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The genesis of the golden age: Accounting for the rise in health and leisure ☆

Carl-Johan Dalgaard [a], Holger Strulik [b,*]

[a] Department of Economics, University of Copenhagen, Øster Farimagsgade 5, Building 26, DK-1353 Copenhagen, Denmark
[b] University of Goettingen, Department of Economics, Platz der Goettinger Sieben 3, 37073 Goettingen, Germany

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ABSTRACT

We develop a life cycle model featuring an optimal retirement decision in the presence of physiological aging. In modeling the aging process we draw on recent advances within the fields of biology and medicine. In the model individuals decide on optimal consumption during life, the age of retirement, and (via health investments) the timing of their death. Accordingly, "years in retirement" is fully endogenously determined. Using the model we can account for the evolution of age of retirement and longevity across cohorts born between 1850 and 1940 in the US. Our analysis indicates that 2/3 of the observed increase in longevity can be accounted for by wage growth, whereas the driver behind the observed rising age of retirement appears to have been technological change in health care. Both technology and income contribute to the rise in years in retirement, but the contribution from income is slightly greater.

1. Introduction

A 20 year-old US male who was born in 1850 could expect to live another 43.7 years upon reaching his 20th birthday; in the 1940 cohort the same number had gone up by more than a decade. At the same time the age of retirement only rose by two years, implying an increase in length of retirement by roughly eight years (Lee, 2001).[1] What were the main driving forces behind the impressive increase in longevity? What drove the changes in the age of retirement? Can the observed increase in years in retirement be expected to continue in the years to come? In an era where the global population is aging rapidly, these are all relevant and important issues to resolve; not least because of the fiscal sustainability problems that are created by an aging global population. In this paper we attempt to offer some progress in this regard.

In the present study we develop a life-cycle model where the representative individual is subject to physiological aging. In modeling the aging process as increasing frailty we draw on recent research in the fields of biology and medicine. In our

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* Corresponding author.
E-mail addresses: carl.johan.dalgaard@econ.ku.dk (C.-J. Dalgaard), holger.strulik@wiwi.uni-goettingen.de (H. Strulik).

[1] It is well known that the labor force participation rate for older individuals has declined monotonically from the 19th century (Costa, 1998; Lee, 2001). From this evidence it is tempting to conclude that the *age of retirement* also must have been monotonically declining. This is not so, as discussed below in the context of our cohort analysis.

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life cycle model aging has three substantive implications: it gradually lowers wage earnings over the life cycle; it works to increase the disutility from work, and it eventually leads to death. Within this framework the individual consumes, saves, and makes deliberate investments in slowing down the aging process thus postponing death. In addition, the individual decides when to optimally retire. In the end, the model allows us to study the impact from income and health technology (broadly defined) on changes in longevity, age of retirement, and thus years in retirement.[2] Formally, the model below extends the framework developed in Dalgaard and Strulik (2014) so as to allow for optimal retirement, disutility from work, and a wage rate that changes over the life cycle due to changes in health.[3]

We proceed to calibrate the model so that it reproduces observed aging, death and health expenditures for the US cohort that was born in 1940. Subsequently, we use the calibrated model to analyze the evolution of longevity and age-of-retirement across cohorts born from 1850 to 1940. We focus on these cohorts as they have all (largely) retired, which means that our model's predictions can be compared to observed rather than estimated age of retirement.

In so doing we establish three main results. First, the increase in life expectancy at age 20 is mainly driven by income, propelling investments in health; 2/3 of the increase in longevity can be accounted for by wage growth. Second, technological progress in health care is responsible for the observed increase in age of retirement for individuals born between 1850 and 1940. Finally, our simulations show that as for years in retirement both income and technological change contributed over the period in question, with the income channel being somewhat stronger. As noted below, various explanations for the rise in the importance of retirement have been put forward in the literature. While income is a familiar explanation for the rise of retirement, we believe this paper is the first to suggest that technological change in health care was a contributor.

Analytically, wage income increases longevity for a simple reason. If an increase in income is solely spent on increasing consumption at the "intensive margin" (i.e., more per period consumption) the utility gains will diminish rapidly. As a result, it is a superior strategy to expand consumption along an "extensive margin" (i.e., by an increasing length of life), which can be attained by making investments that slow down aging. Consequently, rising wages increase longevity.[4] At the same time, we show that wages hold an ambiguous effect on age of retirement. As a result, wage growth is unlikely to have caused the observed path of age of retirement. But it does contribute significantly to an increase in years in retirement, though primarily via longevity.

