de la redistribution sur la santé publique. De même, ces liens déterminent l'impact que peut avoir une politique de santé pour réduire les inégalités de revenu. Ces inégalités peuvent réduire l'efficacité de la politique monétaire, qui peut elle-même influencer les inégalités.

Dans ce papier, nous étudions la relation revenus-santé à l'aide d'un modèle de cycle de vie. En vieillissant, un individu voit son niveau de santé se détériorer progressivement : c'est ce que nous appelons le 'déficit de santé'. Ce déficit a trois effets négatifs : il coûte cher en soins (soins longue durée, traitements, etc.), il réduit la productivité et donc les salaires, et il augmente le risque de décès. Cependant, les individus peuvent ralentir ce déficit en investissant dans la prévention (alimentation saine, sport, vaccins, dépistage...).

Ainsi, notre modèle inclut un effet de la santé sur le revenu : une mauvaise santé réduit les salaires. Il inclut aussi l'effet inverse : les plus riches peuvent investir davantage dans la prévention. Bien que ces deux effets soient connus, la plupart des modèles existants n'en retiennent qu'un seul à la fois.

Notre modèle reproduit bien la corrélation entre revenu et espérance de vie qui est observée dans les données. Il suggère aussi que l'effet du revenu sur la santé est plus fort au début de la vie. À ce moment-là, les individus dont le revenu est plus élevé investissent davantage dans la prévention, restent en meilleure santé et gagnent plus par la suite. Plus tard, la santé devient plus inégale selon les décisions prises plus tôt, et influence davantage les revenus. Nous utilisons ensuite le modèle pour étudier deux types de politiques : des redistributions

de revenus (comme les aides forfaitaires) et des politiques ciblant la santé (comme les subventions à la prévention). Toutes sont financées par un impôt proportionnel sur le revenu du travail.

La subvention santé réduit le prix relatif de la prévention. Cela améliore la santé de tous, et donc la productivité, les salaires et l'espérance de vie. Ces effets vont dans le sens des données empiriques. Cependant, une subvention santé ne saurait pas diminuer les inégalités. Par contre, une politique de redistribution des revenus réduit non seulement les écarts de revenu, mais aussi les inégalités de santé. Par conséquent, elle décourage aussi la prévention. En effet, la redistribution affaiblit le lien entre santé et revenu, et donc réduit l'intérêt d'investir dans sa santé. Cela réduit le niveau de santé dans la population, ainsi que le niveau de revenu moyen et l'espérance de vie.

Notre recherche montre donc que des politiques de santé peuvent aussi impacter la situation économique des agents, et vice-versa.

1. Introduction

The rich live longer. This strong correlation between income and life expectancy persists over time and across countries (see e.g., Kitagawa and Hauser, 1973; Smith, 1998; Cutler et al., 2006, 2011). In the United States, for example, 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 limited to the gap between the very rich and the very poor; they follow a smooth gradient.

Income and life expectancy are linked through multiple channels (Deaton, 2002; Chandra and Vogl, 2010; Cutler et al., 2011; Baker and Stabile, 2012). For example, income could affect health by enabling access to better medical care and nutrition, while health might affect income by shaping one's ability to work and earn a higher wage. In addition, income and health might be correlated with other factors, such as education and habits, often inherited from university-educated and wealthy parents. In fact, all possibilities might be operating simultaneously.

Better understanding the relationship between income and health is important for the design of government policy. For instance, clarifying how income affects life expectancy is essential to assess whether taxes and income redistribution can promote public health. Likewise, understanding how health influences income is also key to evaluating the role of health policy in reducing income inequality.

We address these issues using 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 and are collectively referred to as the health deficit (Mitnitski et al., 2002). This health deficit is harmful for three reasons. First, it generates medical costs, including long-term care, curative treatments, and palliative services. Second, empirical evidence shows that the health deficit reduces labor productivity and therefore wages. Third, it increases the risk of death. Indeed, since biological age is malleable (see Scott, 2023, and references therein), we assume that individuals do not die from age itself, but because of declining health: the time of death is a random variable whose distribution depends on the individual's 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., screening).

Thus, our paper contributes to the tradition launched by the seminal contributions of Grossman (1972) and Ehrlich and Becker (1972), treating health as an investment good whose accumulation required effort and resources.

Our framework therefore features a *health-to-income channel*, as individuals in poorer health earn lower wages. In addition, it captures the reverse *income-to-health channel*, since wealthier individuals have more resources to invest in preventive care. While the existence of both channels is well-recognized, most structural models consider only one at a time, usually the health-to-income channel (see Section 2 below).

We proceed in two steps. First, we study a setup in which individuals are identical ex-ante and differ only ex-post due to the randomness of their time of death. We calibrate this 'median-individual' model to U.S. data, drawing primarily from the National Health Interview Survey and the Medical Expenditure Panel Survey, along with actuarial data from the Human Mortality Database. We validate this model by showing that it provides a good approximation of key patterns in mortality risk and income. However, because individuals are identical ex-ante, all t-year-olds earn the same income and face the same life expectancy. As a result, the model cannot replicate the smooth gradient between income and life expectancy documented in the data.

Therefore, in a second step, we introduce ex-ante heterogeneity in health and in income. First, individuals may differ in their initial health deficit, reflecting characteristics shaped by genetics or early-life conditions (see e.g., Deaton, 2002; De Nardi et al., 2024, for empirical evidence on the lasting impact of early-life factors on health and economic outcomes). Second, individuals may also differ in the permanent component of their labor productivity, capturing variations 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 earning differences over the life cycle).

We find that either source of ex-ante heterogeneity generates a meaningful quantitative relationship between income and life expectancy. Differences in initial health affect life expectancies but also income over the life cycle. Likewise, differences in initial productivity result in distinct income, health investments and hence life expectancies. Moreover, when these two sources of ex-ante heterogeneity are combined, the model suggests that a 10% increase in income at age 50 is associated with a rise in life expectancy of just over one and a half years. Because the model assumes rational expectations and includes no stochastic shocks aside from mortality, the resulting life-expectancy-income gradient is somewhat steeper than existing empirical estimates (Chetty et al., 2016).

In addition, our model supports the hypothesis put forward by Smith (1998) and Cutler et al. (2011) that the causal links between health and income might vary over the life cycle.

Specifically, we find that income has the greatest influence on health early in life, when the incentives to invest in preventive care are strongest. As a result, young and wealthy individuals invest more heavily in prevention, allowing them to maintain better health and earn higher incomes later on. In adulthood, however, health becomes more heterogeneous, reflecting earlier investment decisions, and thus plays a more central role in determining income.

We then use this framework to evaluate the effects of redistributive and health-targeted policies. As a health-targeted policy, we consider a subsidy for preventive care. As a redistributive policy, we examine income redistribution through lump-sum transfers – specifically, a universal basic income. Both policies apply uniformly across the population, regardless of age, health status, or income level. The government finances these expenditures through a proportional tax on labor income. Importantly, these two policies have opposing effects on the relationship between income and health.

More precisely, the health subsidy encourages greater preventive care in youth by reducing its relative price. This leads to a healthier population across all cohorts, as healthier individuals in youth translate into healthier individuals in old age. Hence, labor productivity, wages, and life expectancy increase for the entire population. These effects are intuitive and align with empirical findings 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). Although less is known about the impact of the Affordable Care Act on productivity and wealth, evidence suggests substantial improvements in financial well-being (Miller et al., 2021) and greater labor market flexibility for men with chronic conditions (Connolly et al., 2024).

Turning to the universal basic income, this reduces inequality in disposable income, particularly among older individuals, where health disparities contribute most to labor income gaps. 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). Our model adds a further insight: by compressing income inequality, a universal basic income also narrows the dispersion in preventive care during youth, thereby reducing the spread in age at death.

However, the universal basic income also discourages preventive care in youth. As noted earlier, one reason to invest in prevention is to boost labor productivity and, in turn, income. Since lump-sum transfers are unrelated to health and funded by a labor income tax, they weaken the link between health and income, reducing the incentive to invest in preventive care. This results in a less healthy population across all cohorts, with lower wages

and shorter life expectancy. Hence, our analysis echoes Deaton (2002) in challenging the widespread belief that one of the most effective means of reducing mortality is to eliminate social inequalities by redistributing income.

We conclude with two extensions. First, to keep the baseline model as simple as possible, we initially assumed the absence of any savings mechanism. We now relax this assumption by introducing a risk-free asset that allows agents to transfer resources over time. We show that access to a risk-free asset has similar implications for the income-health relationship as a preventive care subsidy. Both mechanisms encourage greater health investment at a younger age: the subsidy does so by lowering the relative price of preventive care, while the risk-free asset enables individuals to borrow against future income. Since roughly 40% of the U.S. population lives hand-to-mouth (Aguiar et al., 2024), often due to limited access to financial services (see Section 8), our model suggests that a health subsidy could help offset the adverse effects of such borrowing constraints.

Second, we examine an alternative to universal basic income: a redistributive policy implemented through a pay-as-you-go (PAYG) pension system. As expected, this policy qualitatively affects the income-health gradient in a manner similar to basic income. In both cases, individuals are less inclined to invest in preventive care, as these policies insulate old-age disposable income from the consequences of poor health, potentially leading to poorer health outcomes later in life.

