Explaining the increasing inequality in life expectancy across income groups*

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Abstract

We present a novel life-cycle model, grounded in demographic principles, to examine the influence of medical progress, technological progress, and the reduction in age-independent mortality on the rise in life expectancy across socioeconomic groups. Our findings indicate that the expanding disparity in life expectancy across income groups, as well as the growing income inequality among educational groups in the US, can be attributed to a selection process that changes the composition of the initial characteristics (learning ability, schooling effort, and health frailty) of the income groups. This selection process is triggered by the rising income and medical advancements that emerged with the cardiovascular revolution in the 1970s. (JEL: D15, I12, I14, J17, J24)

1 Introduction

Over the past decades we have seen in many countries an increasing inequality not just in terms of income and wealth, but also in terms of health and life expectancy (OECD, 2017; Murtin et al., 2022). For instance, Waldron (2007) shows for the US how the gap in life expectancy at age sixty five between the top half and the bottom half of the income distribution increased monotonically from less than one year for the 1912 birth cohort to almost six years for the 1941 birth cohort. More recent data shows that this trend continues in the US, and the gap turns out to be even larger when more income groups are considered (NASEM and others, 2015). The projected gap in life expectancy between the top and the bottom one percent, for instance, is fourteen years for the 1960 birth cohort (Chetty et al., 2016).

This trend has political, economic, and social consequences. It not only poses challenges to the health sector (Frankovic and Kuhn, 2019), but also affects the public transfer system by making it more regressive, since short-lived and poorer workers end up subsidizing the pension benefits of long-lived and richer workers (Sánchez-Romero and Prskawetz, 2017; Lee and Sanchez-Romero, 2019; Sánchez-Romero et al., 2020). To derive effective policy responses that at least contain if not close the gap and mitigate its consequences, it is necessary to investigate the sources of the increasing inequality in life expectancy. Empirically,

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there are methodological issues that complicate its study, such as reverse causality and the changing meaning of metrics on income and educational attainment over time, among others (Lee and Sanchez-Romero, 2019). To overcome these difficulties, it is necessary to assess the direction of causality between health and income and to account for how inequality is emerging over the life span of individuals, across socio-economic strata, and across cohorts over time.

To this end, we propose a novel life cycle model that accounts for frailty, in the spirit of Vaupel et al. (1979). Individuals decide about their educational attainment, consumption path, labor supply, and health investments. Given an initial health status (initial frailty level) the path of health investments determines the most likely or modal age at death. Dating back to Lexis (1879), the concept of the modal age at death has been used extensively for analyzing longevity in Demography, Actuarial Science, Statistics, and Biology. We propose using the modal age at death as a summary measure of health for several reasons. First, survival functions can be expressed in terms of the modal age at death (Horiuchi et al., 2013) and, therefore, the model generates realistic survival profiles, which are important for relevant policy analysis. Second, akin to the notion of health capital (Grossman, 1972; Fonseca et al., 2020), a higher modal age at death translates into lower mortality. Third, akin to the notion of health deficits (Dalgaard and Strulik, 2014), the modal age at death can be observed across populations and, therefore, has an empirical grounding.[1] Fourth, the modal age at death is calculated using age-specific death rates. Data on age-specific death rates are available since the invention of the life table by John Graunt in 1662 and, therefore, a wealth of data is publicly available data for its estimation. For instance, Fig. 1 shows the evolution of the modal age at death in the US from 1950 to 2019 (see red line) compared to all other countries (black dots) contained in the Human Mortality Database. Fifth, the modal age at death is a category in which individuals would "naturally" think of in terms of planning ahead, since it refers to the age to which individuals assign a high probability of dying. Last, but not least, similar to the health capital model, the modal age at death has no *a priori* upper bound, which allows individuals to invest in postponing their modal age at death as long as it is economically optimal. The fact that the health state has no upper bound, or lower bound, is a desirable feature when integrating this framework in dynamic overlapping generations models.

Within the proposed model, the disparities among individuals in terms of their education, income, life expectancy, and wealth arise from a combination of individual choices and external (childhood) circumstances that shape their demographics and economic decisions.[2] To describe the heterogeneity in childhood circumstances, we use three unobservable characteristics (endowments) – namely learning ability, effort of schooling, and the initial modal age at death – that are equally distributed across each birth cohort at the beginning of life. Differences in the initial modal age at death account for the initial heterogeneity in health. Heterogeneity in the learning ability translate into differences in labor income, while the effort of schooling enables us to accommodate the changing composition of each educational group in terms of learning ability and health across birth cohorts. Altogether, this allows us to account for the fact that with better demographic and economic conditions faced by successive birth cohort, those individuals that remain in the lower educational groups tend

[1] While health deficits are observable at the individual level, their link to survival and the modal age at death can only be established at the population level.

[2] For expositional purposes, we abstract from random health or income shocks over the lifespan of individuals. To justify this assumption, we rely on the study by Huggett et al. (2011) who show that initial heterogeneity (ability to learn, human capital, wealth at age 23) rather than random shocks explain about 61.2, 62.4 and 66.0 percent of the variation in US lifetime earnings, lifetime wealth and lifetime utility, respectively. This is consistent with earlier findings by Cunha et al. (2005) that about 60 percent of earnings variability is foreseen by agents and cannot therefore be attributed to uncertainty.

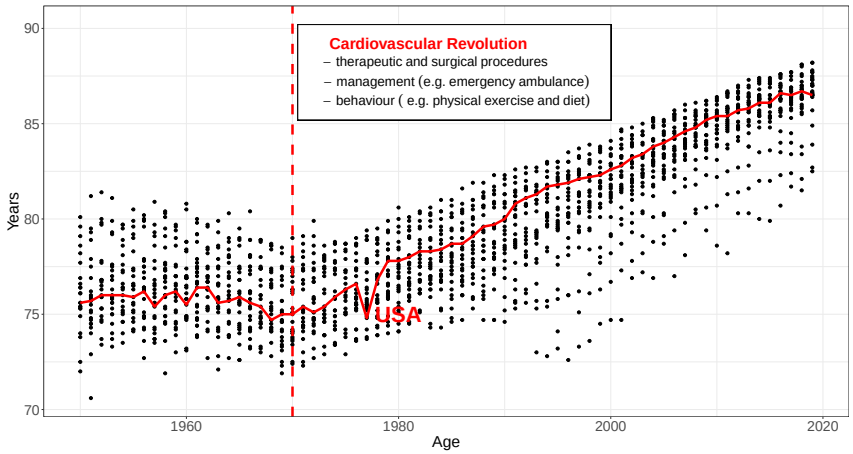


Figure 1: Modal age at death. Dots represent the modal age at death for different countries over time based on data from the Human Mortality Database (HMD). The modal age at death is estimated following Horiuchi et al. (2013).

to be more and more negatively selected.[3]

We apply the model to understand the drivers of increasing inequality in life expectancy by income. In particular, we focus on how exogenous factors, such as the reduction in age-independent mortality (e.g. from infectious diseases or accidents), productivity growth, and medical progress, influence education, income, and health outcomes across different birth cohorts. To do so, we generate results by considering a large number of random draws from the initial distribution of childhood endowments for each birth cohort (taken at twenty year intervals), by solving the life-cycle model for each of these draws, and by thus obtaining distributions of life-cycle outcomes. Then, by allowing individuals to self-select into education according to their initial endowment, we model life-cycle outcomes by educational category and income level. We calibrate the model to US data, seeking to replicate developments of key life-cycle indicators, such as life expectancy, the distribution of education over time, and the increasing gap in life expectancy by education. The model is structurally calibrated using Bayesian melding with the IMIS algorithm (Poole and Raftery, 2000), targeting US data on education, income, and mortality rates. To complete our analysis, we run counterfactual scenarios, in which we shut down each exogenous factor: reduction in age-independent mortality, productivity growth, and medical progress.

Our results show that the model is capable of replicating well the evolution of educational attainment, the distribution of income, and the death rates by education group for the US birth cohorts born in 1900, 1920, 1940, and 1960. In addition, the model is capable of matching the evolution of the life expectancy gap across income groups for the selected birth cohorts, in line with the empirical estimates of NASEM and others (2015) and Chetty et al. (2016) as well as producing realistic results for health spending and the value of life. Running counterfactual analyses, we show that medical progress, the reduction in age-independent mortality, and productivity growth have contributed positively to the increase in life expectancy for all income groups. After controlling for the initial characteristics of individuals, our model suggests that medical progress, the reduction in age-independent mortality, and productivity growth contribute almost equally to the increase in life expectancy

[3]A similar strategy has been applied by Sánchez-Romero et al. (2023) to study the impact of pension reforms across heterogeneous individuals that differ *inter alia* in terms of their life expectancy and income.

across income groups. Therefore, the model suggests that the observed increasing difference in life expectancy across income groups is due to the change in the composition of the initial characteristics, especially the initial heterogeneity in health. This result is consistent with the literature showing that the increasing gap in life expectancy is due to the lower probability of surviving ("survivability") to the ages at which individuals can benefit from the cardiovascular revolution (Dahl et al., 2024), which in our model is controlled by the initial heterogeneity in health. Furthermore, our model results are also consistent with the more classical approach that suggests that wealthier individuals invest more in health, which in turn leverages improvements in medical technology tied to these investments (Hall and Jones, 2007; Frankovic and Kuhn, 2019). These results underscore the importance of creating models capable of controlling for the selection, based on the evolution of the initial characteristics into socioeconomic groups.

The remaining sections of the paper are structured as follows: Section 2 introduces the main theoretical components of the model. Section 3 briefly explains how the initial endowments impact the optimal life cycle choices. Section 4 focuses on the calibration of the model, discussing the process of parameter determination and alignment with empirical data. Section 5 presents our findings. Section 6 summarizes and discusses future directions of research.

2 The model

We consider the life-cycle model of an individual born in year i, in which age t is modeled as a continuous variable between 0 (age 14 in real life) and T (maximum age).[4] Lifetime uncertainty is captured by the survival function $S_i(t)$, which evolves over time according to the following dynamic equation

$$\dot{S}_i(t) = -\mu_i(t, M_i(t))S_i(t). \tag{1}$$

The term $\mu_i(t, M_i(t))$ is the mortality rate of an individual born in year i, which is a function of age (t) and the modal age at death (M). The modal age at death is the most likely age at which an individual will die. This statistical measure is frequently used in demography for analyzing ageing, since it better captures mortality shifts at old age. Moreover, unlike the life expectancy or the median age at death, it is not affected by infant mortality (Horiuchi et al., 2013). To model the relationship between the mortality hazard rate and the modal age at death we follow Canudas-Romo (2008), Horiuchi et al. (2013), Missov et al. (2015), among others, and assume

$$\mu_i(t, M_i(t)) = c_{\mu,i} + be^{b(t-M_i(t))}, \tag{2}$$

where $c_{\mu,i}$ is the Makeham component for the i-th birth cohort and b is the senescence rate. The Makeham component represents the age-independent mortality (or age-independent risk of mortality) and is depicted at the horizontal dashed line in Figure 2. The senescence rate scales the age-related mortality risk. It captures the rate at which mortality exponentially increases with age (see the diagonal dashed line Figure 2). The modal age at death M is the most likely age at which individuals die or the age at which the distribution of deaths peaks (see the vertical dashed line Figure 2).

We assume in the following that $M_i(t)$ represents the health status of an individual born in year i at age t. At time 0 individuals are randomly endowed with an initial modal age at death $M_i(0) = M_0 \sim \mathcal{N}(\mu_M, \sigma_M^2)$, where μ_M is the average initial modal age at death

[4] The maximum age T can be arbitrarily high in the model.