In contrast, technological change in health care works to increase the age of retirement. The intuition is the following. When technology in health improves, individuals age more slowly; both because of a direct impact from the innovation and because of a behavioral response in the direction of more health investments. As a result, the disutility from work declines, inducing individuals to stay on longer in the labor market. In addition, a lower per period consumption level, prompted by greater health investments, elevates the utility gain from working. Hence, technology promotes both longevity and extends working life. While wage growth accounts for a big part of the observed increase in adult life expectancy, the increase in age of retirement is caused by technology. Since technological change both raises longevity and age of retirement, the net impact on years in retirement is theoretically ambiguous. With the aid of the calibrated model however we find that technological change, like wage growth, has worked to increase years in retirement, on net.[5]

We explore a range of extensions of our baseline model. In particular, we study the consequences of allowing the disutility of labor to depend on health, the influence from (exogenous) increases in education, and we discuss the implications of task-specific structural change. In the latter context we compare the health outcomes between blue collar and white collar occupations; the former is assumed to be more strenuous and less economically rewarding than the latter. The physical aspect of the task is captured by assuming a greater amount of health reducing environmental stress on blue collar workers. Unsurprisingly, we find that white collar workers live longer and experience more years in retirement. What is interesting, is that the model also captures that health differences between the two groups, founded during working life, continue to *diverge* after retirement, consistent with the evidence (Case and Deaton, 2005). Overall, the extensions show that human capital accumulation and structural change (in part by raising average wages) may have played a role in accounting for the rise of "the golden age".

The present paper is related to several strands of literature. Our work is related to the literature which models optimal health investments and longevity (e.g., Grossman, 1972; Ehrlich and Chuma, 1990). Kuhn et al. (2015) are particularly related as the authors develop a life cycle model where retirement is optimally determined, in the presence of life prolonging health investments. The authors explain how annuity markets (motivated by uncertain length of life) might lead to overinvestment in longevity, and discuss policy options to restore the first best. Wolfe (1985) and Galama et al. (2013) discuss the implications of retirement in the context of a Grossman (1972) model. In Wolfe (1985), however, retirement is

[2] Technically, technology improvement in our model means that any dollar amount of investment in health is more effective in slowing down the aging process. As a result, "technology" could be anything from improvements in health institutions to scientific discoveries leading to a better mode of conduct at the individual level (washing hands more often in response to the discovery of the germ theory, for instance) or breakthroughs that are more of the nature of "Big Medicine" (e.g., blood pressure controlling medicine).

[3] Empirically, wages appear to be stimulated by improvements in health. See Jaeckle and Himmler (2010) and Hokayem and Ziliak (2014).

[4] An implication is that rising income inequality will be associated with rising inequality in health and longevity, a prediction which is broadly confirmed in empirical studies (e.g. Pappas et al., 1993; Pijoan-Mas and Rios-Rull, 2014; Chetty et al., 2016).

[5] We also examine the impact of changes in the relative price of health investments. From an empirical standpoint, however, this relative price has – if anything – been on the rise during the period in question, which the model suggests works to lower life expectancy and age of retirement. As a result, the observed rise in age of retirement seems to derive from technological change. Moreover, our analysis suggests that the impact from prices on years in retirement appears to be modest.

not determined by way of utility maximization, and in Galama et al. (2013) longevity is not affected by health investment. None of the mentioned studies analyzes the historical origins of rising years in retirement and longevity.[6]

In the analysis below we contrast the impact of changes in income with those pertaining to technology vis-a-vis changes in longevity. This resonates with the debate on whether the observed increase in life expectancy (at birth) was due to income and nutrition (McKeown, 1976; Fogel, 1994) or technological knowledge (Preston, 1975, 1999). Our focus is different in that we study longevity at age 20. But in the context of "adult longevity" for cohorts born 1850–1940, we find that the income channel (here mediated by "health investments" rather than nutrition *per se*) seems to have been the relatively more powerful engine for life extension.