The remainder of the paper is organized as follows. Section 2 reviews the literature. Section 3 lays out the continuous-time setup used for quantitative analysis. Section 4 describes our estimation and calibration procedure. Sections 5 and 6 present the main quantitative results, while Section 7 tests their robustness. Section 8 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 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 affect

TABLE 1. On the links between health 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. below the 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.

children's health, exploiting the fact 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 – help determine 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. 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 the identification of the causal links between health and income. Furthermore, these links may change with age (Smith, 1998; Cutler et al., 2011). Income 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 income. 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).

2.2. **Theoretical literature.** There is growing interest in models that incorporate health dynamics. For example, using an estimated life-cycle setup, De Nardi et al. (2024) show that individual losses from poor health are substantial and largely driven by factors determined early in life. Also worth noting, Capatina (2015) highlights how poor health contributes to income inequality primarily through lower productivity and lost time rather than higher medical expenditures. In turn, Hosseini et al. (2025) argue that disability programs are the main channel through which health inequality generates lifetime earnings inequality. Low and Pistaferri (2015) also study disability programs, evaluating their welfare implications by weighing incentive costs against insurance benefits. Unlike our work, these studies assume that health, and therefore survival probabilities, is exogenous, so they cannot assess the impact of policy interventions on health dynamics (Pashchenko, 2025).

Ozkan (2025) does allow individuals to affect the distribution of health shocks by investing in preventive health capital, thereby influencing their life expectancy. The author argues that public insurance, which covers large curative expenditures, widens the life expectancy gap by reducing the poor's incentives to invest in preventive health. However, unlike our

work, this study assumes that labor productivity, and therefore 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 and higher labor income – a mechanism consistent with our setup. Another study worth noting is by 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 significantly from ours in both their formulation and research question. For example, we follow Dalgaard and Strulik (2014) and Hosseini et al. (2022) by using a frailty index to measure health and treating it as a continuous variable rather than using a categorical variable (e.g., good vs. bad health). In addition, our analysis focuses on how different policy interventions – specifically, health subsidies and a universal basic income – affect the strength of the health-to-income and income-to-health channels, and how these channels shape the income-longevity gradient.

3. Baseline Continuous-time Life-cycle Model

In this section, we present our baseline continuous-time life-cycle model. For simplicity, this baseline setup assumes that all individuals are identical ex-ante and differ only in their time of death. This assumption helps map the model to the data. Section 5 will relax this assumption by introducing ex-ante heterogeneity in both income and health.

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 added value.

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 the firm maximization problem). 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 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

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

[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 $f(t)/(1 - F(t)) = \lambda(t, d(t))$.

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).

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

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

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 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

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

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

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

together with the three intratemporal conditions

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

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).

Unfortunately, there is no closed-form solution to this nonlinear boundary value problem. Therefore, after selecting all parameter values, the next section will solve it numerically using the collocation method proposed by Shampine et al. (2003).

3.3. **Stationary distribution.** So far, we have described the optimization problem for a single individual. However, as will become clear, calibrating the model requires considering the stationary population distribution. Hence, we assume that at each instant, a new cohort of size 1 is born, represented by $n(0) = 1$. Since in the baseline model all individuals are identical and face the same optimization problem, population dynamics are captured by $\dot{n}(t) = -\lambda(t, d(t))n(t) = \dot{\Lambda}(t)$. Thus, $\Lambda(t)$, which indicates the probability of a single

TABLE 2. Model parametrization

Parameter	Value	Description	Parameter	Value	Description
A priori chosen parameters					
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.

individual being alive at age t, also represents the size of the population aged t in the stationary equilibrium.

Therefore, the first moment of the distribution of age at death, referred to as life expectancy, is

$$\mu_T = \int_0^{\bar{T}} t\lambda(t, d(t))\Lambda(t)\ \mathrm{d}t + \bar{T}\left[1 - \int_0^{\bar{T}} \lambda(t, d(t))\Lambda(t)\mathrm{d}t\right].$$

The first term integrates over ages up to the maximum admissible age, weighting each age by its density, $\lambda(d(t))\Lambda(t)$. The second term adjusts for the probability of surviving to the maximum age, $\bar{T}$. Similarly, the variance of age at death is

$$\sigma_T^2 = \int_0^{\bar{T}} (t - \mu_T)^2\lambda(t, d(t))\Lambda(t)\ \mathrm{d}t + (\bar{T} - \mu_T)^2\left[1 - \int_0^{\bar{T}} \lambda(t, d(t))\Lambda(t)\mathrm{d}t\right].$$

Lastly, for future reference, the share of total resources spent on healthcare is

$$H = \frac{\theta \int_0^{\bar{T}} \left[h(t) + Bd(t)\right]\Lambda(t)\ \mathrm{d}t}{\int_0^{\bar{T}} y(t, d(t))\Lambda(t)\ \mathrm{d}t}.$$

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. We group the model parameters into three sets: a subset determined a priori, $\{\alpha, \beta, \theta\}$; a subset estimated directly from data, $\{\rho, d_0, \bar{d}, \gamma, \lambda(\cdot), y(\cdot)\}$; and a subset calibrated within the model to minimize the distance between data targets and model outcomes, $\{A, B\}$. Table 2 summarizes the calibration.

4.1. **A priori chosen parameters.** 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. 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, $\bar{T}$.[2] Assuming an annual time discount factor of 0.95 and normalizing $\rho = 1$, the following relationship must hold

$$e^{-\bar{T}} \approx 0.95^{(105-20)}.$$

However, $\bar{T}$ is determined as part of the model's solution rather than set as a fixed parameter. Therefore, we retain the above equation as a condition to match (i.e. $\bar{T} \approx 4.3$) when setting the third subset of parameters.

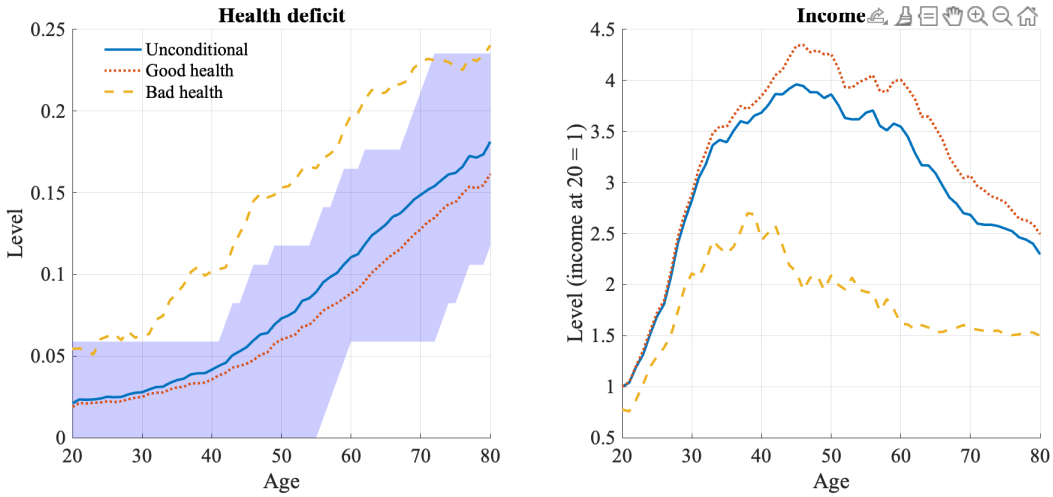
4.2.2. *Health deficit bounds, $\{d_0, \bar{d}\}$.* We follow Mitnitski et al. (2002) and Hosseini et al. (2022) and measure an individual's health status as her cumulative number of health problems. The index, here termed the health deficit, is defined as the ratio of a person's accumulated health issues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

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

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 poor 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.

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 poor health.[3] 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 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

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

[3]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.

yielding $\theta_0^* = -0.09$ $[-0.13, -0.05]$, $\theta_1^* = 0.07$ $[0.04, 0.11]$, and $\theta_2^* = 0.016$ $[0.01, 0.02]$ with 95% confidence intervals in brackets. 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 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

$$\dot{d}(t) = \gamma d(t) \Rightarrow d(t) = d_0 e^{\gamma t}.$$

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.

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.

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.[4] 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

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

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

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.[5] The coefficients μ are estimated by ordinary least squares (OLS). Figure 1 shows health deficits and incomes by age for individuals in good and bad health. To estimate the equation, we pool the series for both groups and run OLS, yielding $\mu_0^* = 2.76$ $[2.56, 2.96]$, $\mu_1^* = 0.94$ $[0.81, 1.07]$, and $\mu_2^* = -20.7$ $[-23.1, -18.3]$. The fit is satisfactory (R-squared ≈ 0.7), and as expected, $y_t(t, d(t)) > 0$ and $y_d(t, d(t)) < 0$ (see subsection 3.1 for the underlying logic).

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 $\mathrm{d}t = \frac{4.3}{85} = 0.05$, since $t = 4.3$ in the model represents a period of 85 (i.e., 105 - 20 = 85 years). 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 reporting bad health face a much higher risk of death compared to those reporting good health, and the difference in risk by health status is significantly greater among younger individuals than older ones. More precisely, the authors find that 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).