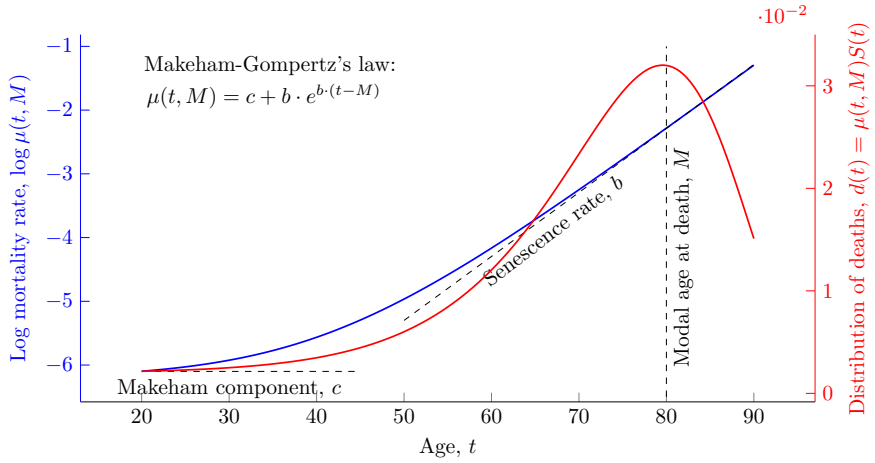


Figure 2: Stylized decomposition of the mortality rate (blue line) and distribution of the age at death (red line) using a Makeham-Gompertz's law.

across the population and where σ_M^2 is the according variance. Individuals can postpone their most likely age at death by investing $m_i(t)$, tantamount to increasing their net health-related consumption, according to the following health production function

$$\dot{M}_i(t) = A_i(E_i, t) m_i(t)^{\gamma_m}. \tag{3}$$

Here, $A_i(E_i, t)$ reflects the effectiveness of health investments for an individual born in year i at age t. We follow Frankovic et al. (2020) and Skinner and Staiger (2015) by modeling medical progress, in the sense of increasing medical effectiveness, as a diffusion process[5]

$$\dot{A}_i = \begin{cases} 0 & \text{if } t < \tau_A - i, \\ g_A(E_i)(A^* - A_i) & \text{if } t \geq \tau_A - i, \end{cases} \tag{4}$$

where τ_A is the year when a new medical technology that reduces the risk of dying is introduced, and A^* is the new medical technological frontier. While Frankovic et al. (2020) and Skinner and Staiger (2015) consider diffusion at the macroeconomic level, we also follow Frankovic and Kuhn (2019) by assuming that access to state-of-the-art medicine at the individual level is depending on education. Thus, the term $g_A(E_i)$ is the inverse of the average time for an individual with educational attainment E_i to have access to the new medical technology. More educated individuals are assumed to have access sooner to the new technology than less educated individuals. The term $\gamma_m \in (0, 1)$ measures the returns-to-scale of health investments in raising the modal age at death. The fact that health can postpone the age at death by slowing down, but not reversing, the ageing process is well in line with the gradual accumulation of health deficits (Rockwood and Mitnitski, 2007; Dalgaard and Strulik, 2014). Moreover, historical data shows that the modal age at death for several countries was roughly constant before 1950 (see Fig. 1). Since we assume that the initial modal age at death is randomly assigned, the value of $M_i(t)$ reflects individual frailty at age t. In particular, given a path of health investments, individuals at age t whose $M_i(t)$ is below the average are expected to die sooner than individuals at the same age t for whom $M_i(t)$ is above average. This is an important feature of the model because it allows to combine the

[5]Solving Eq. (4) yields $A_i(E_i, t) = A^* + (A_i(E_i, \tau_A - i) - A^*)e^{-g(E_i)(i+t-\tau_A)}$ for $t \geq \tau_A - i$.

frailty model (Vaupel et al., 1979) with the Lee-Carter model (Lee and Carter, 1992) (see Section B in the appendix), as the two leading mortality models in the fields of Demography and Actuarial Sciences.

Preferences The expected lifetime utility of an individual born in year i at age 0, conditional on the additional years of schooling (E_i), the maximum age (T_i), the non-health-related consumption path (c_i), and the path of labor supply (ℓ_i), is given by

$$V_i(E_i, T_i, c_i, \ell_i) = \int_0^{T_i} e^{-\rho t} S_i(t) U(c_i(t), \ell_i(t)) dt - \xi_e \int_0^{E_i} e^{-\rho t} S_i(t) dt. \tag{5}$$

The first term on the right-hand side accounts for the discounted and expected stream of utility from consumption and labor (with $U(c,\ell) > 0$, $U'_c(c,\ell) > 0$, $U'_\ell(c,\ell) < 0$, $U''_{cc}(c,\ell) < 0$, $U''_{\ell\ell}(c,\ell) < 0$, and $U''_{c\ell}(c,\ell) \leq 0$). The second term reflects the expected effort of attending school (Sánchez-Romero et al., 2016; Restuccia and Vandenbroucke, 2013; Oreopoulos, 2007), which is an increasing function of the length of schooling, E_i. The parameter ρ is the subjective discount rate and $S_i(t)$ is the survival probability to age t.

Budget constraint We assume that individuals start and terminate their life course (at age T_i) without financial wealth, such that $k_i(0) = k_i(T) = 0$. To rule out adverse selection of health investments on the mortality risk premium, we assume individuals do not purchase annuities. The lifetime budget constraint then reads

$$\int_0^{T_i} (c_i(t) + m_i(t)) e^{-rt} dt = \int_{E_i}^{T_i} w_i(t) H_i(t) \ell_i(t) e^{-rt} dt, \tag{6}$$

where $c_i(t)$ and $m_i(t)$ denote non-health-related consumption and health investments, respectively, and where r denotes the market interest rate. Consumption and health spending across the life course are financed by the labor income earned after the completion of schooling at age E_i. The labor income earned at age t is given by the product of labor supply, $\ell_i(t)$, age-specific productivity $H_i(t)$, and the wage rate per efficient unit of labor $w_i(t)$.

The age-specific productivity increases with schooling, following a standard Ben-Porath technology, and declines at a rate that is proportional to the mortality rate. Thus, we specify the productivity dynamics as

$$\dot{H}_i(t) = \begin{cases} \xi_h H_i(t)^{\gamma_h} - \phi \mu_i(t, M_i(t)) H_i(t) & \text{for } t \leq E_i, \\ f(t, E_i) H_i(t) - \phi \mu_i(t, M_i(t)) H_i(t) & \text{for } t > E_i, \end{cases} \tag{7}$$

where ξ_h denotes the innate learning ability, $\gamma_h \in (0,1)$ denotes the returns-to-scale of education, ϕ denotes the influence of health on labor productivity, $\mu_i(t, M_i(t))$ denotes the mortality rate as a function of time and the modal age at death M_i, and $f(t, E_i)$ denotes the contribution of experience, as measured by the gap $t - E_i \geq 0$, to productivity. Note that our formulation is consistent with the literature showing that productivity declines faster for less healthy individuals (Weil, 2007; Kotschy, 2021; Bloom et al., 2024, among many others), and with the way in which early-life health and educational outcomes jointly determine mid-life health and income (Smith, 2007; van Kippersluis et al., 2010). Note here that a higher modal age at death reduces the mortality rate – see Eq. (2)– and hence positively affects the labor productivity of the individual and allows the individual to remain productive over an extended stretch of time. Furthermore, variation in the modal age at death across individuals of the same age implies variation in their productivity and in the extent to which they are able to retain productivity over their life course.

3 Optimal life cycle allocation

To understand how individuals optimal allocate their resources to postpone their modal age at death, we define three concepts: i) the value of human capital ψ_H, ii) the value of life ψ_S, and iii) the value of delaying the modal age at death ψ_M.

The value of human capital (VoHK) at age t is tantamount to the discounted value of the earnings stream over the remaining working life

$$\psi_i^H(t) := \frac{\partial V_i/\partial H_i(t)}{\partial V_i/\partial k_i(t)} H_i(t) = \int_t^{T_i} w_i(\tau) H_i(\tau) \ell_i(\tau) e^{-\int_t^\tau r ds} d\tau. \tag{8}$$

As becomes evident from the human capital dynamics, see Eq. (7), individuals with a better health status (i.e. greater M_i) are better able to remain productive and enjoy a higher income, ceteris paribus their innate learning ability levels. For this reason VoHK increases with health.

The value of life (VoL) at age t is the willingness to pay for reducing the risk of dying at age t (Rosen, 1988; Costa and Kahn, 2004), which can be calculated as the marginal rate of substitution between financial wealth and the probability of survival

$$\psi_i^S(t) := \frac{\partial V_i/\partial S_i(t)}{\partial V_i/\partial k_i(t)} S_i(t) = \int_t^{T_i} \frac{U(c_i(\tau), \ell_i(\tau)) - \xi_e \mathbf{1}(t \leq E_i)}{U_c(c_i(\tau), \ell_i(\tau))} e^{-\int_t^\tau r ds} d\tau. \tag{9}$$

The VoL is the monetized value of the expected utility at time t of an individual or, equivalently, the monetary value of the discounted stream of utility over the remaining life span. Eq. (9) implies that individuals with higher human capital consume more and enjoy more leisure so that, for this reason, they are willing to pay more for reducing their risk of dying.

The value of delaying the modal age at death (VoDeM) is the willingness to pay for postponing the most likely age at which an individual will die.[6] To fully understand VoDeM, however, we draw on the earlier interpretation of the modal age at death $M_i(t)$ as a proxy for the individual's health or frailty. Notably, improvements in health allow the individual to defer mortality and, at the same time, better preserve it's human capital. Similar to VoHK and VoL, the VoDeM can be calculated as the marginal rate of substitution between the financial wealth and the modal age at death

$$\psi_i^M(t) := \frac{\partial V_i/\partial M_i(t)}{\partial V_i/\partial k_i(t)} = \int_t^{T_i} \frac{-\partial \mu_i(\tau, M_i(\tau))}{\partial M_i(\tau)} (\psi_i^H(\tau)\phi + \psi_i^S(\tau)) e^{-\int_t^\tau r ds} d\tau. \tag{10}$$

VoDeM amounts to the discounted stream of values associated with an improvement in health, as proxied by $M_i(t)$, from age t onward. This value is determined by the sum of the gains in human capital, as valued by $\psi_i^H \phi$, and survival, as valued by ψ_i^S, that are afforded through the reduction in mortality over the remaining life course that is implied by a higher $M_i(t)$.[7] According to equation (10) VoDeM is driven by two opposing forces. On the one hand, VoDeM rises with an increase in the value of human capital (ψ_i^H) and the value of life (ψ_i^S), holding age-related mortality constant. On the other hand, VoDeM declines when the age-related mortality falls, ceteris paribus ψ_i^H and ψ_i^S. Therefore, our model suggests

[6] Note the conceptual difference to the expected age at death, which is the life expectancy (Canudas-Romo, 2008). In a model without mortality uncertainty, the modal age at death coincides with the life expectancy, and with the maximum age.

[7] Kuhn et al. (2015) provide a similar conceptualization of a value of health (or generalized VoL to account for the impact of health on the disutility of labor). Freiberger et al. (2024) provide a generalized value of health, taking into account the prevention of large scale health shocks and acute and chronic treatment following them.

that individuals with high incomes and poor health (measured through a low modal age at death) are the most willing to invest in health, whereas individuals with low incomes and good health are less willing to invest in health. Moreover, if two individuals have the same labor income stream along their lives, the individual with worse health will invest more (resp. less) early (resp. later) in life than the individual with better health.

3.1 Economic problem

The individual maximizes (5) by choosing the optimal path of $m_i(t)$, $c_i(t)$, $\ell_i(t)$ as well as the optimal number of (additional) years of education E_i and the maximum age T_i subject to (1)–(3), (6) and (7). The model is solved using the current value Hamiltonian (see the solution in Section A in the appendix). From the first-order conditions we obtain the optimal investment in health as

$$m_i(t) = \left(\gamma_m A_i(t) \psi_i^M(t)\right)^{\frac{1}{1-\gamma_m}}. \tag{11}$$

Eq. (11) suggests that, akin to the finding in Frankovic et al. (2020), health investments increase with medical effectiveness, A_i, and with the VoDeM (or value of health), ψ_i^M. Plugging (11) into (3) shows how the dynamics of the modal age at death depends on medical effectiveness and the value that each individual assigns to delaying the modal age at death

$$\dot{M}_i(t) = A_i(t)^{\frac{1}{1-\gamma_m}} \left(\gamma_m \psi_i^M(t)\right)^{\frac{\gamma_m}{1-\gamma_m}}. \tag{12}$$

This result has two important implications. First, it implies that increases in the value of human capital or in the value of life (hence in ψ_i^M) raise the modal age at death even when there is no change in medical effectiveness (i.e. $\dot{A}_i = 0$). Second, the contribution of a marginal increase in the value of delaying the age at death to the increase in the modal age at death is γ_m times smaller than that of a marginal increase in the state of health technology.

From the first-order conditions and the envelope conditions we obtain the laws of motion for consumption, labor, and health investments

$$\frac{\dot{c}_i(t)}{c_i(t)} = \sigma_c(t)\big(r - \rho - \mu_i(t, M_i(t))\big), \tag{13}$$

$$\frac{\dot{\ell}_i(t)}{\ell_i(t)} = \sigma_\ell(t)\left(\rho - r + \frac{\dot{w}_i(t)}{w_i(t)} + f(t, E_i) + (1-\phi)\,\mu_i(t, M_i(t))\right), \tag{14}$$

$$\frac{\dot{m}_i(t)}{m_i(t)} = \frac{1}{1-\gamma_m}\left(r + \frac{\dot{A}_i(t)}{A_i(t)} - \frac{-\dot{\psi}_i^M(t)}{\psi_i^M(t)}\right), \tag{15}$$

where $\sigma_c = -U_c/(cU_{cc})$ is the intertemporal elasticity of substitution (IES) in regard to consumption, $\sigma_\ell = U_\ell/(\ell U_{\ell\ell})$ is the IES in regard to labor. Eq. (13) is the standard Euler-condition in the absence of annuities. For $r > \rho$, the consumption path follows an inverted U-shape, with the decline setting in once the mortality hazard becomes sufficiently high. For a given lifetime income, the consumption profile will thus decline sooner for individuals with lower modal ages at death.

Eq. (14) shows how hours worked evolve over the working life according to the difference between ρ and r and the change in the wage rate, in experience, and in health, as proxied by the mortality rate. Eq. (14) implies that individuals work harder today when the interest exceeds the rate of time preference, so that they can save more today; and work harder in the future when earnings are expected to increase either because of productivity growth or

growth in individual experience. The impact of mortality on labor is a priori ambiguous. On the one hand, the mortality risk reduces future consumption and leisure (since both are normal goods), and as a consequence the future labor supply increases. On the other hand, mortality is associated (through a decline in health) with an erosion of human capital, which creates an incentive to work harder today while productivity is still high. The net effect of mortality depends on the impact of health on human capital. For $\phi < 1$, individuals shift labor to older ages, while for $\phi > 1$ individuals work harder today.