Also related are studies that examine "the rise of retirement" (e.g., Sala-i-Martin, 1996; Gruber and Wise, 1998; Kalemli-Ozcan and Weil, 2011; Bloom et al., 2007, 2014). Among these studies Bloom et al. (2014) is particularly related. Bloom et al. (2014) develop a life cycle model and use it to gauge the impact of changes in income and life expectancy on age of retirement using cohort data for the US. The authors find income to have been the most important driver of age of retirement. We obtain a different result since aging is endogenous in our model. This difference in modeling strategy allows us to capture technological change in health care. Moreover, our model adds another channel through which income matters to retirement: that greater income admits health improvements, which reduces the individual's disutility from work and promotes a longer work-life. In the calibration this additional channel serves to "mute" the total effect of income on retirement compared to the Bloom et al. (2014) setting.[7]

Finally, this research is related to a recent literature which tries to explain the long-run evolution of macroeconomic aggregates in the US, such has health expenditures (Hall and Jones, 2007), leisure (Ramey and Francis, 2009) or schooling (Restuccia and Vandenbroucke, 2013). To this list we add life expectancy (at age 20) along with years in retirement.[8]

The paper proceeds as follows. The next section describes the model, which is calibrated in Section 3. Section 4 discusses comparative statics, whereas Section 5 provides our cohort analysis of longevity and retirement. Section 6 discusses extensions of the model and a final section is reserved for concluding remarks.

2. The model

2.1. Physiological basics: deficit accumulation

Our theory is built upon a physiologically founded notion of human aging: Aging is defined as the intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death (Arking, 2006). In gerontology the fact of aging is explained by applying reliability theory to the human body (Gavrilov and Gavrilova, 1991). That is, aging is understood as declining redundancy within the body in its totality. As redundancy recedes, expiry ("system failure") becomes increasingly likely.[9] From an empirical perspective this process has been captured by the so-called frailty index, which is developed by Mitnitski and Rockwood and various coauthors in a series of articles (e.g., Mitnitski et al., 2002a, 2002b, 2005; Rockwood and Mitnitski, 2006). This description is what we draw on in the following.

Specifically, we follow Dalgaard and Strulik (2014) and implement a parsimonious description of the process of health deficit accumulation (or increasing frailty), which extends Mitnitski et al. (2002a, 2002b) by allowing health expenditure and technology to have an impact on aging. In concrete terms, health deficits D are evolving with age t according to

$$\dot{D}(t) = \mu\left(D(t) - a - Ah(t)^{\gamma}\right), \quad D(t) \leq \bar{D}, \tag{1}$$

where initial deficits $D(0)$ are given. The parameter a captures environmental influences on aging beyond the control of the individual, the parameters $A > 0$ and $0 < \gamma < 1$ reflect the state of the health technology, and h is health investment. While A refers to the general power of health expenditure in maintenance and repair of the human body, the parameter γ specifies the degree of decreasing returns of health expenditure; the larger γ, the larger the relative productivity of cost-intensive high-technology medicine in maintaining and repairing highly deteriorated human bodies. It is worth pointing out that the interpretation of A is necessarily a broad one; the parameter captures all factors that ensure that a dollar of

[6] In a general equilibrium setting the evolution of life expectancy and its interplay with growth has been studied by e.g. Cervellati and Sunde (2005), Hazan and Zoabi (2006) and Galor and Moav (2007). See also Heijdra and Romp (2009) who analyze the impact from pension reform in a general equilibrium setting, in the presence of a realistic (but exogenously given) mortality process. Our model is also related to earlier studies that considered the impact of health expenditure on the probability to survive in a two-period overlapping generations framework (e.g. Chakraborty, 2004). In the Chakraborty model retirement is in a sense also endogenous, but since it coincides by construction with time of death it is less suitable to address the questions raised in our paper.

[7] Kalemli-Ozcan and Weil (2011) point out that the 20th century has witnessed a decline in uncertainty about life expectancy which may have helped propel the desire for early retirement; their numerical experiments suggest, however, that income should be a quantitatively more important determinant. Sala-i-Martin (1996) argues technological change has served to dilute the human capital of older workers making publically funded early retirement programs desirable; Gruber and Wise (1998) also highlight public retirement programs along with the tax disincentives for remaining in the labor market. Hence, pointing to an important impact from *health* technology on years in retirement appears to be a novel notion in this literature.