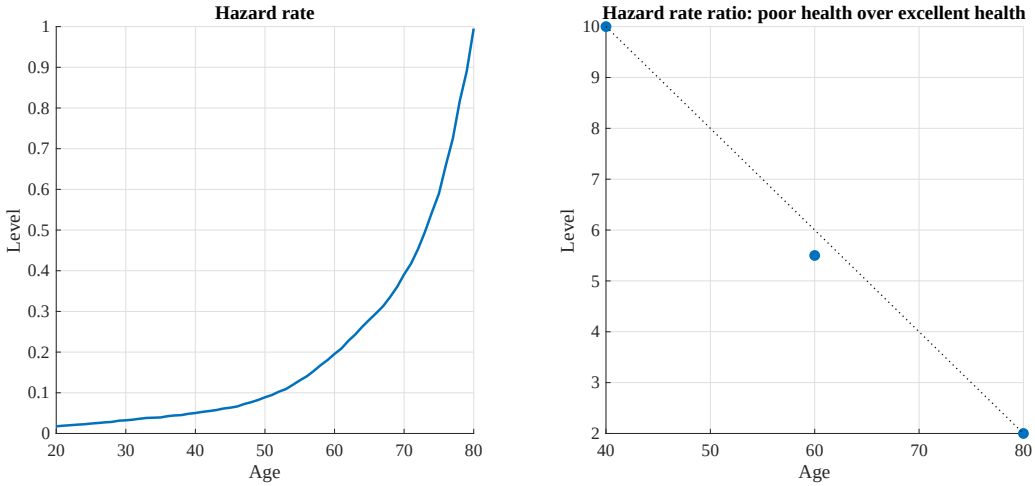
We specify the functional form for $\lambda(t, d(t))$ as

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

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.

[5] See Low and Pistaferri (2015) and De Nardi et al. (2024) for similar assumptions about how health status affects income.

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).

We estimate the λ 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.

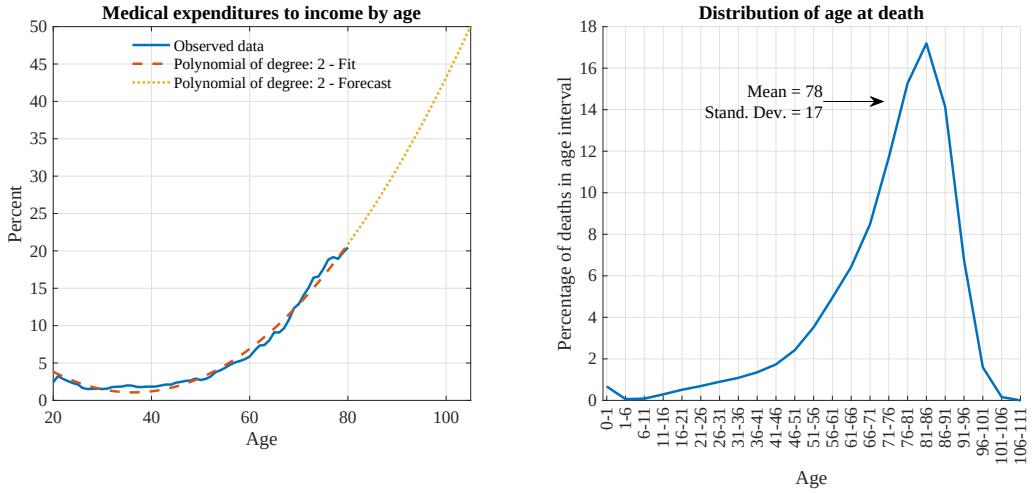
$$\begin{aligned}
\lambda_0 + \lambda_1 + \lambda_2 d_u(20) &= \lambda_u(20), \\
\lambda_0 + \lambda_1 e^{(80-20)\times 4.3/85} + \lambda_2 d_u(80) &= \lambda_u(80), \\
\lambda_0 + \lambda_1 e^{(80-20)\times 4.3/85} + \lambda_2 d_p(80) &= 2\left[\lambda_0 + \lambda_1 e^{(80-20)\times 4.3/85} + \lambda_2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

The resulting parameter values are $\lambda_0 \approx -0.08$, $\lambda_1 \approx 0.02$ and $\lambda_2 \approx 3.8$. As expected, $\lambda_t(t, d(t)) > 0$ and $\lambda_d(t, d(t)) > 0$ (see subsection 3.1 for the underlying logic). Furthermore, the calibrated functional form implies that individuals aged 60 years reporting poor health have a hazard rate approximately 4 times higher than those in excellent health, slightly falling short of the 5.5 times higher documented by Cho et al. (2022).

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

4.3.1. *Monetary cost of health deficit, B.* The left panel of Figure 3 uses data from the 2022 MEPS to construct the median total medical expenditures as a percentage of income by

FIGURE 3. Medical expenditures and distribution of age at death



Notes. The left panel displays median total medical expenditures as a percentage of income by age, with data sourced from the 2022 MEPS. The right panel shows the age-at-death distribution, with data from the 2022 HMD.

age. This ratio remains almost constant before rising around 60 years. For example, fitting a quadratic time trend to extend the series until the age of 105 suggests that at that age, medical expenditures represents 50% of income. This exponential increase is consistent with De Nardi et al. (2016), who find, for example, that medical expenses more than double between ages 70 and 90, and that average medical expenditures for an American aged 65 or older are 2.6 times the national average.

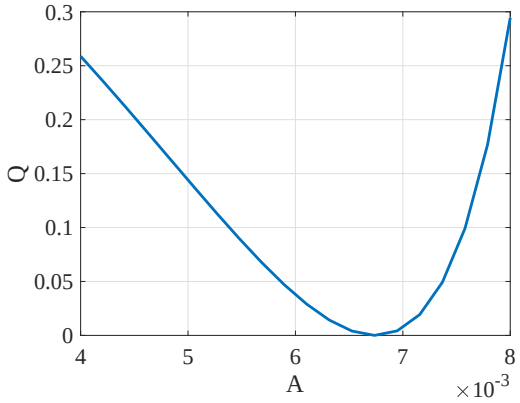
In our setup, medical expenditures are divided between preventive care, h, and the monetary cost of health deficit, Bd. Recall that the latter can be viewed as long-term care expenditures, as well as curative or palliative care. Importantly, at the maximum admissible age, $\bar{T}$, there is no incentive to spend on preventive care, as death is imminent, and all medical expenditures are the monetary cost of health deficit.[6] 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 $\frac{\theta B\bar{d}}{y(\bar{T},\bar{d})} = 0.5$ (see above). Since we consider 105 years as the maximum admissible age, we set

$$B = 0.5\frac{y(\bar{T},\bar{d})}{\theta\bar{d}} \approx 1.9.$$

4.3.2. *Efficiency of preventive care, A.* We calibrate the remaining parameter, A, using a simple grid search, evaluating parameter values over a predefined range to ensure that the

[6]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.

FIGURE 4. Grid search results



	Data	Model
Maximum lifespan ($\bar{T}$)	4.36	4.35

Notes. The left panel displays the objective function $Q(A)$ against its arguments, A. The right panel compares the empirical targets with the model-implied outcomes.

key condition upon which most of our calibration rests holds (see subsection 4.2.1)

$$e^{-\bar{T}} \approx 0.95^{(105-20)},$$

or equivalently $\bar{T} = 4.36$. Our grid search method then minimises the objective function

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

The left panel in Figure 4 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. Furthermore, the value of the loss function at the minimum is nearly zero.

4.4. **Assessment.** To evaluate the model's performance on non-targeted criteria, we first assess key statistics related to the age at death stationary distribution.[7] These statistics include the first three moments of the age-at-death distribution and the probability of reaching 75 conditional on being alive at 50. We compute the skewness of the age-at-death distribution and the conditional probability using a 10,000-agent Monte Carlo simulation, while the explicit formulas for the other statistics are provided in subsection 3.3. As shown in Table 4, the model fits the age-at-death distribution well, with all statistics falling within a plausible range.

Now, we assess the life-cycle dynamics of an individual reaching the maximum lifespan of 105 years. Figure 5 compares key model-implied paths with those observed in the data, as described in previous subsections. Starting with the top-left panel, our model generates a health deficit path that increases monotonically with age in a convex manner, consistent with the data. In addition, the model implies a probability of survival at a given age,

[7]At each instant, a new cohort of size 1 is born, leading to a stationary population distribution (see subsection 3.3).

TABLE 4. Non-targeted moments: data and model

	Data	Model
Mean age at death	78	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: In the model, we compute the skewness of age at death and the probability of reaching 75, conditional on being alive at 50, using a 10,000-agent Monte Carlo simulation. In the data, both measures come from the 2022 actuarial tables in the HMD.

$\Lambda(t)$, that closely matches the data, although a larger share of agents ($\approx 6\%$) reach the maximum admissible age compared to the data (0.2%). This occurs because the model underestimates mortality at very old ages, which also explains why the skewness of the age-at-death distribution is not sufficiently negative (recall Table 4).

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 reasonably close to the observed pattern.

The bottom-center panel plots the ratio of total medical expenditures to income by age. While this ratio remains fairly constant in the data before rising around 60 years, the model shows a decrease before surging around age 85. Three factors contribute to this behavior – two affect the numerator, and one affects the denominator. First, preventive care decreases with age (see the bottom-right panel). The reasoning is simple: the health deficit is a stock variable that impacts the remainder of an individual's life. Thus, a unit of preventive care at time t provides benefits over the period $[t, T]$. As t nears T, the time available to reap these benefits shortens, reducing the incentive for preventive care. Second, as the health deficit increases with age, so does its associated monetary cost (see the bottom-right panel). Third, income follows the hump-shaped pattern mentioned earlier. Together, these factors drive the dynamics of the share of income allocated to healthcare.