Eq. (15) shows the evolution of health investments with age. Health investments tend to increase with age in line with the interest rate (i.e. individuals prefer to save today and invests the proceeds on health later in life) and in line with the rate in which personal access to effective health increases, while they decline as the value of postponing the age at death decreases. It is worth recalling that the lag in access to effective health technology tends to imply that less educated individuals respond less in terms of health utilization to improvements in the state-of-the art medicine, an effect that exacerbates an initial disadvantage (Frankovic and Kuhn, 2019). Furthermore, the value of delaying the age at death declines faster for frailer individuals. Consequently, for a given lifetime income, frailer individuals will reduce their health investments earlier so that inequality in health tends to increase over the life course both in respect to education and in respect to frailty.

The optimal educational attainment is chosen from a set of potential educational possibilities ($E_i \in \mathbf{E}$) as the argument that maximizes the expected utility (5). According to Sánchez-Romero et al. (2016), the optimal educational attainment satisfies that the returns to education exceeds the pecuniary and non-pecuniary cost of education. The pecuniary cost of education is the average return lost in the capital market due to postponing the entrance in the labor market, whereas the non-pecuniary cost of education is determined by the ratio between the effort of schooling and the value of human capital. Noting that individuals expect to die with close to certainty before reaching the maximum age T_i, its optimal choice amounts to a "technical" closure of the model. Further details on the first order conditions can be found in Appendix A.

4 Model calibration

The model is calibrated to represent US males born in the years 1900, 1920, 1940, and 1960; i.e. $i \in \{1900, 1920, 1940, 1960\}$. The wage rate per hour worked is considered to be a function of exogenous wage rate growth (g_w), educational attainment (E_i), learning ability (ξ_h), experience ($t - E_i$), and the health status. In our model the health status is captured by individual frailty, which in turn is represented by the modal age at death. Frailty aggregates all factors affecting mortality, e.g. acquired health deficits, environmental risks and innate biological frailty (Zarulli, 2012). We consider two educational groups: college and non-college. We set the educational attainment, E_i, at 4 years for non-college education, and at 8 years for college education. The returns-to-education γ_h is set at 0.66, similar to Cervellati and Sunde (2013). To estimate the returns to experience, we assume the logarithm of the wage rate can be approximated by a standard quadratic function on experience (see section D for the regression analysis). The main parameter values of the model are summarized in Table 1.

Individuals are assumed to have the following preferences:

$$U(c, \ell) = \log(c) + \alpha \frac{(1-\ell)^{1-\frac{1}{\chi}} - 1}{1 - \frac{1}{\chi}} + \bar{u}. \tag{16}$$

Table 1: Model parameters

	Symbol	Value	Reference
Preferences			
Base level utility	$\bar{u}$	6.0	Target: Avg. value of life of \$6M
IES on cosumption	σ_c	1.0	
IES on labor	χ	0.5	Keane (2022)
Share parameter of consumption	α	2.67	Target: Average labor supply of 33% of available time
Subjective discount factor	ρ	0.0	Lee et al. (2000) and Boucekkine et al. (2002)
Prices			
Exogenous wage rate growth	g_w	g_{wt}	US Bureau of Labor Statistics, Gordon (2017), and Human Mortality Database (2024)
Interest rate	r	2.5%	Target: Average growth rate of consumption of 1%
Initial wage rate	w	1.0	
Mortality			
Senescence rate	b	0.1000	Missov et al. (2015)
Makeham component	$\mathbf{E}[c_{\mu,1900}]$	0.003817	See equation (17)
	$\mathbf{E}[c_{\mu,1920}]$	0.001025	
	$\mathbf{E}[c_{\mu,1940}]$	0.000990	
	$\mathbf{E}[c_{\mu,1960}]$	0.000938	
Health investments			
Initial state of medical effectiveness	$A(0)$	0.15	
Final state of medical effectiveness	A^*	0.20	Fonseca et al. (2020)
Education-specific time to access state-of-the-art medicine	$g_A(E)$		Frankovic and Kuhn (2019); Frankovic et al. (2020) and Skinner and Staiger (2015)
Returns to health investments	γ_m	0.11	Target: Life expectancy at top 1% income for the 1960 birth cohort
Human capital			
Returns to education	γ_h	0.66	Cervellati and Sunde (2013)
Health impact on income	ϕ	0.848	Bloom et al. (2024)
Returns to experience	β_1	0.033959	Section D
Returns to experience-squared	β_2	-0.000533	Section D

Consumption and leisure (i.e., $1-\ell$, when available time is normalized to one) are considered additive separable. We express the utility from consumption in logarithmic terms for consistency with balanced growth. This implies that the intertemporal elasticity of substitution on consumption σ_c is one. Leisure, rather than labor, is modeled in the utility function to account for the fact that the Frisch elasticity is increasing with age and might differ across educational groups (Keane, 2022). The term χ is the intertemporal elasticity of substitution on labor $\sigma_\ell = \chi$. The Frisch labor supply elasticity is $\chi\frac{1-\ell}{\ell}$, and $\bar{u}$ is the base level utility. Following Nishiyama and Smetters (2014), who also model heterogeneous individuals, and Keane (2022) we set the Frisch labor supply elasticity in the vicinity of 1.0 for prime age workers. Thus, we set χ at 0.5 and α at 2.67, so that prime aged individuals work 33.0 percent of their available time, which is similar to the average working hours reported by Nishiyama and Smetters (2014).

The baseline utility $\bar{u}$ is set at 6.0 to match a value of life close to \$6M for the median individual belonging to the 1960 birth cohort.[8] This parameter is important for modeling a rising health share with increases in income (Hall and Jones, 2007). The exogenous wage

[8]The value of $\bar{u}$ needs to be positive and sufficiently large to compensate for the negative effect on lifetime utility of the effort of schooling.

growth for the period 1900–2019 is based on time series data on the real hourly compensation of workers, as taken from the US Bureau of Labor Statistics and Gordon (2017). Consistent with our modeling setting, we detrended this data by the marginal impact of gains in health (proxied by the adult survival ratio) on the wage rate. The adult survival ratio in the US for the period 1933–2019 is taken from the Human Mortality Database (2024). After year 2019 we assume wages to increase at an annual rate of 0.875 percent, which is the last predicted value using a LOESS regression on our constructed time series. We set the interest rate r at 2.5 percent and the subjective discount factor ρ at 0 percent, to match the increase by age of the cross-sectional per capita consumption profile in the US, which is close to 1 percent per year after controlling for the impact of mortality.[9]

Reduction in age-independent mortality. All birth cohorts are assumed to start with the same distribution of the initial modal age at death $\mathcal{N}(\mu_M, \sigma_M^2)$, and hence age-related mortality rates. To account for the evolution of the age-independent mortality risk, we calculate the Makeham component for each birth cohort i (i.e. $c_{\mu,i}$) using the following relationship

$$c_{\mu,i} = \mu_{30,i} - b \cdot \exp(\, b \cdot (30 - \mu_M)), \tag{17}$$

where $\mu_{30,i}$ is the observed mortality rate at age 30 for US males born in 1900, 1920, 1940, and 1960, and b is the senescence rate. We set b at 0.10, which is within the values estimated by Missov et al. (2015). The mortality rates are taken from the US Social Security Administration (Bell and Miller, 2005).

Medical technology. We set the elasticity of the demand for health care (γ_m) at 0.11 to match the life expectancy for the top 1 percent income group for the 1960 birth cohort as estimated by Chetty et al. (2016). In our analysis, we assume that the cardiovascular revolution sets in from the 1970s onwards (Hansen and Strulik, 2017; Ford et al., 2007). We set the initial state of the medical frontier $A(t)$ for $t < \tau_A$ at 0.15, where τ_A is the year 1970. The latest state of the medical frontier is set at 0.20 in order to replicate the increase in the average life expectancy for the cohorts born between 1900 and 1960. The value for the latest state of the medical frontier implies that the medical productivity increases at an annual rate of 0.72 percent over the period 1965 to 2005, which is the same as in Fonseca et al. (2020). Following Skinner and Staiger (2015) we set the time gap between the highly educated (vanguards) and less educated group (laggards) at 10 years. This is in line with Cutler et al. (2006) and Link and Phelan (1995) who show that higher educated individuals enjoy a health premium for their education when a new medical technology is introduced.

Impact of health on income. To parameterize the value of ϕ we follow Bloom et al. (2024) who estimate that a 1 percent increase in adult survival leads to a 1.06 percent increase in labor productivity. In our life cycle model, the estimated value from Bloom et al. (2024) is equivalent to setting ϕ at $0.848 (= 1.06 \cdot 0.8)$.[10] This value implies, according to Eq. (14), that a one percent increase in the mortality rate increases the labor supply in the

[9] In many life cycle model with survival probabilities, it is standard to assume no subjective discount factor (Lee et al., 2000; Boucekkine et al., 2002).

[10] Note that Bloom et al. (2024) regress the logarithm of labor productivity to adult survival, which is defined as the probability of surviving to age 65 conditional on being alive at age 15 (i.e. $S(65)/S(15)$). Given that in our model setup, the logarithm of labor productivity is proportional to the logarithm of adult survival, we need to multiply the estimated coefficient 1.06 from Bloom et al. (2024) by the average adult survival in their sample, which is 0.80.

next period by approximately $0.175 (\approx \chi(1-\phi) = 1.15 \cdot (1-0.848))$ percent, ceteris paribus all other variables. We thus find the net effect of health improvements on the intertemporal supply of labor to be small due to the offsetting impacts of a higher demand for leisure as opposed to a greater productivity. This is in line with recent findings of almost no effect of the gains in health on labor supply (Stephens Jr. and Toohey, 2022).

4.1 Bayesian melding

In addition to the standard parameters, we calibrate the initial unobservable characteristics, namely the initial modal age at death M_0, the learning ability ξ_h, and the schooling effort ξ_e, of the agents using the Bayesian melding technique with the IMIS algorithm (Poole and Raftery, 2000; Raftery and Bao, 2010). The Bayesian melding provides an inferential framework for deterministic models taking into account both model's inputs and outputs. An important feature of this method is that it allows to calibrate the set of initial unobservable characteristics of the model simultaneously without facing the Borel paradox. The (unconditional) distributions of these characteristics are fixed across birth cohorts. The fixation of the characteristics across all birth cohorts allows us to observe compositional changes taking into account selection effects (Sánchez-Romero et al., 2023). We provide the details of the Bayesian melding calculations in Section E.

Marginal posterior distribution of characteristics We define $\theta = (\mu_M, \sigma_M, \mu_{\xi_h}, \sigma_{\xi_h}, \mu_{\xi_e}, \sigma_{\xi_e}, \rho_\xi)$ as a set of inputs. These inputs consist of the mean and standard deviation of the modal age at death (μ_M, σ_M), the learning ability level $(\mu_{\xi_h}, \sigma_{\xi_h})$, the effort devoted to schooling $(\mu_{\xi_e}, \sigma_{\xi_e})$, and the absolute value of the correlation between learning ability and schooling effort (ρ_ξ). These randomly drawn input sets are calibrated using a Bayesian Melding approach to best match the observed life-cycle outcomes in the data. Using the calibrated input sets θ, we generate the marginal posterior distribution of the characteristics (i.e., the initial modal age at death, learning ability level, and effort in schooling), obtained after drawing a sample of 3 000 input sets with repetition Raftery and Bao (2010). This is shown in Figure 10 in Appendix E. The dashed vertical lines illustrate the values of the parameter set with the highest likelihood, which is the one used to generate our results.

These characteristics are assumed to remain constant across cohorts. This assumption implies that, while the unconditional distribution of the characteristics is identical across cohorts, the conditional distribution by socioeconomic group—e.g., education or income—may vary across birth cohorts. This variation enables control for selection effects into specific socioeconomic (SE) groups. In the results section, we use income as the SE variable because mortality by income level has not been used in the calibration process.

Figure 3 illustrates how the distributions of the characteristics for each educational group change across birth cohorts. The upper panels show that college educated individuals born in 1900 had on average a higher initial modal age at death, a lower learning ability, but also a lower schooling effort than individuals with less than college education. As we move to more recent birth cohorts (see bottom panels), the figure shows a convergence between the two educational groups in terms of the initial modal age at death, learning ability, and schooling effort. As a consequence, the learning ability of non-college educated individuals belonging to the 1960 birth cohort is on average lower than of non-college educated individuals born in 1900. Similar patterns are being obtained for Austria in Sánchez-Romero et al. (2023), without considering health as an unobservable characteristic.