We can also use the figure to compare the share of income spent on medical care across the entire population (denoted by H in subsection 3.3) in both the data and the model. Using the trapezoid rule to compute the necessary integrals, we find that the ratio is 6.5% in the

FIGURE 5. Non-targeted life-cycle paths: data and model

Notes. Refer to the corresponding subsection above for details on data sources. Since the time series for the health deficit, income, and medical expenditures relative to income extend only to 80 years in the data, we extrapolate them to 105 years using simple polynomial fits.

data and 9% in the model.[8] All told, the model does a fair job at approximating individuals' medical expenditures. Lastly, the top-right panel confirms that utility flows remain positive, ruling out the preference-for-death scenario.

In sum, although stylized, the baseline model replicates key empirical patterns of mortality and health well. This gives us confidence that it provides a plausible framework for introducing income and health heterogeneity to study the health-to-income and income-to-health channels. We turn to this next.

5. Income and health heterogeneity in the baseline model

Let us now assess the model's ability to capture the correlation between income and life expectancy discussed in Section 2, focusing on two key channels: (i) the health-to-income channel, where health affects income, and (ii) the income-to-health channel, where income affects health. For simplicity, let us from now on denote by $\hat{d}_0$ and $\hat{\mu}_0$ the initial health deficit

[8]The share of individuals' income spent on medical care, 6.5%, obtained from the 2022 MEPS and the 2022 HMD, is significantly lower than the ratio of total health expenditures to GDP, 17.6%, reported in the 2023 National Health Expenditure Fact Sheet. While explaining this difference is beyond the scope of this paper, a likely reason is that survey data focus on individuals' direct expenditures on healthcare services, whereas the share of GDP spent on healthcare includes broader expenditures, such as government spending on public health, hospital funding, and other indirect costs.

and the permanent income component in the baseline model without ex-ante heterogeneity just described.

5.1. **Income and life expectancy.** In the baseline model, individuals differ only in their time of death, not in health status or income. Hence, everyone aged t has the same life expectancy, preventing the model from capturing the known correlation between income and life expectancy. To address this, we introduce permanent, ex-ante heterogeneity that generates cross-sectional variation in income, health, and life expectancy. We consider two types of ex-ante heterogeneity: one related to health and another related to income. First, individuals might differ in their initial health deficit, d_0, reflecting variations in genetics and early-life experiences. This aligns with recent research highlighting the lasting impact of early-life factors on health and economic outcomes (see De Nardi et al., 2024, and references therein). Formally, individual i draws d_0 from a uniform distribution $d_0 \sim \mathcal{U}(\hat{d}_0 - \epsilon_d, \hat{d}_0 + \epsilon_d)$, where parameter $\hat{d}_0$ is the initial health deficit in the baseline model, and $\epsilon_d \geq 0$ determines the degree of cross-sectional variation. Second, individuals might differ in their permanent income component, μ_0, capturing variations in work ethic, productivity, or access to better education and professional networks. Formally, individual i draws μ_0 from a uniform distribution $\mu_0 \sim \mathcal{U}(\hat{\mu}_0 - \epsilon_y, \hat{\mu}_0 + \epsilon_y)$, where $\hat{\mu}_0$ is the permanent income component in the baseline model, and $\epsilon_y \geq 0$ (see Appendix A for the firm maximization program with heterogeneous agents).[9]

5.1.1. *Income heterogeneity.* To isolate the impact of each channel, we introduce one source of heterogeneity at a time. We begin by allowing individuals to differ in their permanent income components while keeping their initial health deficits identical. The solid blue line in the right panel of Figure 6 shows the resulting relationship between income and life expectancy at age 50 in the stationary population distribution. Specifically, we run a Monte Carlo simulation with 50,000 agents, where each individual draws $\mu_0 \sim \mathcal{U}(2.8, 3.2)$ and $d_0 = \hat{d}_0$. We then identify all individuals alive at age 50, divide them into five income groups, compute their life expectancy as the mean age at death within each group, and plot the results. Since extending the bounds of the uniform distribution only increases the length of the solid blue line, we choose to remain within the neighborhood of the baseline calibration.

Income contributes to life expectancy inequality. Higher income enables greater investment in preventive care, which reduces health deficits and, in turn, lowers mortality risk. An indirect effect reinforces this relationship: lower health deficits reduce medical expenditures

[9]We choose the uniform distribution because solving our model involves a nonlinear boundary value problem with an endogenous maximum admissible time, which is numerically challenging and does not always yield a solution. This constraint forces us to stay close to the parameter space explored in the baseline calibration.

Bd and increase income, further enabling investment in preventive care. This creates a self-reinforcing cycle. Previous structural studies that assume exogenous health overlook these mechanisms (see Section 2). From a quantitative perspective, the blue line suggests that a 10% increase in income at age 50 corresponds to a rise in life expectancy of approximately nine months. For comparison, Chetty et al. (2016) put the figure at around 6 months.[10]

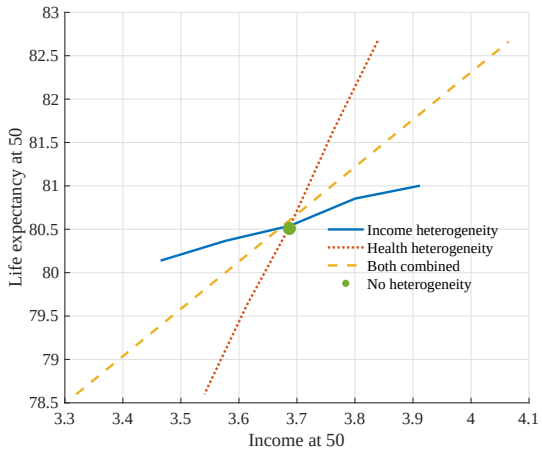
At face value, this suggests that income heterogeneity alone could account for the observed correlation between income and life expectancy. However, there is an important caveat. In our model, income differences are permanent *and* known to individuals. Furthermore, individuals perfectly understand the impact of their choices on their health and mortality risk. By contrast, in Chetty et al. (2016), individuals at a given income percentile at age t may have experienced unexpected income fluctuations earlier in life and could face them in the future. Moreover, individuals often face cognitive limitations when making health-related decisions. For instance, Keane and Thorp (2016) show that consumers, particularly the elderly, struggle to make optimal choices regarding health insurance, healthcare, and retirement planning. These difficulties often result in behaviors indicative of confusion, such as selecting dominated insurance plans or failing to respond adequately to financial incentives. Together, unexpected income fluctuations and cognitive limitations could help explain why the link between income and mortality risk may appear stronger in our model than in the data.

Remark. The income-to-health deficit described above rests on the idea that higher disposable income enables greater investment in preventive care. This aligns with recent empirical evidence. For example, Kurani et al. (2020) found that individuals living in the least deprived census block groups in Minnesota, Iowa, and Wisconsin were roughly twice as likely to complete recommended screenings for breast cancer, cervical cancer, and colorectal cancer compared to those in the most deprived census block groups. Bauer et al. (2022) document comparable outcomes across the US, highlighting that counties with higher social vulnerability had significantly lower odds of receiving the recommended cancer screenings. In addition, French et al. (2019) report that lower-income households purchase less healthful foods than

[10]More precisely, Chetty et al. (2016) estimate that in 2014, the expected age at death for 40-year-old American men in the second quartile of the income distribution (mean \$47k) was approximately 81 years, while for those in the third quartile (mean \$83k), it was around 85 years. Assuming a linear relationship between expected age at death and income in that segment of the income distribution – an assumption supported by further results in the paper – an increase in income of 10% correlates with an increase in life expectancy of about six months. Though our figure reports the correlation at age 50, the link between these variables remains similar at age 40.

higher-income households, even when adjusting for factors such as education, marital status, and race.

FIGURE 6. Income and life expectancy



	Age at death		
	Mean	Dispersion	Skewness
Data	78	17	-1.5
No heterogeneity	76	19	-0.5
Income heterogeneity	76	19	-0.5
Health heterogeneity	76	20	-0.5
Both combined	76	20	-0.5

Notes. The left panel plots the relationship between income and life expectancy at age 50 under different sources of ex-ante heterogeneity. The results are based on a Monte Carlo simulation with 50,000 agents. The right panel reports the first three moments of the resulting age-at-death distribution.

5.1.2. *Health heterogeneity.* We now shut down ex-ante income heterogeneity, but allow individuals to differ in their initial health deficits. Specifically, we proceed exactly as before, but individuals now draw $d_0 \sim \mathcal{U}(0.018, 0.022)$ and $\mu_0 = \hat{\mu}_0$. The dashed red line plots the resulting link between income and life expectancy at 50. As in models by Capatina (2015), De Nardi et al. (2024), and Hosseini et al. (2025), the dashed red line illustrates how health contributes to income inequality. Three mechanisms drive this effect. First, our calibration finds $y_d(t, d(t)) < 0$, meaning individuals with higher initial health deficits tend to earn less later in life. Second, higher health deficits lead to greater medical expenditures (captured by $Bd(t)$), reducing resources available for preventive care. This accelerates health deficit accumulation, further lowering income. Third, worse health lowers the effective discount factor (see subsection 3.2), making individuals less patient – a pattern recently documented by De Nardi et al. (2024). Less patient individuals invest less in preventive care, which worsens health deficits and further suppresses income.

The dashed red line also illustrates how health contributes to life expectancy inequality: individuals with high d_0's have a life expectancy roughly 4 years lower than those with low d_0's (78.5 versus 82.5 years). Higher health deficits, both due to a higher initial value and the accelerated accumulation described earlier, lead to higher hazard rates. This increased

likelihood of death at each period reduces life expectancy. The strong link between health at 50 and life expectancy appears in the data as well. Using a sample of over 1,200 individuals aged 50 to 54 from the 2022 HRS, we find a strong correlation between health and the expected probability of living to age 75 or beyond. Individuals in excellent health report a median expected probability of 80%, while those in poor health report a median expected probability of 40%.

5.1.3. *Both income and health heterogeneity.* Overall, either source of ex-ante heterogeneity enables the model to generate a meaningful link between income and life expectancy, suggesting the importance of both the health-to-income and the income-to-health channels. This aligns with Deaton (2016), who argues that while there is almost certainly some causal relationship between income and mortality, poor health also threatens income, creating a reverse causality from health to income throughout the life course.

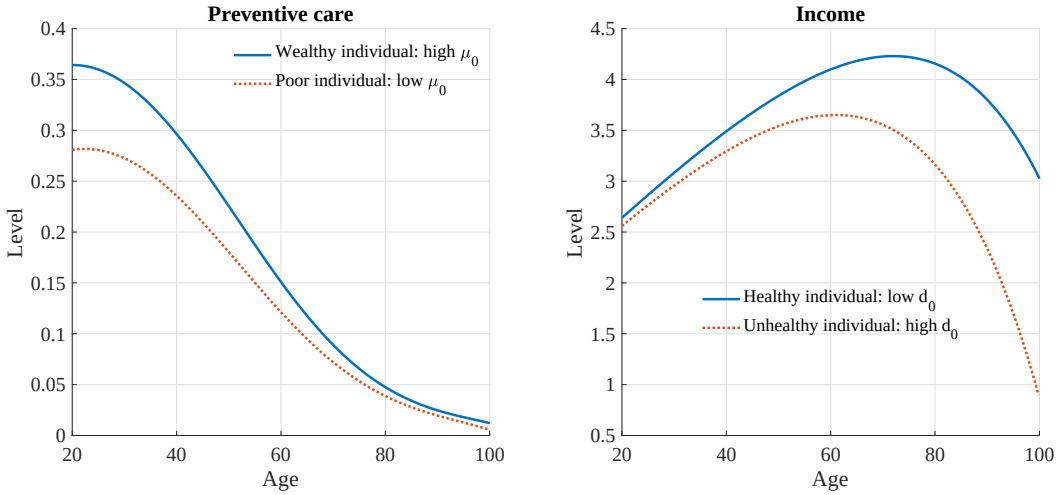
Not surprisingly, combining both sources of ex-ante heterogeneity (dashed yellow line in the figure) results in a link between income and life expectancy that falls in the middle of the range established by considering each source individually. In other words, the dashed yellow line is broadly the sum of the two vectors that determine the solid blue and dotted red lines. Turning briefly to the right panel in Figure 6, the table shows that even with meaningful ex-ante heterogeneity, the model still matches the age at death distribution fairly well. This is due to the uniform initial distributions for d_0 and μ_0 in the neighborhood of the baseline calibration.

5.2. **Income-to-health and health-to-income channels over the life-cycle.** As discussed in Section 2, some studies argue that the casual links between health and income may change with age (Smith, 1998; Cutler et al., 2011). More precisely, income 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 income.

Our model supports this hypothesis. Consider the first part of the conjecture: the income-to-health channel is strongest early in life. In our framework, higher income improves health by increasing investment in preventive care. Put differently, if preventive care use were unrelated to income, income would have no effect on health. The left panel of Figure 7 shows how preventive care changes with age for a wealthy individual ($\mu_0 = 3.2$) and a poor individual ($\mu_0 = 2.8$). The gap is widest early in life, as greater early-life investment sets wealthy individuals on a more favorable health trajectory. Over time, this gap narrows, collapsing near the end of life when additional preventive care provides little benefit (see Subsection 4.4).

Now consider the second part of the conjecture: the health-to-income channel is strongest in adulthood. Subsection 5.1 outlined the three mechanisms through which initial health

FIGURE 7. Intensity of income-to-health and health-to-income channels



Notes. The left panel shows how preventive care use changes with age for a wealthy individual ($\mu_0 = 3.2$) and a poor individual ($\mu_0 = 2.8$). The right panel of shows how income evolves with age for a healthy individual ($d_0 = 0.018$) and an unhealthy individual ($d_0 = 0.022$) .

deficits lead to meaningful income differences later in life. The right panel of Figure 7 illustrates this by showing how income evolves with age for a healthy individual ($d_0 = 0.018$) and an unhealthy individual ($d_0 = 0.022$). Early in life, the income gap is small because initial health deficits are similar. Over time, it widens as healthier individuals accumulate deficits more slowly, reducing their discount rate and medical expenses Bd, while enabling greater preventive care use. This feedback loop amplifies income differences later in life, favoring those with better initial health.

In sum, the model confirms that the strength of both channels varies with age. This finding highlights the need to account for both factors when assessing the idea that subsidy and redistribution policies could be effective tools of public health. We turn to this next.

6. Preventive subsidies and redistribution policies

Developed countries subsidize preventive care and implement redistributive policies, though the extent and approach vary. In the U.S., preventive care is subsidized through public programs and insurance regulations, including Medicare, Medicaid, the Affordable Care Act, and tax benefits for employer-based insurance. In turn, the U.S. tax system is progressive, and programs like the Earned Income Tax Credit and food assistance provide additional redistribution. In this section, we explore how health subsidies and redistribution policies shape the relationship between income and mortality risk. In what follows, we keep both sources of heterogeneity, d_0 and μ_0, active.

6.1. **Government policies and budget constraint.** The government reimburses a share s_h of investment in preventive care and provides lump-sum transfers ω. To balance its budget, it finances these expenditures through a proportional earnings tax τ. These three instruments apply uniformly to all individuals, regardless of age, income, or health. As a result, individuals' budget constraints (3) now become

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

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 and subsidies. To compute the government budget constraint, we aggregate over all individuals. Each individual is characterized by age t, initial health deficit d_0, and initial permanent income component μ_0. Any individual variable $x \in \{c, h, d, y, \lambda\,\Lambda\}$ can then be expressed as $x(t, d_0, \mu_0)$ and aggregated as

$$X = \underbrace{\int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \underbrace{\int_{\hat{d}_0-\epsilon_d}^{\hat{d}_0+\epsilon_d} \underbrace{\int_0^{\bar{T}(d_0,\mu_0)} x(t, d_0, \mu_0)\Lambda(t, d_0, \mu_0)\,\mathrm{d}t}_{\text{Age dimension}}\, \mathcal{U}_{\left[\hat{d}_0\pm\epsilon_d\right]}\,\mathrm{d}d_0}_{\text{Health dimension}}\, \mathcal{U}_{[\hat{\mu}\pm\epsilon_y]}\,\mathrm{d}\mu_0}_{\text{Income dimension}}\,. \tag{10}$$

Here, $\mathcal{U}_{\left[\hat{d}_0\pm\epsilon_d\right]}$ and $\mathcal{U}_{[\hat{\mu}\pm\epsilon_y]}$ are the uniform distributions governing d_0 and μ_0, while $\bar{T}(d_0, \mu_0)$ is the maximum admissible age of an individual characterized by d_0 and μ_0. Aggregating the linear equation equation (9) yields the government budget constraint

$$s_h\theta H + \omega P = \tau Y, \tag{11}$$

where P is the size of the population, obtained from equation (10) with 1, the size of each newborn generation, instead of $x(t, d_0, \mu_0)$. Therefore, the left-hand side represents government expenditures and the right-hand side represents total revenues. Given any pair $\{s_h, \omega\}$, the government chooses τ to ensure that equation (11) is satisfied.

6.2. **Income and life expectancy with government intervention.** Our model predicts a positive link between income and life expectancy. Now, we examine how government intervention through subsidies, s_h, or transfers, ω, shapes this relationship. Our goal is not to

rank these policies but to understand their qualitative effects on the income-health gradient. For simplicity, we will calibrate each instrument to a round figure, though the underlying logic applies more broadly to any calibration. As a result, the degree of government intervention will vary across instruments, which is not a concern for our positive analysis.

To isolate the impact of each policy, we introduce them one at a time. We start with a 10% preventive care subsidy ($s_h = 0.1$) and no lump-sum transfers ($\omega = 0$). To balance the government budget constraint, equation (11) implies a 0.7% income tax. The dotted red line in the left panel of Figure 8 shows the resulting relationship between before tax earnings, $y(t, d(t))$, and life expectancy at age 50 in the stationary population distribution. As a benchmark, the solid blue line shows the no-policy scenario discussed in Subsection 5.1. The effect of government intervention on total net income, $(1 - \tau)y(t, d(t)) + \omega$, is discussed at the end of this subsection.