Model fit Figure 4 shows the fit of the model to education, demographic, and economic data for selected birth cohorts. The figure is divided into eleven panels. The top panels are

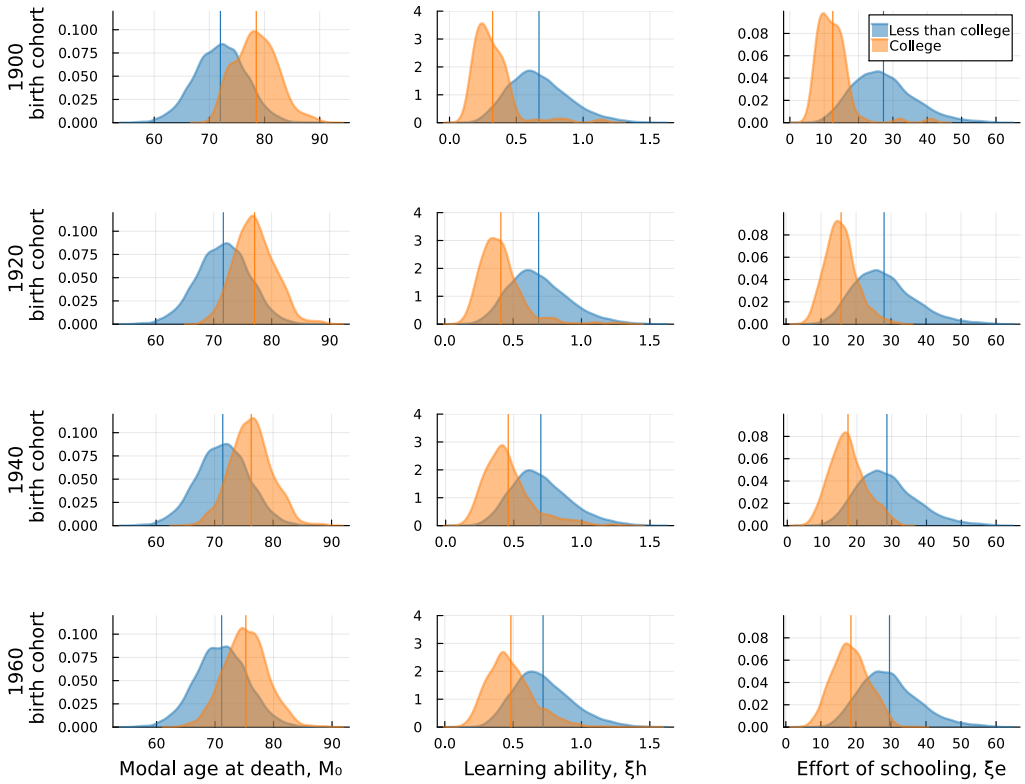


Figure 3: Conditional marginal posterior distributions of individuals' characteristics on educational attainment: selected birth cohorts (1900, 1920, 1940, 1960). The first column on the left shows the distribution of the initial modal age at death (M_0), the second column is the distribution of the learning ability level (ξ_h), and the last column on the right is the distribution of the effort of schooling (ξ_e). The characteristics of individuals with less than college are represented in blue, while the individuals with college education are represented in orange.

devoted to education data (Panel A) and to the distribution of the wage rate at age 40 by education group (Panels B and C). The middle panels depict cohort age-specific mortality rates (in logs) for the selected birth cohorts (Panels D–G). The bottom panels show the age-specific mortality rates (in logs) by education group for the 1940 birth cohort (Panels H and I) and for the 1960 birth cohort (Panels J and K). Figure 4 shows that the model is capable of replicating sufficiently well education, demographic, and economic data, even if Panel A illustrates that the model tends to underestimate the proportion of individuals achieving college education compared to the actual data. Moreover, it should be observed that the age-specific mortality rates generated by the model deviate slightly from the recorded mortality rates for ages 80 and above. However, this is not of deep concern since most of the data for the ages above 80 are still unknown and are based on extrapolations.

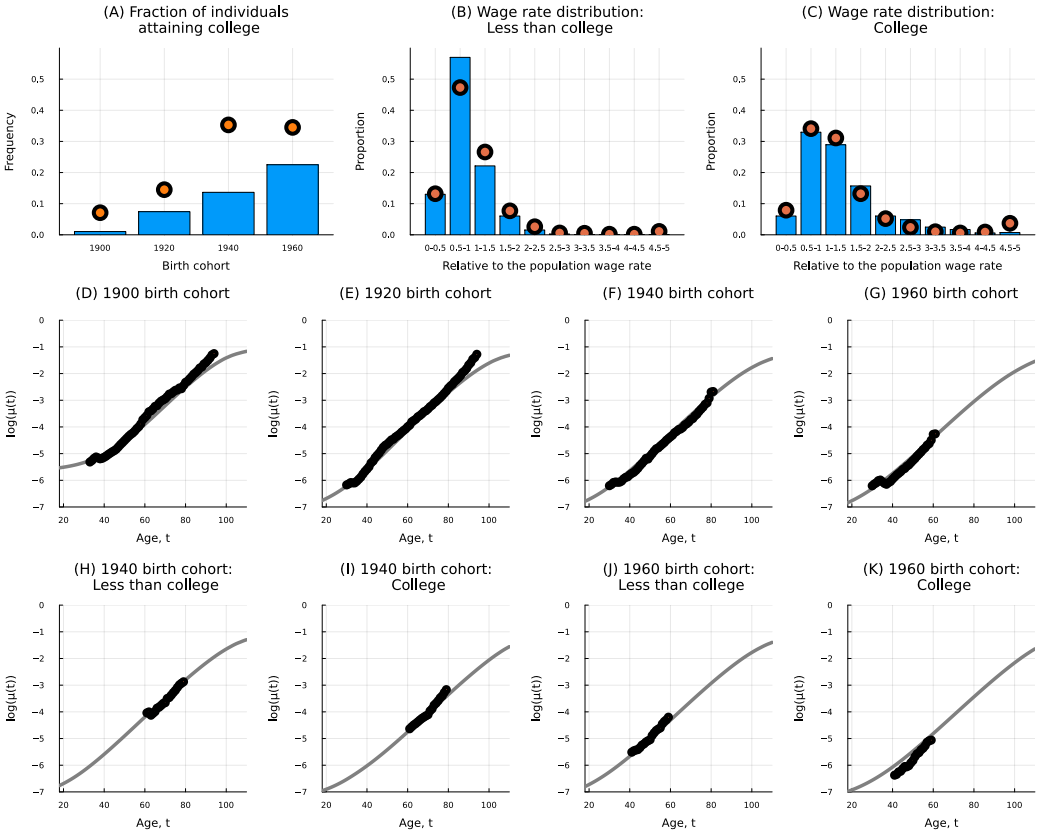


Figure 4: Model fit.

Notes: Black-red dots depict actual data on education, on the wage rate distribution for college and non-college educated workers between ages 40-44 in year 2000, on death rates for each birth cohort, and on mortality rates for college and less than college individuals belonging to the 1940 and 1960 birth cohorts. Source: Data on educational attainment by birth cohort is taken from Goujon et al. (2016); cohort data on age-specific mortality rates by birth cohort have been collected from the US Social Security Administration (Bell and Miller, 2005). Cohort age-specific mortality rates by educational attainment have been calculated using data from CDC (Mortality Multiple Cause Files). See the details of the estimations in appendix C. The wage rate per hour worked for college and non-college educated individuals aged 40-44 in the year 2000 is taken from IPUMS-CPS data and controlled for race, occupation, industry, and state.

4.2 Cross-validation: Evolution of life expectancy by income across birth cohorts

Our model is capable of endogenously generating realistic age-specific mortality rates that differ by socioeconomic group and birth cohort. To validate our simulation results on the evolution of mortality, Figure 5 shows a comparison between our estimated life expectancy at age fifty for males born in 1920 and 1940 by wage quintile (violet and yellow bars) and the estimated life expectancy at age fifty for males born in 1930 (green bars), taken from the Committee on the Long-Run Macroeconomic Effects of the Aging U.S. Population (NASEM and others, 2015). We choose the data on males born in 1930, rather than in 1960, because the mortality data on males born in 1930 relies less on extrapolations. Figure 5 shows that our model is capable of generating a realistic life expectancy at age fifty by income quintile, since the estimates for males born in 1930 by income quintile lie between the values we estimate for the neighbouring 1920 and 1940 cohorts.

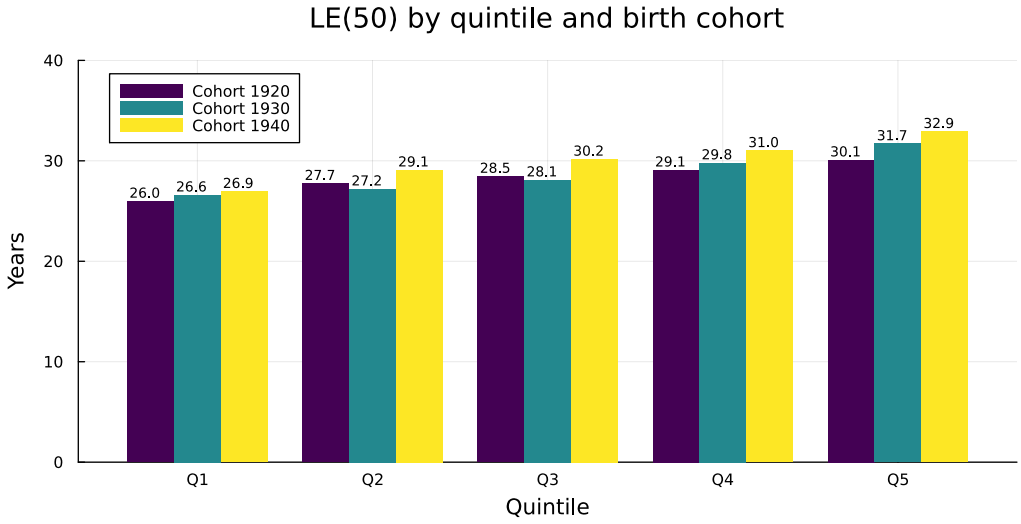


Figure 5: Estimated life expectancy at age fifty for US males born in 1920, 1930, and 1940, by income quintile. *Source: Data for 1920 and 1940 is generated with the life cycle model. Data for 1930 comes from NASEM and others (2015), which was estimated using Health and Retirement Study data.*

To complement the previous validation, Figure 6 shows the expected age at death for forty-year old men born in 1900, 1920, 1940, and 1960 by wage rate percentile simulated by the model. This figure shows a positive relationship between the wage rate percentile and the expected age at death for all birth cohorts, which becomes stronger for the most recent ones. The difference in the expected age at death for forty-year old men between the 5th and 95th percentile groups is close to $2.0 (= 73.9 - 71.9)$ years for the 1900 birth cohort, while this difference is $10.8 (= 86.0 - 75.2)$ years for the 1960 birth cohort. Comparing the red dots to the blue dots shows that the model is capable of generating differences in expected age at death by income that are consistent in shape with recent cross-sectional death rate estimates for the US (Chetty et al., 2016). This is important because the model has not been calibrated to target this data, which therefore cross-validates the model results.

In addition, Figure 6 shows that the widening difference in life expectancy across income

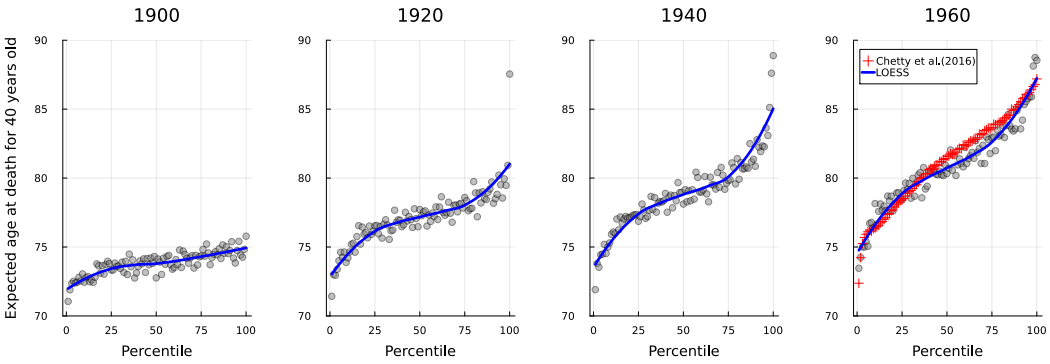


Figure 6: Expected age at death for forty-year old US males born in 1900, 1920, 1940, and 1960 by wage rate percentile (age group 40-44). Notes: Gray dots depict the mean expected age at death in each percentile group generated by the model. The blue line is the predicted value of running a loess regression across all the expected ages at death. The red triangles correspond to the estimated race- and ethnicity-adjusted life expectancy for forty-year old men by household income percentile, 2001–2014 from Bergeron et al. (2022).

groups is the result of a slow increase in life expectancy between the 1900 and 1960 birth cohorts for the lowest percentile groups (3.3 years for the 5th percentile group) as compared to the highest percentile groups (12.1 years for the 95th percentile group). In other words, the years of life gained at age forty per decade are 0.55 and 2.02 for the 5th and 95th percentile group, respectively. In the next subsection, we focus on explaining the divergence in the years gained.

5 Simulation results

In this section, we use the model to clarify the causes behind the increasing mortality gap between socioeconomic groups. To represent different socioeconomic groups, we stratify our sample by wage quintile groups. The wage rate is selected as the defining variable instead of the labor income for two primary reasons. First, wage rate data has been used to calibrate the model, ensuring consistency with empirical data (see Figure 4). Second, labor income is influenced not only by the wage rate but also by labor supply, making it less stable than the wage rate. To facilitate intra-cohort comparisons, we designate age forty as the reference age group.

The model demonstrates that the widening gap in life expectancy across income groups, observed between the birth cohorts of 1900 and 1960, is primarily driven by a selection process shaped by a combination of external factors. These external factors include productivity growth, reductions in non-age-related mortality, improvements in medical technology, and the education-specific timing to benefit from the cardiovascular revolution (CVR).

The model shows that these external factors have disproportionately benefited individuals with a specific set of initial characteristics, such as good initial health, high learning ability, and low schooling effort. Although some of these characteristics were relatively evenly distributed within the 1900 birth cohort, the influence of external factors over time has led to a more pronounced selection of these traits across different income groups.