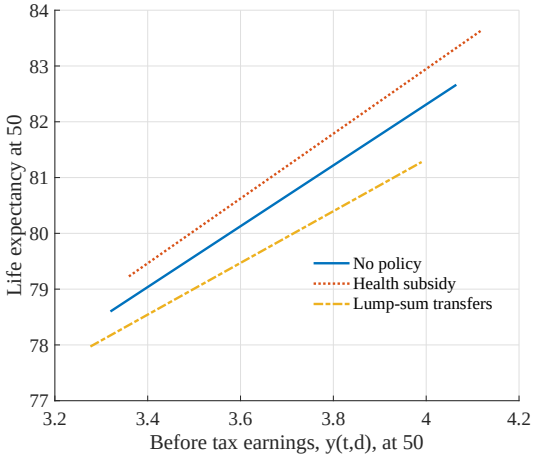
Comparing the solid blue and dotted red lines highlights three effects. First, health subsidies raise life expectancy at age 50 across all income levels, shifting the curve upward. By lowering the cost of preventive care, subsidies make it more attractive, leading individuals to allocate a larger share of income to it. This, in turn, reduces mortality risk through the mechanisms outlined in Subsection 5.1. In the right panel of Figure 8, this upward shift corresponds to an increase of 12 months in the mean age at death. Second, health subsidies raise earnings at age 50 for all individuals, shifting the curve rightward. As discussed earlier, greater preventive care slows the accumulation of health deficits, allowing individuals to enjoy higher earnings. Third, health subsidies widen the life expectancy gap between rich and poor by steepening the curve. While all individuals increase their preventive care investment by a similar *proportion* – about 20% in this case (see right panel) – the *absolute* increase is larger for wealthier individuals. As a result, their health deteriorates more slowly, extending their life expectancy more than that of lower-income individuals. This explains why the dispersion of age at death increases by about 4 months.[11]

We now consider lump-sum transfers $\omega = 1.7$ and no preventive care subsidy ($s_h = 0.0$). To balance the government budget constraint, equation (11) implies a 50% income tax.[12]

[11] A 10% price reduction leading to a 20% increase in consumption suggests that preventive care is a superior good. Indeed, in our model wealthier individuals allocate a larger share of their income to it than those with lower incomes. Hall and Jones (2007) finds a similar pattern, as health investment in their framework, like in ours, extends lifespan and improves quality of life.

[12] $\omega = 1.7$ implies that the absolute change in the mean age at death matches that of the subsidy scenario with $s_h = 0.1$, as shown in the right panel of Figure 8.

FIGURE 8. Income and life expectancy with government intervention



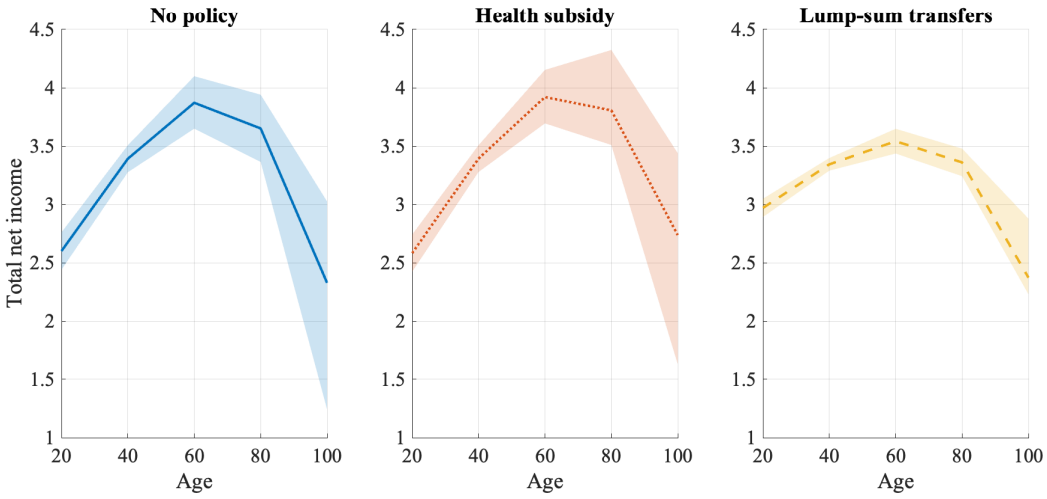
	Age at death	
	Mean	Dispersion
No policy	76.0	19.5
Health subsidy	77.0	19.8
Lump-sum transfer	75.0	19.2
	(Normalised) Initial preventive care	
	Poor and unhealthy	Rich and healthy
No policy	1.00	1.00
Health subsidy	1.21	1.22
Lump-sum transfer	0.95	0.84

Notes. The left panel shows the relationship between before tax earnings, $y(t, d(t))$, and life expectancy at age 50 under government actions, based on a Monte Carlo simulation with 50,000 agents. The right panel reports the first two moments of the resulting age-at-death distribution, and initial preventive care investment. In the latter case, values are normalized to 1 under the no-policy scenario, with reported numbers under the health subsidy and lump-sum transfer indicating the relative change from this benchmark. Poor and unhealthy refer to an individual with $\mu_0 = 2.8$ and $d_0 = 0.022$; rich and healthy refer to an individual with $\mu_0 = 3.2$ and $d_0 = 0.018$.

The dashed yellow line in the left panel of Figure 8 shows the resulting relationship between before tax earnings and life expectancy at age 50 in the stationary population distribution.

Comparing the solid blue and dashed yellow lines highlights three effects. First, lump-sum transfers reduce life expectancy at age 50 across all income levels, shifting the curve downward. By weakening the link between health deficits and earnings (an income tax of 100% would eliminate them completely), these transfers lower the incentive to invest in preventive care, leading to higher mortality risk. In the right panel of Figure 8, this downward shift corresponds to a decrease of 12 months in the mean age at death. Second, lump-sum transfers reduce earnings, $y(t, d(t))$, at age 50 for all individuals, shifting the curve leftward. As noted earlier, reduced preventive care accelerates health deficit accumulation, which in turn lowers earnings across the entire population. Third, lump-sum transfers narrow the life expectancy gap between the rich and the poor, flattening the curve. Wealthier individuals experience a larger decline in preventive care use, both in relative and absolute terms, compared to poorer individuals. This is due to their reduced incentives to invest in preventive care and the decrease in disposable income due to income redistribution. In contrast, the impact on preventive care investment for poorer individuals is influenced by two opposing factors: the reduced incentives to invest in preventive care and the increased disposable income resulting from lump-sum transfers. As a result, their preventive care

FIGURE 9. Total net income dispersion as a function of age



Notes. Median total net income, $(1-\tau)y(t,d(t))+\omega$, with shaded areas representing the 25th and 75th percentiles. Results from a 180,000-agent Monte Carlo simulation.

investment declines less than that of wealthier individuals (see right panel), leading to a smaller decrease in life expectancy. This explains why the dispersion of age at death falls by about 4 months.

In sum, the two policies have opposite effects on the income-health link, and hence, on the age at death distribution. The key difference lies in how they shape incentives for preventive care. Health subsidies encourage greater preventive care by reducing its relative cost, while income redistribution discourages it by weakening the link between earnings and health deficits.

These findings do not imply that income redistribution is inherently flawed. Figure 9 shows median total net income, $(1-\tau)y(t,d(t))+\omega$, with shaded areas representing the 25th and 75th percentiles. Compared to the no-policy and health subsidy scenarios, lump-sum transfers significantly reduce income inequality by narrowing the gap between these percentiles. This effect is especially pronounced among the elderly, where the lowest-income individuals receive substantially higher incomes than in the other two scenarios. Moreover, lump-sum transfers weaken the link between total net income and age, reducing inequality across cohorts. As a result, redistribution increases consumption for those with the highest marginal utility of consumption. A utilitarian central planner might accept shorter life expectancy and lower earnings in exchange for higher per-period utility for lower-income individuals. Evaluating this trade-off quantitatively is beyond the scope of this paper.

7. Extensions to the baseline model

We now extend the baseline model. In subsection 7.1, we introduce a risk-free asset, allowing agents to transfer resources safely over time. In subsection 7.2, we incorporate retirement, with individuals receiving a pension financed by a pay-as-you-go system. For simplicity, this section assumes no government intervention in the form of health subsidies or lump-sum transfers, setting $s_h = \omega = 0$. Appendix B relaxes this assumption and shows that our main conclusions about government intervention remain as in the baseline model.

7.1. **Risk-free asset.** Our baseline model abstracts from savings tools for mathematical tractability. This might not be as strong an assumption as it first appears: many households hold little wealth. For instance, Aguiar et al. (2024) find that 40% of US households live hand-to-mouth, and this behaviour persist over time: hand-to-mouth households often remain so for years. Nonetheless, we now relax the no-savings assumption, introducing a risk-free asset that allow agents transfer resources safely over time. 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

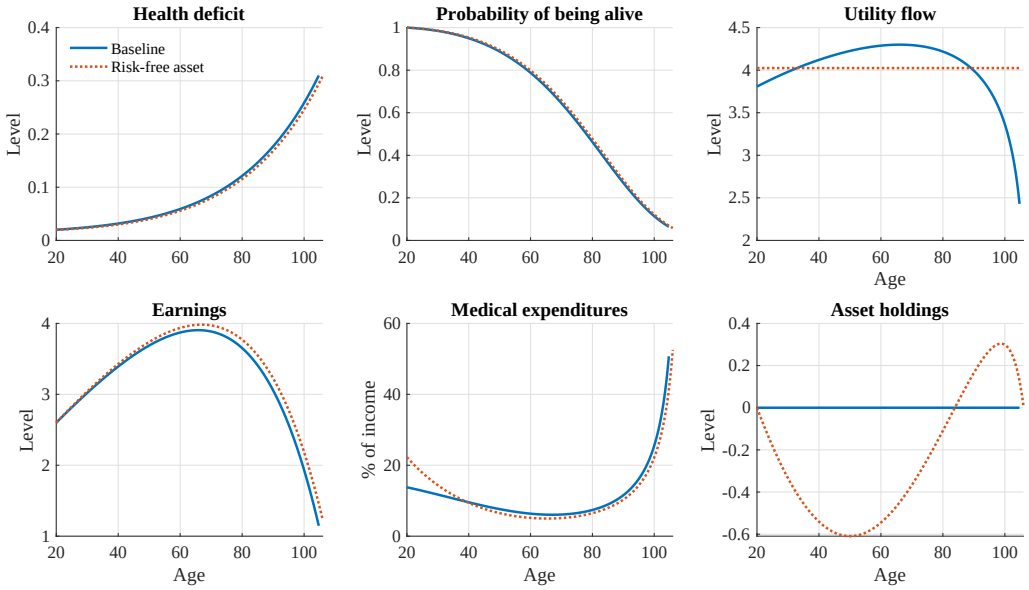
conditions for the extended model, as they result from the same methodology used in the baseline case.

To explore how access to a risk-free asset shapes the link between income and mortality, we retain the calibration of the baseline model (see Table 2), and set the risk-free rate equal to the discount rate, $r = \rho$, as common in most macroeconomic models. As a result, individuals will choose a perfectly flat consumption path. To illustrate, Figure 10 tracks the life-cycle dynamics of the median individual reaching the maximum lifespan $\bar{T}$. For comparison, blue lines represent the baseline model without saving tools studied so far.

As expected, the risk-free asset generates constant utility flows, for individuals find it optimal to smooth consumption. More importantly, individuals borrow during their youth against future earnings to increase initial consumption and fund higher preventive care, reflected in increased medical expenditures early in life. Higher preventive care improves the individual's health trajectory, leading to higher future earnings, lower medical costs, and reduced mortality risk. Indeed, while the solid blue and dotted red lines appear to overlap in the top middle panel displaying the probability of being alive at every age, the dotted red line is slightly above, resulting in an increase in life expectancy at birth of almost one year. In the bottom right panel, asset holdings decrease during youth and late life. This is due to time-varying incentives to invest in preventive care and because these stages correspond to the lowest income periods, as earnings follow a hump-shaped pattern.

These life-cycle paths suggest that access to a risk-free asset has similar effects on the income-mortality link as the preventive care subsidy studied in Subsection 6.2. Both mechanisms transfer resources from the old to the young – savings through borrowing against future earnings and the subsidy through taxation – to support higher preventive care early in life. To see this point more clearly, the left panel of Figure 11 shows the relationship between earnings and life expectancy at age 50 in the stationary population when both sources of ex-ante heterogeneity, d_0 and μ_0, are active. The solid blue line represents the baseline model without policy intervention, while the dotted red line represents the no-policy scenario with access to a risk-free asset. Access to a risk-free asset increases life expectancy at all earning levels (upward shift) and raises them (rightward shift), while also widening the life expectancy gap between rich and poor (steeper slope). These effects mirror those of the preventive care subsidy, s_h, funded through a constant earnings tax, τ, in Figure 8. The steeper slope arises because rich, healthy individuals can leverage their higher expected future earnings and borrow more during youth than their poor, unhealthy counterparts. As a result, they invest much more in preventive care early in life. For instance, an individual with the best (worst) possible health and highest (lowest) earnings increases preventive care at age 20 by 80% (60%) compared to the baseline model without savings.

FIGURE 10. Life-cycle paths of the median individual: baseline model and risk-free asset extension

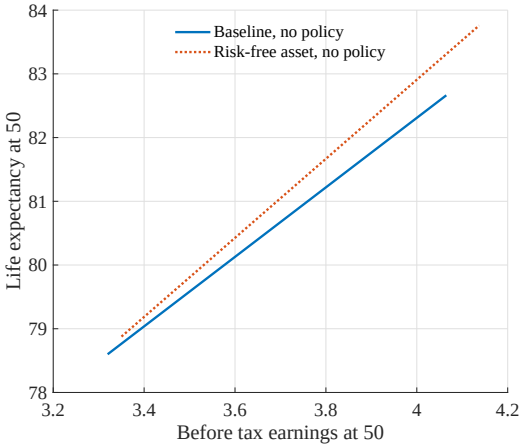


Notes. Solid blue lines represent the baseline model without saving tools. Dotted red lines represent the risk-free asset extension. Both models retain the baseline calibration presented in Table 2, with the risk-free extension further setting $r = \rho$.

The right panel of the figure further illustrates how the risk-free asset increases life expectancy. It also shows that mean disposable income in this extension is lower than in the baseline model. With access to a risk-free asset, individuals hold negative asset positions for most of their lives, making the economy a net borrower (see Figure 10). As a result, interest payments to foreign lenders exceed income received from abroad, reducing overall disposable income. Lastly, Appendix B shows that access to a risk-free asset does not alter our main conclusions about government intervention.

7.2. **Pay-As-You-Go (PAYG) pension scheme.** In the baseline model, individuals supply one unit of labor inelastically at each instant $t \in [0, T]$ and receive a real wage equal to their labor productivity, $y(t, d(t))$. In reality, however, most individuals retire before the end of their lives. To capture this, we now 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,

FIGURE 11. Income and life expectancy: baseline model and risk-free asset extension



	Age at death		Total net income	
	Mean	Dispersion	Mean	Dispersion
Baseline	76.0	19.5	3.43	0.52
Risk-free asset	76.9	19.6	3.04	0.37

Notes. The left panel plots the relationship between income and life expectancy at age 50 in the baseline model and the risk-free extension under no government intervention. The results are based on a Monte Carlo simulation with 100,000 agents. The right panel reports the first two moments of the resulting total net income and age-at-death distributions.

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 $\hat{t} \to \infty$ restores the baseline model.[13] Assuming no health subsidies and no lump-sum transfers, the government budget constraint is

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

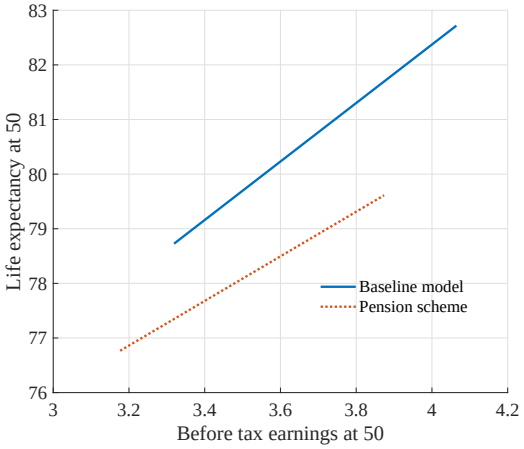
where $P_{>\hat{t}}$ denotes the population above age $\hat{t}$, and $Y_{<\hat{t}}$ represents the total labor earnings of individuals below that age (see Appendix A for analytical expressions). This formulation corresponds to a PAYG pension system, in which current workers' contributions finance the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

To examine how a PAYG pension scheme affects the relationship between income and mortality, we keep the baseline model calibration (see Table 2) and abstract from the risk-free asset described in the previous subsection. Since the effective retirement age in the US is

[13]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.

around 65, we set $\hat{t} = (65 - 20) \times 4.3/85$. In addition, we set $k = 1.7$, which requires a labor earnings tax rate of 17% to balance the government's budget. The implied pension replacement rate, defined as $\frac{k}{(1-\tau)y(\hat{t},d(\hat{t}))}$, is 55% for the median agent. This aligns with the empirical evidence documenting a net pension replacement rate in the US in 2022 slightly above 50%.[14]

FIGURE 12. Income and life expectancy: baseline model and pension scheme



	Age at death		Total net income	
	Mean	Dispersion	Mean	Dispersion
Baseline	76.0	19.5	3.43	0.52
Pension scheme	73.8	18.7	2.48	0.52

Notes. The left panel plots the relationship between income and life expectancy at age 50 in the baseline model and the pension scheme extension under no government intervention. The results are based on a Monte Carlo simulation with 100,000 agents. The right panel reports the first two moments of the resulting total net income and age-at-death distributions.

The left panel of Figure 12 shows the relationship between earnings and life expectancy at age 50 in the stationary population when both sources of ex-ante heterogeneity, d_0 and μ_0, are active. The solid blue line represents the baseline model, while the dotted red line represents the extension with the pension scheme. Clearly, the PAYG scheme has very similar effects on the income-mortality link as the lump-sum transfers discussed in Subsection 6.2. Both mechanisms weaken the link between health and earnings later in life – the pension scheme by enabling retirement and the lump-sum transfers through taxation. This weaker link discourages preventive care during youth, raising mortality risk and lowering labor earnings at age 50. Lastly, Appendix B confirms that, in the PAYG extension, lump-sum transfers and health subsidies shape the income and age distributions in the same way as in the baseline model.