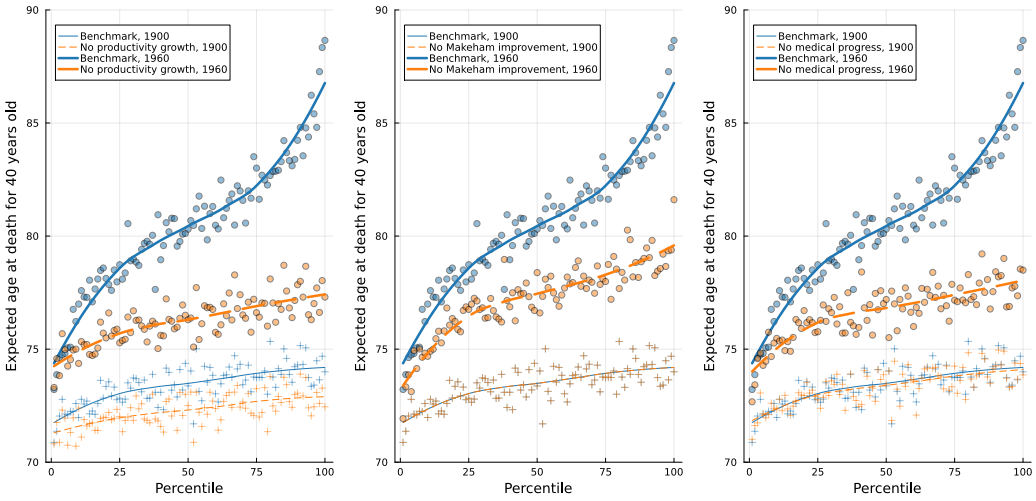
To generate the results, we have used the parameter set θ^* with the highest likelihood obtained through Bayesian melding. Using this parameter set, we have simulated 10 000

initial characteristic profiles (M_0, ξ_h, ξ_e), which reflect the heterogeneity within each of the four U.S. birth cohorts analyzed—1900, 1920, 1940, and 1960—and solved their life cycle models. However, the model could not be solved for all potential (including extreme) combinations of characteristics across all cohorts and counterfactual scenarios. This filtering process reduced the sample size to $n = 7\,415$ unique initial combinations of characteristics, which were consistently applied across cohorts and experimental scenarios.

In the following, we first focus on analyzing how the external factors positively influence the correlation between income and life expectancy. Subsequently we show how the positive correlation between income and life expectancy is driven by a selection process on a set of characteristics that is amplified by the external factors.

5.1 External factors driving life expectancy disparities

To empirically validate that external factors have disproportionately benefited the life expectancy of individuals with higher income, we first analyze model-generated longitudinal data spanning the 1900 to 1960 birth cohorts. Figure 7 illustrates the correlation between income and life expectancy under the benchmark and three counterfactual experiments in which the influence of a single factor has been shut off—no productivity growth (Exp. 1), no improvement in non-age-related mortality (Exp. 2), and no medical progress (Exp. 3). We conducted an additional experiment where we shut off the education-specific timing to benefit from the cardiovascular revolution. However, the influence of this factor is significantly smaller compared to the other three. For the sake of brevity, we have chosen not to present the results here.



(a) Bench. vs. Exp. 1 (b) Bench. vs. Exp. 2 (c) Bench. vs. Exp. 3

Figure 7: Differential impact of productivity growth (Panel a), Improvement in non-age-related mortality (Panel b), and medical progress (Panel c) on life expectancy across income groups: 1960 vs. 1900 birth cohorts.
Source: Authors' simulations.

Specifically, Figure 7 compares, for each percentile of the income distribution, the increase in the expected age at death between the 1900 and 1960 birth cohorts within the benchmark

scenario to the respective increase within the counterfactual experiments. We see in this figure that all three factors have contributed to a widening in the life expectancy gap across income groups. For instance, Panel (a) shows that the productivity-driven increase in income for the 1900 as opposed to the 1960 birth cohort has induced the life expectancy of individuals with higher income to grow at a faster rate compared to lower-income groups. Panels (b) and (c) show that the reduction in non-age-related mortality and medical innovation, respectively, have disproportionally benefited, in terms of life expectancy gains, those in higher income brackets.

Also note in Panel (a) that for the 1900 birth cohort, productivity growth already influences the expected age at death. This is because, in the benchmark scenario, individuals benefit from productivity growth, which allows them to invest more in health and delay their age at death. This effect is absent in Panel (b) because the Makeham component remains the same for the 1900 birth cohort in both simulations. Regarding medical progress, the impact is negligible because, by the time the cardiovascular revolution occurs, individuals born in 1900 are already sixty years old.

To quantify the differential impact of income on life expectancy mediated by each external factor, shown in Figure 7, we specify the regression analysis:

$$\Delta LE^{b}_{1960,p} - \Delta LE^{c}_{1960,p} = \beta^{c}_{0} + \beta^{c}_{1} p + u^{c}_{1960,p}, \tag{18}$$

where $\Delta LE^{b}_{1960,p} - \Delta LE^{c}_{1960,p}$ gives the change in life expectancy between the 1960 and 1900 birth cohorts that is attributable to factor c at the percentile level p. The term p denotes the income percentile and ranges between 1 and 100. β^{c}_{1} is the income gradient of the change in life expectancy attributable to factor c, which is our parameter of interest, and u is a counterfactual–specific error term.

Table 2: Marginal Effect of Income on Life Expectancy by External Factor

	Dependent variable:		
	$\Delta LE^{b}_{1960,p} - \Delta LE^{c}_{1960,p}$		
	Experiment 1	Experiment 2	Experiment 3
p (percentile)	0.064(0.004)	0.064(0.004)	0.049(0.004)
Constant	0.487(0.235)	−0.115(0.241)	0.746(0.210)
Observations	100	100	100
R^2	0.721	0.711	0.653
Adjusted R^2	0.718	0.708	0.649
Residual Std. Error (df = 98)	1.164	1.195	1.043
F Statistic (df = 1; 98)	253.320	241.273	184.136

Note: *p<0.1; **p<0.05; ***p<0.01. Exp. 1: No productivity growth, Exp. 2: No improvement in non-age-related mortality, Exp. 3: No medical progress.

The results, as reported in Table 2, show that individuals who are one percent higher in the income distribution benefited from an additional increase in life expectancy of approximately 0.064 years due to productivity growth, of 0.064 years due to the reduction in non-age-related mortality, and of 0.049 due to medical progress.

5.2 The role of selection

The impact of the external factors—productivity growth, medical technological progress, and reductions in non-age-related mortality—on the dispersion of the expected age at death

across income groups is strongly amplified by selection. This is because individuals with advantageous characteristics—better initial health (M_0), higher learning ability (ξ_h), or lower schooling effort (ξ_e)—are more prone to benefit from productivity growth, medical advances, and the reductions in non-age-related mortality. To understand the selection process, we start by presenting in Table 3 the mean of the main characteristics at age forty by income quintile (obtained in the benchmark simulation) for those individuals who belong to the 1900 and 1960 birth cohorts.

Table 3: Mean values at age forty for the main characteristics by wage quintile and birth cohort (in 2000 US dollars): Benchmark simulation

Wage Quintile	**Initial modal age at death**	**Learning ability**	**Effort of schooling**	**College share**	**Labor income**	**Expected age at death for 40 years old**
Birth cohort: 1900						
Q1	71.60	0.38	16.05	0.02	4 529	72.37
Q2	72.10	0.53	22.25	0.01	6 522	73.17
Q3	72.22	0.64	27.18	0.01	8 325	73.52
Q4	72.28	0.77	31.99	0.01	10 770	73.84
Q5	72.12	1.00	38.02	0.00	16 699	74.13
Birth cohort: 1960						
Q1	69.80	0.44	18.81	0.12	17 746	76.49
Q2	71.40	0.58	24.69	0.13	24 888	78.96
Q3	72.28	0.67	28.67	0.16	31 742	80.51
Q4	72.86	0.76	31.15	0.25	41 363	81.85
Q5	73.99	0.88	32.16	0.47	70 956	84.61

Note: We take as the reference income $37 339, which is the median value of earnings of male full-time, year-round workers in the US in year 2000, see Money Income in the US 2000 (U.S. Census Bureau, 2001).

Table 3 reports for the 1900 birth cohort how despite the clear gradient in terms of learning ability, effort of schooling, and labor income, the life expectancy and the initial modal age at death are quite similar across the quintile groups. In contrast, for the 1960 birth cohort, this table shows that the learning ability and the effort of schooling are more evenly distributed than for the previous cohort. However, the expected age at death, initial modal age at death, college share, and to a lower extent labor income have a clearer gradient now. This is because the external factors reduced the relative cost of education for the 1960 birth cohort and some individuals with good characteristics selected themselves into higher education, which allowed them to have higher income and better health, leaving the group with less than college education with worse characteristics.

To see the influence of this selection process on the expected age at death, we compare in Figure 8 the distribution of the age at death for the 1900 and 1960 birth cohorts under the benchmark and the distribution that would result from taking the distribution of the characteristics by income quintile for the 1900 birth cohort and applying it to the 1960 birth cohort. Comparing panels A and B in Figure 8, we can show that the difference in the expected age at death across quintiles is minimized, which suggests that the selection effect is the main driver of the increasing gap in life expectancy across income groups.

To assess the contribution of the selection of characteristics to the shift in the distribution in the age at death for 40 years old by income, we run additional regressions in which the increase in the modal age at death until age 40 for the 1960 birth cohort is regressed on the characteristics and the educational choice. This regression approach was chosen because it directly isolates the effects of specific characteristics on changes in modal age at death,

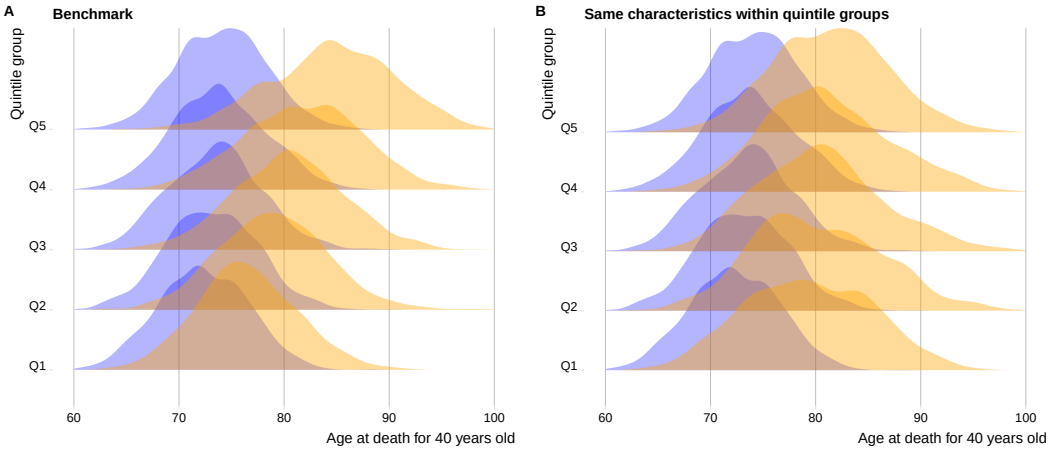


Figure 8: Distribution of the age at death for forty year old individuals by income quintile and birth cohort under two different simulation scenarios.
Note: 1900 birth cohort are in blue, while 1960 birth cohorts are in orange.

providing insights into how these characteristics interact with income to influence outcomes. We choose the increase in the modal age at death because according to (1)–(3) and (11) this measure depends directly on income. Contrary to the life expectancy that depends on income and the initial health state. Moreover, we standardized the covariates in order to better compare the influence of each characteristic on the increase in the modal age at death. The mean and the standard deviation of each characteristic can be seen in Table 5.

The regression equation that we use is:

$$\log(\mathbf{y}) = \mathbf{Z}\boldsymbol{\alpha} + \mathbf{Z}\boldsymbol{\gamma}\mathbf{1}_{\mathrm{C}} + \boldsymbol{\varepsilon}_{\mathrm{C}}, \tag{19}$$

where $\mathbf{y}$ is the $n \times 1$ vector with the expected increase in the modal age at death until age 40 for the n individuals born in year 1960, $\mathbf{Z}$ is the $n \times 3$ matrix containing the standardized covariates $(\mathbf{z}_{M_0}, \mathbf{z}_{\xi_h}, \mathbf{z}_{\xi_e})$, where $z_x = \frac{x-\bar{x}}{\mathrm{sd}(x)}$ is the variable x standardized. $\boldsymbol{\alpha}$ is 3×1 vector of regression coefficients associated with the skill groups NC, while $\boldsymbol{\gamma}$ is a 3×1 vector of regression coefficients capturing the differential effect of college education on the increase in the modal age at death. $\mathbf{1}_{\mathrm{C}}$ is a dummy vector $n \times 1$ indicating whether individuals belong to skill group C, such that $\mathbf{1}_C$ equals 1 for individuals in group C and 0 otherwise. $\boldsymbol{\varepsilon}$ represents the $n \times 1$ vector of residuals. To solve this regression we apply the two-stage residual inclusion (2SRI) method (Terza et al., 2008), because education is a binary endogenous variable in the model.