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

8. Concluding remarks

Our model shows that investing in preventive care during youth brings long-term benefits for income as well as life expectancy. For example, higher initial income, health subsidies, and access to a risk-free asset in youth all raise life expectancy. Our model also shows that poor health increases economic inequality by decreasing disposable income, especially in later years. Therefore, authorities wishing to limit the extent of inequality may choose to provide income protection in later life, through policies such as income redistribution or the PAYG pension system discussed earlier.

Our paper highlights two areas for future research. First, our model abstracts from morbidity risk – the possibility of developing a severe condition that does not affect life expectancy but raises medical costs and reduces labor productivity. We could address this by introducing a specific hazard rate for such health conditions, allowing it to depend on both current age and the current health deficit. Adding morbidity risk would create a role for health insurance markets, offering a richer setting to explore the income-health gradient and the possible role of government intervention. Second, our model assumes that agents inelastically supply one unit of labor. Another possible extension would treat labor supply as an endogenous choice, opening new channels through which government policy affects macroeconomic outcomes. For instance, policies that raise life expectancy, such as a health subsidy, could increase labor supply among older individuals by extending their planning horizon. This would also generate general equilibrium effects: shifts in labor supply would affect government revenues and, in turn, the tax rate required to balance the budget.

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Appendix A. Representative firm

A.1. **Baseline model.** We have a distribution of individuals differing in

- age t,
- initial health deficit $d_0 \sim \mathcal{U}(\hat{d}_0 - \epsilon_d, \hat{d}_0 + \epsilon_d)$,
- permanent component of productivity $\mu_0 \sim \mathcal{U}(\hat{\mu}_0 - \epsilon_y, \hat{\mu}_0 + \epsilon_y)$.

Note that when $\epsilon_d = \epsilon_y = 0$, we obtain the representative median agent model presented in Section 3. When $\epsilon_d > 0$ and $\epsilon_y > 0$, we have the heterogeneous agent model presented in Section 5. Each individual is therefore characterized by (t, d_0, μ_0). We assume it supplies one unit of labor, and has a specific labor productivity given by $y(t, d(t, d_0, \mu_0))$.

For each type of individual (t, d_0, μ_0), a representative firm demands $l^d(t, d_0, \mu_0)$ units of labor and pays a unit wage of $w(t, d_0, \mu_0)$. We assume that the firm runs a constant return to scale technology with labor as only input. Moreover, the firm can perfectly substitute between each type of labor. Therefore, the firm solves the following maximization program

$$\max_{l^d(t,d_0,\mu_0)} \Big\{ \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_{\hat{d}_0-\epsilon_d}^{\hat{d}_0+\epsilon_d} \int_0^{\bar{T}(d_0,\mu_0)} y(t, d(t, d_0, \mu_0))\, l^d(t, d_0, \mu_0)\, \mathrm{d}t\, \tfrac{1}{2\,\epsilon_d}\, \mathrm{d}d_0\, \tfrac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0 - \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_{\hat{d}_0-\epsilon_d}^{\hat{d}_0+\epsilon_d} \int_0^{\bar{T}(d_0,\mu_0)} w(t, d_0, \mu_0)\, l^d(t, d_0, \mu_0)\, \mathrm{d}t\, \tfrac{1}{2\,\epsilon_d}\, \mathrm{d}d_0\, \tfrac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0 \Big\},$$

where $\bar{T}(d_0, \mu_0)$ is the maximum admissible age for an individual equipped with d_0 and μ_0. It is implicitly given by $d(\bar{T}, d_0, \mu_0) = \bar{d}$. The solution to the maximization problem is $w(t, d_0, \mu_0) = y(t, d(t, d_0, \mu_0))$ and the profit of the firm is nil.

Equilibrium on the competitive labor market requires that for each type (t, d_0, μ_0) of individuals, labor demand is equal to labor supply

$$l^d(t, d_0, \mu_0) = 1 \times \Lambda(t, d_0, \mu_0)\,.$$

The right-hand side accounts that each individual supplies one unit of labor and that there is a mass $\Lambda(t, d_0, \mu_0)$ of individuals of type (t, d_0, μ_0), which is given by

$$\Lambda(t, d_0, \mu_0) = \mathrm{e}^{-\int_0^t \lambda(u, d(u, d_0, \mu_0))\mathrm{d}u}\,,$$

where $\lambda(t, d(t, d_0, \mu_0))$ is the hazard rate, as defined in equation (8). Finally, total production is

$$Y = \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_{\hat{d}_0-\epsilon_d}^{\hat{d}_0+\epsilon_d} \int_0^{\bar{T}(d_0,\mu_0)} y(t, d(t, d_0, \mu_0))\, \Lambda(t, d_0, \mu_0)\, \mathrm{d}t\, \frac{1}{2\,\epsilon_d}\, \mathrm{d}d_0\, \frac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0\,.$$

A.2. **Model with PAYG pensions.** We now consider the extension with PAYG pensions as presented in subsection 7.2. The mandatory retirement age is $\hat{t}$ and we assume that $\bar{T}(d_0, \mu_0) \geq \hat{t}$ for all (d_0, μ_0). This means that at least one individual of each type survives

until the retirement age. Following exactly the same approach as above, total production (or equivalently the wage bill) is

$$Y_{<\hat{t}} = \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_{\hat{d}_0-\epsilon_d}^{\hat{d}_0+\epsilon_d} \int_0^{\hat{t}} y(t, d(t, d_0, \mu_0))\, \Lambda(t, d_0, \mu_0)\, \mathrm{d}t\, \frac{1}{2\,\epsilon_d}\, \mathrm{d}d_0\, \frac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0\,.$$

Similarly, the mass of retirees is

$$P_{>\hat{t}} = \int_{\hat{\mu}_0-\epsilon_y}^{\hat{\mu}_0+\epsilon_y} \int_{\hat{d}_0-\epsilon_d}^{\hat{d}_0+\epsilon_d} \int_{\hat{t}}^{\bar{T}(d_0,\mu_0)} \Lambda(t, d_0, \mu_0)\, \mathrm{d}t\, \frac{1}{2\,\epsilon_d}\, \mathrm{d}d_0\, \frac{1}{2\,\epsilon_y}\, \mathrm{d}\mu_0\,.$$

Appendix B. Government intervention

This appendix shows that allowing access to a risk-free asset or introducing a PAYG pension scheme does not alter our main insights on government intervention.

B.1. **Risk-free asset.** We analyze the same two policies as before: a 10% health subsidy and lump-sum transfers of $\omega = 1.7$, both financed by an earnings tax that balances the government budget. Table 5 presents the first two moments of the total net income and age-at-death distributions. In the risk-free asset extension, total net income includes an additional term, $(r + \lambda(d(t)))s(t)$, which is absent in the baseline model, where net income is given by $(1 - \tau(t))y(t, d(t)) + \omega(t)$. Despite this, government action has similar effects in both setups. The health subsidy increases both moments of the age-at-death distribution compared to the no-policy scenario while having little impact on the total net income distribution. Lump-sum transfers, on the other hand, lower both moments of the age-at-death distribution and halve income inequality.

The only notable difference is that in the baseline model, lump-sum transfers reduce mean disposable income compared to the no-policy scenario, whereas in the risk-free asset extension, they increase it. As explained in the main text, mean disposable income in the risk-free asset extension is lower than in the baseline model. Indeed, individuals maintain negative asset positions for most of their lives, making the economy a net borrower (see Figure 10). As a result, interest payments to foreign lenders exceed the income received from them, reducing overall disposable income. Lump-sum transfers discourage preventive care, leading individuals to borrow less for health investments. This, in turn, lowers interest payments to foreign lenders, mitigating the overall negative impact of borrowing. Consequently, mean disposable income rises with lump-sum transfers compared to the no-policy scenario in the risk-free asset extension.

B.2. **PAYGO pension scheme.** In this extension, we set lump-sum transfers to $\omega = 0.8$, rather than $\omega = 1.7$ as in the baseline model. This is because the government is already financing pensions for the elderly. Setting $\omega = 1.7$ would result in very wealthy elderly individuals and an excessively high labor income tax rate. Therefore, we set $\omega = 0.8$, which,

TABLE 5. Income and life expectancy under government intervention

	Age at death		Total net income	
	Mean	Dispersion	Mean	Dispersion
Baseline				
No policy	76.0	19.5	3.43	0.52
Health subsidy	77.0	19.8	3.48	0.55
Lump-sum transfer	75.0	19.2	3.33	0.24
Risk-free asset				
No policy	76.9	19.6	3.04	0.37
Health subsidy	78.1	19.8	3.00	0.36
Lump-sum transfer	75.3	19.1	3.20	0.20
Pension scheme				
No policy	73.8	18.7	2.48	0.52
Health subsidy	74.3	19.9	2.47	0.52
Lump-sum transfer	73.2	18.6	2.47	0.17

together with the pension scheme, requires a labor earnings tax rate of 50% to balance the government budget. This is the same tax rate needed to balance the government budget in the baseline model with lump-sum transfers (see Subsection 6.2). As for health subsidies, we consider the same 10% rate. Table 5 confirms that lump-sum transfers and health subsidies continue to shape the income and age distributions in the same way as in the baseline model.

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