According to (19) the constant term represents the log of the expected value of the dependent variable when all the characteristics are at their mean values (because scaled variables have a mean of 0). A positive value in α_i implies that a one standard deviation in the characteristic $i \in \{M_0, \xi_h, \xi_e\}$ is associated with a $e^{\alpha_i} - 1$ increase in the modal age at death if the individual is not college educated, and of $e^{\alpha_i + \gamma_i} - 1$ if college educated. Note that income also increases given the positive relationship between the increase in the modal age at death and income. The results shown in Table 4 suggest that the average forty-year-old individual benefits from an increase in the modal age at death of 5.36 years ($= \exp 1.68$). However, individuals with an initial modal age at death or a learning ability level that is one standard deviation higher than the average experience additional increases in the modal age

Table 4: Contribution of each individual characteristic on the increase in the modal age at death (evaluated at age 40) of individuals born in year 1960 by educational choice: Benchmark simulation

	Dependent variable:	
	First stage ($\mathbf{1}_{\mathrm{C}}$) *probit* (1)	Second stage ($\mathbf{y} = \mathbb{E}[\Delta M(40)]$) *glm: Gamma (link = log)* (2)
Residuals		0.126(0.002)
Initial health	1.905(0.060)	0.008(0.0004)
Learning ability	2.780(0.114)	0.062(0.001)
Schooling effort	−6.352(0.205)	−0.009(0.001)
Initial health×College		0.056(0.001)
Learning ability×College		0.157(0.002)
Schooling effort ×College		−0.235(0.003)
Constant	−3.515(0.101)	1.680(0.0004)
Observations	7 415	7 415
Log Likelihood	−1 080.270	4 214.678
Akaike Inf. Crit.	2 168.539	−8 413.356

at death of 0.8 percent and 6.4 percent for non-college-educated individuals, respectively, and 6.6 percent and 24.5 percent for college-educated individuals. This outcome is primarily explained by the higher income resulting from the additional accumulation of human capital. It is also notable that the influence of learning ability is approximately four times greater than that of the initial modal age at death.

In contrast, individuals with a one standard deviation higher effort of schooling benefit 0.9 percent less than the average individual if they are non-college educated and 21.7 percent less if they are college educated. This result is explained by two reinforcing mechanisms. First, a greater effort of schooling increases the cost of attaining college education. Second, this effect is more pronounced for college-educated individuals compared to non-college-educated individuals, as the additional non-pecuniary cost of schooling reduces the value of life, thereby diminishing the willingness to invest in health.

It is also important to understand how the characteristics interacts with each factor. To do so, we calculate the difference between the increase in the modal age at death in the benchmark and in each experiment, in which we shut off the evolution of each external factor, and regress it with respect to the characteristics conditional on the education choice in the benchmark and the counterfactual. The resulting regression equation is:

$$\mathbf{y}^c = \mathbf{Z}\boldsymbol{\alpha}^c + \mathbf{Z}\boldsymbol{\beta}^c \mathbf{1}^c_{\mathrm{NC}\to\mathrm{C}} + \mathbf{Z}\boldsymbol{\gamma}^c \mathbf{1}^c_{\mathrm{C}\to\mathrm{NC}} + \mathbf{Z}\boldsymbol{\delta}^c \mathbf{1}^c_{\mathrm{C}\to\mathrm{C}} + \boldsymbol{\varepsilon}^c, \tag{20}$$

where $\mathbf{y}^c$ is the $n \times 1$ vector representing the difference in the increase in the modal age at death until age forty between the baseline and the counterfactual experiment c for the n individuals born in 1960. $\mathbf{Z}$ is the $n \times 3$ matrix containing the standardized covariates $(\mathbf{z}_{M0}, \mathbf{z}_{\xi h}, \mathbf{z}_{\xi e})$. $\boldsymbol{\alpha}^c$ is the 3×1 vector of regression coefficients associated with the reference group—individuals who are non-college-educated both in the benchmark and in the experiment—while $\boldsymbol{\beta}^c$, $\boldsymbol{\gamma}^c$, and $\boldsymbol{\delta}^c$ are 3×1 vectors of regression coefficients that capture

the marginal effects of belonging to any of the other three potential combinations of educational choices between the benchmark and the experiment. The terms $\mathbf{1}^c_{ee'}$, for $e, e' \in \text{NC}, \text{C}$, are $n \times 1$ dummy vectors indicating whether individuals belong to the education groups non-college or college in the benchmark and in the experiment. $\boldsymbol{\varepsilon}^c$ is the $n \times 1$ vector of residuals for the counterfactual experiment. Similar to (19), we solve this regression using the two-stage residual inclusion (2SRI) method (Terza et al., 2008).

Table 5: Summary statistics: Characteristics of the 1960 birth cohort by experiment

		Experiment 1		**Experiment 2**		**Experiment 3**	
Benchmark→Experiment	Symbol	Mean	Std.	Mean	Std.	Mean	Std.
Total							
Population size	n	7415		7415		7415	
Initial health	M_0	72.07	4.6	72.07	4.6	72.07	4.6
Learning ability	ξ_h	0.67	0.22	0.67	0.22	0.67	0.22
Schooling effort	ξ_e	27.1	8.96	27.1	8.96	27.1	8.96
Non-College→Non-College							
Population size	n	5740		5730		5742	
Initial health	M_0	71.13	4.42	71.12	4.41	71.13	4.42
Learning ability	ξ_h	0.72	0.21	0.72	0.21	0.72	0.21
Schooling effort	ξ_e	29.58	8.29	29.58	8.3	29.57	8.3
Non-College→College							
Population size	n	6		16		4	
Initial health	M_0	72.04	4.83	77.28	5.16	78.27	1.91
Learning ability	ξ_h	0.64	0.27	0.88	0.26	1.12	0.11
Schooling effort	ξ_e	24.88	10.02	25.65	3.61	27.72	1.64
College→Non-College							
Population size	n	1594		1181		1496	
Initial health	M_0	75.06	3.56	74.71	3.63	74.9	3.54
Learning ability	ξ_h	0.49	0.17	0.52	0.17	0.5	0.17
Schooling effort	ξ_e	18.8	5.05	20.09	4.75	19.15	4.89
College→College							
Population size	n	75		488		173	
Initial health	M_0	79.68	3.66	76.62	3.48	78.46	3.4
Learning ability	ξ_h	0.37	0.13	0.39	0.14	0.36	0.15
Schooling effort	ξ_e	13.95	3.76	14.94	3.93	13.69	4.07

Note: Exp. 1: No productivity growth; Exp. 2: No improvement in non-age-related mortality; Exp. 3: No medical progress.

Table 6 shows the critical role of individual characteristics, such as initial health, learning ability, and schooling effort, in shaping the impact of external factors—productivity growth, reductions in non-age-related mortality, and medical progress—on the increase in modal age at death. The results are broken down into three experiments: no productivity growth (Exp. 1), no improvement in non-age-related mortality (Exp. 2), and no medical progress (Exp.3).

The first column presents the impact of individual characteristics on the increase in the modal age at death for forty years old driven by productivity growth. According to (20), the constant term informs that productivity growth had an expected impact on the increase in the modal age at death for forty years old of 1.525 years for the average individual when all the characteristics are at their mean values. Recall that positive values indicate a greater disparity in income and life expectancy. The results reveal that individuals with a one standard deviation higher initial health experience a faster rise in the modal age at death, translating into higher income and life expectancy gains due to increased health and productivity: 0.039 years for the reference group—non-college educated individuals, 0.095

Table 6: Average impact of individual characteristics by educational attainment on the increase in modal age at death at age 40 for the 1960 birth cohort, conditional on external factors (second stage)

	Dependent variable: $\mathbf{y}^c = \Delta M^b(40) - \Delta M^c(40)$		
	Bench - Exp. 1	Bench - Exp. 2	Bench - Exp. 3
Residuals (NC→C)	−0.802(0.036)	−1.363(0.039)	−0.784(0.107)
Residuals (C→NC)	0.774(0.006)	0.888(0.006)	0.784(0.006)
Residuals (C→C)	0.942(0.023)	0.130(0.008)	0.495(0.015)
Non-College→Non-College (Reference)			
Initial health	0.039(0.001)	0.043(0.001)	0.037(0.001)
Learning ability	0.105(0.002)	0.030(0.001)	0.091(0.002)
Schooling effort	−0.079(0.002)	−0.070(0.001)	−0.078(0.002)
Non-College→College			
Initial health	0.056(0.055)	0.109(0.014)	0.206(0.071)
Learning ability	0.312(0.050)	−0.055(0.020)	0.153(0.042)
Schooling effort	−0.316(0.042)	−0.432(0.055)	−0.577(0.225)
College→Non-College			
Initial health	0.343(0.003)	0.283(0.003)	0.332(0.003)
Learning ability	0.946(0.008)	0.802(0.007)	0.921(0.007)
Schooling effort	−1.341(0.010)	−1.066(0.010)	−1.282(0.009)
College→College			
Initial health	0.177(0.009)	0.107(0.003)	0.235(0.006)
Learning ability	−0.114(0.046)	−0.016(0.011)	0.320(0.027)
Schooling effort	0.077(0.050)	−0.174(0.013)	−0.695(0.029)
Constant	1.525(0.001)	0.195(0.001)	1.141(0.001)
Observations	7.415	7.415	7.415
R^2	0.968	0.989	0.978
Adjusted R^2	0.968	0.988	0.978

Note: Exp. 1: No productivity growth; Exp. 2: No improvement in non-age-related mortality; Exp. 3: No medical progress.

(= 0.039 + 0.056) years for those individuals who move from college to non-college due to the increase in productivity, 0.382 (= 0.039 + 0.343) years for individuals who decided to move from non-college to college due to the increase in productivity, and 0.216 (= 0.039 + 0.177) years for those individuals whose educational decision to stay in college is not affected by the increase in productivity. The impact of the increase in productivity for individuals with above-average learning ability levels is stronger than for those with above-average initial health. Note from Table 5 that, despite the fact that the marginal impact of the raise in productivity becomes negative for the learning ability coefficient for those who stay in college, they benefit from productivity growth because they have below-average learning ability levels.

The impact of productivity growth on the increase in the modal age at death for individuals with above-average schooling effort is negative. From Table 5, we observe that the majority of individuals facing this negative effect belong to the group who remain in non-college education, as their schooling effort is above-average. For individuals who remain in college education, the impact is almost negligible (i.e., −0.002 = 0.077 − 0.079).

The second and third columns in Table 6 demonstrate the effects of reductions in non-age-

related mortality and medical progress on the increase in the modal age at death by individual characteristics. Comparing the constant term across the three counterfactual experiments suggests that the impact on the increase in the modal age at death of productivity growth is greater than that of medical progress and much greater than the reduction in non-age related mortality. The sign and magnitude of the coefficients associated to characteristics are very similar to those in the first column, except for individuals transitioning from college to non-college. However, as shown in Table 5, the small sample size of this group prevents us from drawing definitive conclusions.

6 Conclusion

We have devised a novel life-cycle model, based on demographic principles, that endogenously determines the most likely age at death. Individuals decide about their educational attainment, consumption path, labor supply, and health investments, conditional on a set of unobservable characteristics that control for heterogeneity in health, labor income, and education. The model has been calibrated using the Bayesian melding to replicate the evolution of the educational attainment, the distribution of income, and the death rates for US males born in 1900, 1920, 1940, and 1960.

In this paper, we show that our life-cycle model can accurately replicate the widening disparity in life expectancy across cohorts by socioeconomic status. Through counterfactual analyses, we find that medical progress, productivity growth, and the reduction in age-independent mortality have all positively contributed to the increase in life expectancy.

The findings presented in this paper also show the critical role of individual characteristics, such as initial health, learning ability, and schooling effort, in shaping the impact of external factors—productivity growth, reductions in non-age-related mortality, and medical progress—on the increasing mortality gradient across socioeconomic groups. In particular, we obtain that selection effects significantly amplify life expectancy disparities, as individuals with advantageous traits are more likely to benefit from these external improvements. Moreover, the results highlight the dynamic interplay between education, income, and health outcomes, demonstrating that higher learning ability, lower schooling effort, and to a smaller extent lower initial frailty, not only lead to greater life expectancy gains but also contribute to increasing inequalities across income groups.

Our results suggest that in order to model the widening gap in life expectancy across socioeconomic status, it is necessary to develop models that account for the negative selection process within lower socioeconomic groups. The strong role of the selection process across cohorts also suggests that policies, such as universal health insurance, targeted provisioning of (state-of-the-art) health care, or educational campaigns, which are aimed at curbing the socio-economic gradient to the gains in longevity may need to be readjusted over time so as to keep track of the compositional changes, especially in regard to the underlying health traits, within observable social groups.

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A Life cycle model

The problem of the individual is to maximize (5) subject to (1)–(3), (6), (7) and the boundary conditions on capital $k(0) = k(T) = 0$. To solve the problem we use the following current-value Hamiltonian

$$\begin{aligned}\mathcal{H} =& S(U(c,\ell) - \xi_e \mathbf{1}(t \leq E)) \\ &+ \lambda_k(rk + wH\ell\mathbf{1}(t > E) - c - m) \\ &+ \lambda_H\big(\xi_h H^{\gamma_h}\mathbf{1}(t \leq E) + f(t,E)H\mathbf{1}(t > E) - \phi\mu(M)H\big) \\ &- \lambda_S\mu(M)S \\ &+ \lambda_M A m^{\gamma_m},\end{aligned} \tag{21}$$

and the inequality conditions $\nu_1(1-\ell)$ and $\nu_2\ell$. The term $\mathbf{1}(\cdot)$ is an indicator function that takes the value of one when the inequality is satisfied and zero otherwise, and $(\lambda_k, \lambda_H, \lambda_S, \lambda_M)$ are the set of shadow prices associated with the state variables: capital (k), human capital (H), survival (S), and modal age at death (M), respectively.

Solution Given number of years of schooling E, to achieve a maximum the individual problem must fulfill the following necessary conditions for all $t \in [0, T]$:

- First-order conditions (FOCs):

$$\begin{aligned} c: &\qquad \lambda_k = SU_c \\ \ell: &\qquad wH\lambda_k = -SU_\ell + \nu_1 - \nu_2 \text{ for } t > E \\ m: &\qquad \lambda_k = \lambda_M A\gamma_m m^{\gamma_m - 1}\end{aligned}$$

  where ν_1 and ν_2 are the Lagrange multipliers associated with the Kuhn-Tucker conditions.

- Envelope conditions (ECs):

$$\begin{aligned}\dot{\lambda}_k &= \lambda_k(\rho - r) \\ \dot{\lambda}_H &= \lambda_H\big(\rho - \xi_h\gamma_h H^{\gamma_h - 1}\mathbf{1}(t \leq E) - f(t,E)\mathbf{1}(t > E) + \phi\mu(M)\big) - \lambda_k w\ell\mathbf{1}(t > E) \\ \dot{\lambda}_S &= \lambda_S(\rho + \mu(M)) - \big(U(c,\ell) - \xi_e\mathbf{1}(t \leq E)\big) \\ \dot{\lambda}_M &= \lambda_M\rho + \mu'(M)(\lambda_H\phi H + \lambda_S S)\end{aligned}$$

- Transversality conditions (TCs)

$$\lambda_k(T) > 0, \qquad \lambda_H(T) = \lambda_S(T) = \lambda_M(T) = 0$$

- Optimal maximum age condition

$$\mathcal{H}(\mathbf{x}(T^*), \mathbf{X}(T^*), \boldsymbol{\lambda}(T^*), T^*) = 0,$$

  where $\mathbf{x}(T)$ is the vector of controls at time T $(c(T), \ell(T), m(T))$, $\mathbf{X}(T)$ is the vector of states at time T $(k(T), H(T), S(T), M(T))$, and $\boldsymbol{\lambda}(T)$ is the vector of adjoints at time T $(\lambda_k(T), \lambda_H(T), \lambda_S(T), \lambda_M(T))$. This condition implies that the optimal maximum longevity T is reached when the value of life is equal to the instantaneous consumption; i.e. $U/U_c = c$. For computational issues, we define a $T^{\star\star}$ such that $S(T^{\star\star}) \approx 10^{-3}$ and impose the restriction that $T = \min\{T^*, T^{\star\star}\}$.

- Optimal educational attainment: Given the optimal path of consumption, health investments, labor supply, and the maximum age condition for each possible E, the individual chooses the number of years of education that maximizes (5).

B Frailty model

In this section we show that our mortality hazard rate contains features of the health-deficit model (Dalgaard and Strulik, 2014), the Lee-Carter model Lee and Carter (1992), and the frailty model (Vaupel et al., 1979). To do so, we use the definition of the mortality hazard rate in terms of the modal age at death

$$\mu(t, M(t)) = c_\mu + be^{b(t-M(t))}.$$

Substituting the modal age at death at time t by the integral from 0 to t of (3) gives

$$\mu(t, M(t)) = c_\mu + be^{bt-b\left(M_0+\int_0^t A(s)m(s)^{\gamma_m}ds\right)}.$$

where $M_0 \sim \mathcal{N}(\mu_M, \sigma_M^2)$ is the initial modal age at death, which is distributed according to a normal distribution with mean μ_M and standard deviation σ_M, and the integral term is the total increase in the modal age at death from age 0 until age t, which we will denote from now on by $\Delta M(t) = \int_0^t A(s)m(s)^{\gamma_m}ds$. Multiplying the terms inside the parenthesis by the natural rate of ageing, we have

$$\mu(t, M(t)) = c_\mu + be^{bt-bM_0-b\Delta M(t)}. \tag{22}$$

Ageing process. A realistic feature of the health-deficit model (Dalgaard and Strulik, 2014) is that individuals accumulate health deficits as they age. The higher are the number of health deficits, the faster they accumulate (i.e. the faster is the ageing process), and the greater is the probability of dying. Individuals can reduce the speed of aging by investing in health. This feature is shared in our framework. Taking the logarithm of the difference between the mortality rate and the age-independent mortality (i.e. the Makeham component) and differentiating with respect to t gives the speed of ageing or the senescence rate

$$\frac{\partial}{\partial t}\log(\mu(t, M(t)) - c_\mu) = b(1 - A(t)m(t)^{\gamma_m}). \tag{23}$$

The above equation shows that similar to the health-deficit model (Dalgaard and Strulik, 2014), individuals can reduce the senescence rate from it's natural level, b, by investing in health. In particular, given an initial modal age at death M_0 our model implies that individuals who invest more (resp. less) in health will have a lower (resp. higher) senescence rate and hence a higher probability of living longer.

Lee-Carter model. Similar to the Lee and Carter (1992) model, our senescence rate will not change monotonously over the life cycle due to the fact that health investments vary non-monotonously over the life span. Rearranging Eq. (22) and taking the log, we obtain

$$\log\left(\mu(t, M(t)) - c_\mu\right) = \underbrace{\log be^{b(t-\mu_{M_0})}}_{\mathbf{a}_t} + \underbrace{(-b)\Delta M(t)}_{\mathbf{b}_x\mathbf{k}_t} + \underbrace{(-b)(M_0 - \mu_M)}_{\epsilon},$$

where the first term on the right hand side corresponds to the general shape of the mortality schedule $\mathbf{a}_t$, the second term corresponds to the rate of decline in mortality over time $\mathbf{b}_x\mathbf{k}_t$, and the last term is the residual term $\epsilon \sim \mathcal{N}(0, b^2\sigma_M^2)$ in the Lee-Carter model.

Frailty model. By rearranging the terms in (22) we obtain the standard frailty model (Vaupel et al., 1979) augmented with gains in senescence

$$\begin{aligned}\mu(t, M(t)) &= c_\mu + e^{\log b - bM_0 + bt - b\Delta M(t)} \\ &= c_\mu + Zae^{b(1-t^{-1}\Delta M(t))t},\end{aligned} \tag{24}$$

where

$$Z = \exp\left\{-b(M_0 - \mu_M) - \frac{b^2\sigma_M^2}{2}\right\}$$
$$a = \exp\left\{\log b - b\mu_M + \frac{b^2\sigma_M^2}{2}\right\}$$

There are several key findings from (24). The first thing to notice is that the mortality hazard rate exactly coincides with the frailty model when there are no health investments (i.e. $\Delta M(t) = 0$). Second, given that $\mathbf{E}[Z]$ is equal to zero by definition, we obtain that in two populations with similar μ_M, the population with the highest σ_M^2 will have on average a higher mortality than the population with the lowest σ_M^2 (Vaupel et al., 1979).

C Age-Specific Mortality Rates by Educational Attainment

We compute period life tables by education based on the *Mortality Multiple Cause Files* provided by the Centers for Disease Control and Prevention (from now on *CDC data*).[11] These data contain information about the resident status, age, education level, and the cause of death for defunct people on a yearly basis. The *CDC data* for years earlier than 1989 do not provide information about education, and therefore we restrict the data to the 1989-2019 period. We only consider US-residents for which age is known, reported in years, and smaller or equal to 100. We recode the causes of death following the shortlist of causes of death provided by the *Human Cause-of-Death Database (HCD)*.[12] The shortlist of causes of death is the same for all countries contained in the HCD database and contains 16 different causes of death. The causes of death in the CDC data before 1999 are coded using ICD-9 codes, which for comparability with successive years we convert into ICD-10 codes using the conversion tables provided by the Canadian Institute for Health Information (CIHI).[13] Some ICD-9 codes cannot be converted. As these codes are unrelated to heart diseases, we group them and convert the underlying cases to *Other causes*. It is important that heart diseases are converted correctly since they are used to build the counterfactual mortality rates under the presumption that the cardiovascular revolution had not occurred.

We merge the IPUMS USA population data with the CDC data to estimate the mortality rates by education for male US-residents using the year of observation, age, and highest educational attainment. We are able to compute mortality rates by educational attainment for 1990, 2000, and yearly from 2000 to 2019 because the IPUMS USA population data are available only on a decennial basis for years earlier than 2000. The educational attainment, for both the CDC data and the IPUMS USA data, is recoded such that we obtain two distinct groups, college and less than college. We assign individuals with more than 12 years

[11] https://www.cdc.gov/nchs/data_access/vitalstatsonline.htm

[12] https://www.mortality.org/Data/HCD

[13] https://secure.cihi.ca/free_products/conversion-tables-ICD10CA-ICD9-ICD9CM-CCI-CCP-en.xlsx

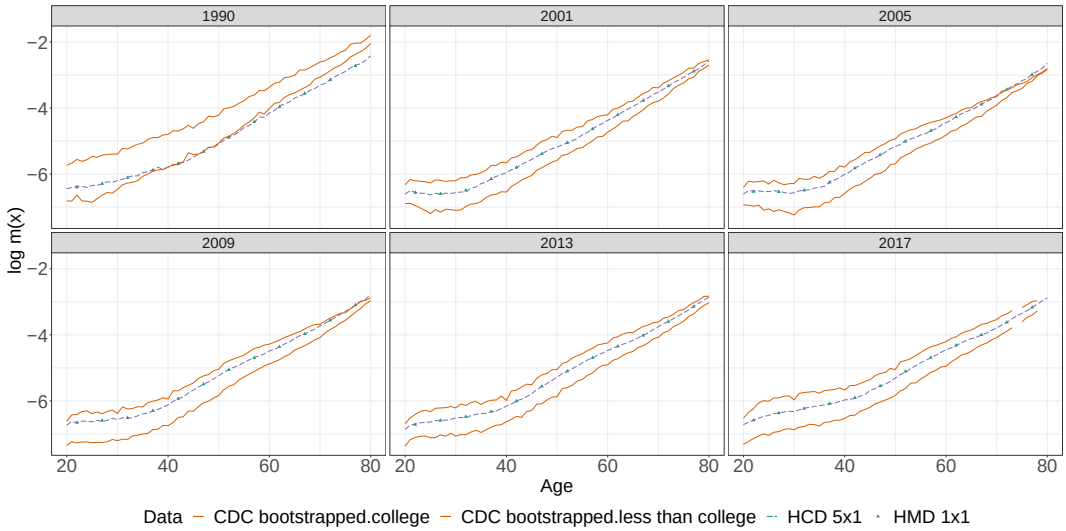

Figure 9: Mortality Rates: mortality rates by educational attainment as estimated from CDC data as compared to the mortality rates for males obtained from the Human Mortality Database (HMD) and the Human Cause of Death Database (HCD).

of completed education to the *college* group, with 12 or less than 12 years of completed education to the *less-than-college* group, whereas individuals with no formal education are not considered. Since the CDC data contain observations with unknown education, we estimate bootstrapped mortality rates by cause of death for US-resident males aged between 20 and 95 years. First, we exclude observations with unknown educational attainment from the CDC data. Then we compute the share of observations excluded by age and year of observation, exclude the same share of observations by age and year of observation from the IPUMS USA population data - the educational attainment of the excluded individuals is random - and compute the mortality rates by year, age, education and cause of death using the resulting population by year, age and education. We repeat this process 1,000 times and average over the obtained mortality rates to obtain robust bootstrapped mortality rates by year, age education and cause of death. If there is no unknown educational attainment for a combination of age, year and cause of death, we do not need to estimate bootstrapped mortality rates. The mortality rates by education and year are not smooth for higher ages, most of the time mortality rates start becoming erratic from age 80 to 85 onwards. To smooth the mortality rates we apply a Kannisto model for ages above 80. Finally, we estimate the life table by education for males resident in the United States between ages 20 and 95. In Figure 9 we compare the estimated log-mortality rates by education to the log- mortality rates from the Human Mortality database (HMD) and the Human Cause-of-Death database (HCD).[14][15] Our estimates appear to be consistent with the HMD and HCD mortality rates since both lie between the mortality rates for higher (college) and lower (less-than-college) educated people.

[14] https://www.mortality.org/
[15] https://www.mortality.org/Data/HCD

D Wage rate estimation

To estimate the components (β_1, β_2, ϕ) of the wage equation we collected individual data from IPUMS USA on labor income and on the average number of hours worked by age, year, education, industry, occupation and race. We calculated the years of experience as the difference between the age of each individual and the age at completing school, and we grouped the years of experience into five-year intervals. Before estimating the returns to experience (β_1, β_2), we regressed the logarithm of the wage rate per hour worked using the fixed effects model $\log w_{etios} = \rho_E E + \gamma_e + \gamma_t + \gamma_i + \gamma_o + \gamma_s + \epsilon_{etios}$, where ρ_E is the return to education, E denotes the additional years of education starting from age 14, γ_e is the experience fixed effect, γ_t is the year fixed effect, γ_i is the industry fixed effect, γ_o is the occupation fixed effect, and γ_s is the state fixed effect.

Table 7 presents results for three alternative models. In model (1) both education groups (college and non-college) are included into the regression, whereas models (2) and (3) provide regressions for the two education groups in separate. From the regression results on the years of experience from model (1) we approximated the working ability profile using a quadratic function of the years of experience $\hat{\gamma}_e = \text{Ct} + \beta_1 \text{Exp} + \beta_2 \text{Exp}^2 + u_e$, where Ct is a constant term, Exp is the years of experience, and u_e is the error term.

Table 7: Estimated return on experience by education

	Dependent variable: log(wage per hour)		
	Aggregated education	college	less than college
	(1)	(2)	(3)
5 to 9 years of experience	0.223***	0.211***	0.171***
	(0.010)	(0.014)	(0.017)
10 to 14 years of experience	0.381***	0.404***	0.319***
	(0.010)	(0.013)	(0.016)
15 to 19 years of experience	0.494***	0.510***	0.421***
	(0.010)	(0.013)	(0.016)
20 to 24 years of experience	0.564***	0.552***	0.471***
	(0.009)	(0.012)	(0.015)
25 to 29 years of experience	0.585***	0.560***	0.506***
	(0.009)	(0.012)	(0.015)
30 to 34 years of experience	0.593***	0.547***	0.511***
	(0.010)	(0.013)	(0.016)
35 to 39 years of experience	0.630***		0.514***
	(0.013)		(0.017)
returns to education: college	0.047***		
	(0.001)		
returns to education: less than college	0.035***		
	(0.001)		
returns to education		0.060***	0.024***
		(0.001)	(0.001)
Constant	2.167***	2.058***	2.400***
	(0.043)	(0.068)	(0.065)
Total Weighted Observations	12,552,970	6,532,227	6,020,743
Observations	101,258	58,800	42,458
Adjusted R^2	0.316	0.238	0.176
Residual Std. Error	0.602 (df = 101172)	6.882 (df = 58716)	6.514 (df = 42373)
Note:			*p<0.1; **p<0.05; ***p<0.01

E Bayesian melding

Let $F(\cdot)$ denote our life-cycle model. The model is used to replicate the educational, mortality, and income distribution of the cohorts born in 1900, 1920, 1940, and 1960 in the US. Each cohort is assumed to be represented by a set of $J = 1\,000$ heterogeneous agents, whose permanent and unobservable characteristics are randomly assigned at birth and are identical across cohorts. Let the set of permanent and unobservable characteristics of the j-th agent be $(M_{0j}, \xi_{h,j}, \xi_{e,j})$, where M_{0j} is the initial modal age at death, ξ_{hj} is the learning ability level, and ξ_{ej} is the effort of schooling. The initial modal age at death M_0 is assumed to be Normal-distributed $\mathcal{N}(\mu_M, \sigma_M^2)$. The pair of unobservable characteristics (ξ_h, ξ_e) are assumed to be drawn from a bivariate Clayton copula formed by two Gamma distributions

$$(\xi_h, \xi_e) \sim C\left(\Gamma_1\left(\frac{\mu_{\xi_h}^2}{\sigma_{\xi_h}^2}, \frac{\sigma_{\xi_h}^2}{\mu_{\xi_h}}\right), \Gamma_2\left(\frac{\mu_{\xi_e}^2}{\sigma_{\xi_e}^2}, \frac{\sigma_{\xi_e}^2}{\mu_{\xi_e}}\right)\right) \tag{25}$$

Let $\mathcal{D}$ denote the combination of the normal distribution and the copula; i.e. $\mathcal{D} = \mathcal{N} \times C(\Gamma_1, \Gamma_2)$. Our calibration problem involves seven parameters or inputs,

$$\theta = (\mu_M, \sigma_M, \mu_{\xi_h}, \mu_{\xi_e}, \sigma_{\xi_h}, \sigma_{\xi_e}, \rho_\xi) \in \Theta \subseteq \mathbb{R}^7$$

taken from the feasible parameter set

$$\Theta = (\underline{\mu_M}, \overline{\mu_M}) \times (\underline{\sigma_M}, \overline{\sigma_M}) \times (\underline{\mu_{\xi_h}}, \overline{\mu_{\xi_h}}) \times (\underline{\sigma_{\xi_h}}, \overline{\sigma_{\xi_h}}) \times (\underline{\mu_{\xi_e}}, \overline{\mu_{\xi_e}}) \times (\underline{\sigma_{\xi_e}}, \overline{\sigma_{\xi_e}}) \times (\underline{\rho_\xi}, \overline{\rho_\xi}),$$

where the symbols $\underline{x}$ and $\overline{x}$ denote the minimum and maximum value of parameter x, respectively. The pair $(\mu_M,\, \sigma_M)$ is the mean and the standard deviation of the initial modal age at death, μ_{ξ_h} is the average learning ability, μ_{ξ_h} is the average effort of schooling, σ_{ξ_h} is the standard deviation of the learning ability, σ_{ξ_e} is the standard deviation of the effort of schooling, and ρ_ξ is the absolute value of the correlation between learning ability and effort of schooling.

Let the prior distribution on inputs $p(\Theta)$ be the product of uninformative priors $p(\Theta) = \mathcal{U}(\Theta)$. Let $\phi \in \Phi$ be the set of outputs generated from the model F given the inputs θ; i.e. $F(\theta) = \phi$. Let us denote by ϕ^C the set of outputs used for the calibration and by ϕ^N the set of outputs not used for the calibration, i.e. $\phi = (\phi^C, \phi^N)$. The outputs ϕ^C are a sequence of economic and demographic data for the 1900, 1920, 1940, and 1960 birth cohorts, which we will fit to actual data. In particular, we generate: i) the fraction of individuals within each birth cohort with non-college education $\phi^{1C}(\theta)$; ii) the average cohort-life expectancy at age 14 for each birth cohort $\phi^{2C}(\theta)$; iii) the wage rate distribution of workers aged 40–44, who were born in year 1960, with non-college education $\phi^{3C}(\theta)$ and with college education $\phi^{4C}(\theta)$, respectively; iv) education- and age-specific mortality rates for the 1940 and 1960 birth cohorts; and v) age-specific mortality rates for the 1900, 1920, 1940, and 1960 birth cohorts.[16] Let the prior distribution on outputs used for the calibration $q(\Phi^C)$ be

$$q(\Phi^C) = \prod_{j=1}^{4} q_j(\Phi^{jC}) \text{ with } q_j(\Phi^{jC}) = \begin{cases} 1 & \text{if } ||\phi^{jC}(\Theta) - \widehat{\phi}^{jC}|| < \Delta_j\sqrt{n}, \\ \epsilon & \text{otherwise,} \end{cases}$$

where $\widehat{\phi}^{jC}$ is the vector of observed data for sequence j and Δ_j is the maximum threshold allowed for the difference between the generated and the observed sequence of data j.

[16] To generate the data for each birth cohort, we draw a sample of N individuals with their unobservable characteristics for a given set of inputs $\theta \in \Theta$ using the distribution $\mathcal{D}$.

Let the likelihood of the model's output for calibration ϕ^C be given by

$$\mathcal{L}(\phi^C|\text{data}) \propto \exp\left\{-\frac{1}{2}\sum_{j=1}^{4}(\phi^{jC}(\theta) - \widehat{\phi}^{jC})^T \mathbf{W}_j^{-1} (\phi^{jC}(\theta) - \widehat{\phi}^{jC})\right\} \tag{26}$$

where $\mathbf{W}_j$ is the weighted matrix for the sequence of data j, which is assumed to be an identity matrix $\mathbf{I}$.

We implement the Bayesian melding with the IMIS algorithm, with the prior as importance sampling distribution. Specifically, we pursue the following steps:

- Initial stage:
  1. Draw $N_0 = 7\,000$ independent and identically distributed (i.i.d.) samples, $\{\theta_1, \ldots, \theta_{N_0}\}$, from the joint prior distribution on inputs $p(\Theta)$.
  2. For each θ_i sampled, run the model to obtain the set of outputs, i.e. $F(\theta_i) = \phi_i^C$.
  3. Calculate the likelihood of each model output ϕ_i^C
  $$\mathcal{L}(\phi_i^C|\text{data}) \text{ for } i = \{1, \ldots, N_0\}$$
  4. Construct the importance weights for each θ_i
  $$\omega(\theta_i) \propto \frac{q(\phi_i^C)\mathcal{L}(\phi_i^C|\text{data})}{\sum_{i=1}^{N} q(\phi_i^C)\mathcal{L}(\phi_i^C|\text{data})} \text{for } i = \{1, \ldots, N_0\}$$
- Importance Sampling Stage: For $k = 1, 2, \ldots$
  1. Choose the current maximum weight input as the center
  $$\theta^{(k)} = \arg\max_{\theta \in \{\theta_1, \ldots, \theta_{N_{k-1}}\}} \left(w(\theta_1), \ldots, w(\theta_{N_{k-1}})\right)$$
  .
  2. Estimate $\Sigma^{(k)}$ as the weighted covariance of the $B = 100$ inputs with the smallest Mahalanobis distances to $\theta^{(k)}$, where the distances are calculated w.r.t. the covariance of the prior distribution and the weights are $\left(w(\hat{\theta}_1) + \frac{1}{N_k}, \ldots, w(\hat{\theta}_B) + \frac{1}{N_k}\right)$. The Mahalanobis distance is:
  $$d(\theta, \theta^{(k)}) = \sqrt{(\theta - \theta^{(k)})' * Q * (\theta - \theta^{(k)})}$$
  where Q is the covariance matrix of the prior distribution on inputs
  3. Sample B new inputs $(\theta_{N_{k-1}+1}, \ldots, \theta_{N_{k-1}+B})$ from the multivariate Gaussian distribution
  $$H_k : \mathcal{N}(\theta^{(k)}, \Sigma^{(k)}) \tag{27}$$
  4. Calculate the likelihood of the new inputs and combine the new inputs with the previous ones. Form the importance weights:
  $$w(\theta_i) = c \cdot q_2(\phi_i^C)\mathcal{L}(\phi_i^C) \times \frac{p(\theta_i)}{q^{(k)}(\theta_i)} \text{ for } i = N_{k-1} + 1, \ldots, N_k. \tag{28}$$
  with $q^{(k)}(\theta_i) = \frac{N_0}{N_k} p(\theta_i) + \frac{B}{N_k} \sum_{s=1}^{k} H_s(\theta_i)$, where $H_s(\theta_i)$ is the probability of having input θ_i in the multivariate Gaussian distribution $\mathcal{N}(\theta^{(s)}, \Sigma^{(s)})$, c is a scaling factor that guarantees $\sum_i^{N_k} w(\theta_i) = 1$, and $N_k = N_0 + kB$.

- Resample stage: Once the expected fraction of unique points in the resample $\sum_{i=1}^{N_k}(1-(1-w(\theta_i))^J)$ is at least 0.632 out of 3 500 random draws, we resample $J = 200$ inputs with replacement from $\{\theta_1, \ldots, \theta_{N_k}\}$ with weights $\{\omega(\theta_1), \ldots, \omega(\theta_{N_k})\}$ to approximate the posterior distribution of the inputs and outputs.

The model and the Bayesian melding with the IMIS algorithm have been programmed in Julia. To find the initial co-state variables of the life cycle model, we have used the Mixed Complementary Problems solvers contained in the NLSolve package. The Bayesian melding has reached the stopping criteria after 55 iterations.

Marginal posterior distribution of inputs Figure 10 shows the marginal posterior distribution of the model inputs θ. Figure 10 suggests the existence of multiple combinations of $\theta \in \Theta$ that provide a likely fit to the data. Therefore, the Bayesian melding is a good option for calibrating the model in order to avoid invertibility problems (Poole and Raftery, 2000). Moreover, an important feature of the calibration strategy is that it is not necessary to run sensitivity analysis against these parameters. This is because the calibration already provides the results of combining different parameters values.

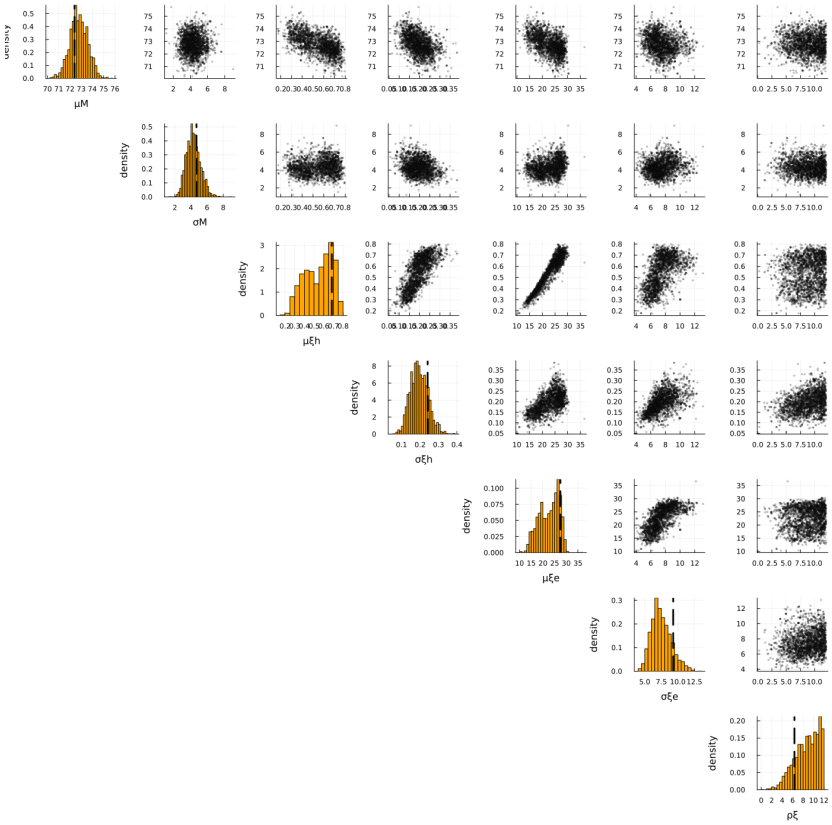

Figure 10: Marginal posterior distributions of inputs θ. The terms μ_M, σ_M, μ_{ξ_h}, σ_{ξ_h}, μ_{ξ_e}, σ_{ξ_e}, ρ_ξ stand for the average initial modal age at death, the standard deviation (std.) of the initial modal age at death, the mean learning ability, the std. of the learning ability, the mean effort of schooling, the std. of the effort of schooling, and the correlation between the learning ability and the effort of schooling, respectively.