[8] See also the interesting debate on the link between life expectancy and schooling within the US (Hazan, 2009; Cervellati and Sunde, 2013; Hansen and Lønstrup, 2012; Strulik and Werner, 2016).

[9] As young adults the functional capacity of our organs is estimated to be tenfold higher than needed for mere survival (Fries, 1980); this functional redundancy declines as we age.

health investment is more effective in slowing down aging. Accordingly, the list would include technological knowledge, the effectiveness of health care institutions etc. The parameter μ, the "natural" rate of aging, is estimated with great precision by Mitnitski et al. (2002a, 2002b). In developed countries the average μ is around 0.04; with each birthday the average citizen obtains four percent more health deficits. Finally, death occurs when health deficits reach an upper boundary $\bar{D}$. Direct evidence on the existence of an upper boundary for D is found in Rockwood and Mitnitski (2006).[10]

2.2. Labor supply and wages

In order to capture the labor market consequences of aging we assume that human productivity deteriorates as a result of mounting bodily deficits. Specifically, suppose the age-earnings curve is given by the following health-adjusted Mincer equation

$$w(t) = w_0 \exp\left(\phi_1 t - \phi_2 t^2\right)\left(\frac{D_0}{D(t)}\right)^{\nu}. \tag{2}$$

The impact of schooling is included in the constant w_0 since our individuals are assumed to have finished schooling when our analysis begins (their initial age is 20 years in our benchmark scenario). The ϕ's capture the impact of experience on wages and the augmented last term captures the direct influence of poor health. Accordingly, we assume no influence from deficits on wages at "peak health", which is assumed to be health at age 20 (i.e., D_0). We thus assume that part of the reason why wages decline at the end of work life is deteriorating health. For reasonable values of ν and ϕ the wage profile is hump shaped over the life cycle such that, once retired, individuals do not find it worthwhile to re-enter the workforce.[11]

Our individual under investigation is assumed to own one unit of indivisible labor. At each age he decides whether to supply this labor endowment or not. We assume that the initial wage is high enough at the beginning of the working life such that he indeed supplies labor. Consequently there is a decision about optimal retirement at age R, which has to be taken together with the decision about optimal life-length T. We assume that the model's parameters support $R < T$ so that the individual retires before he dies.

2.3. The optimization problem

The individual maximizes utility from consumption $c(t)$ over life, taking disutility from work into account. We consider a representative member of a cohort, for which reason the maximization problem can be viewed as deterministic to a first approximation. By considering a deterministic framework we are following the mainstream economic literature on health, e.g. Grossman (1972), Ehrlich and Chuma (1990), Hall and Jones (2007).[12]

The initial age is for convenience normalized to zero. This is the age at entry into the workforce and will later be assumed to be at age 20 in the calibration. Life before 20 is thus summarized in the initial conditions. Furthermore, to avoid notational clutter, we suppress a time index denoting the birth year of the cohort. Summarizing, intertemporal utility is given by

$$V = \int_0^T \mathrm{e}^{-\rho t}\left\{u(c) - \beta \cdot \ell\right\}\ \mathrm{d}t. \tag{3}$$

Instantaneous utility from consumption $u(c) = (c^{1-\sigma} - 1)/(1-\sigma)$ for $\sigma \neq 1$ and $u(c) = \log(c)$ for $\sigma = 1$. The parameter ρ is the rate of time preference and $\ell \in \{0, 1\}$ is a toggle-variable that assumes the value of unity if the individual is working. The parameter β measures the disutility from work. For the benchmark version we assume that declining health is not a motive for retirement. We investigate a health motive for retirement in Appendix D.

Besides labor income the individual receives a capital income from wealth k, which bears interest at rate r. We do not restrict k to be non-negative, so there may be periods in life, where it is optimal to go into debt and k is therefore negative. At death we assume (without loss of generality) that $k(T) = \bar{k} \geq 0$. Thus the individual is assumed to inherit wealth $k(0) = k_0$, and to leave a bequest $k(T) = \bar{k}$ (which both could be zero).

[10] Dalgaard and Strulik (2015) provide an extensive comparison between the standard economic approach to aging, according to which humans are thought to accumulate "health capital" (Grossman, 1972) with our physiological approach, according to which humans accumulate "health deficits". Empirical advantages of our approach include: it avoids the counterfactual predictions of the Grossman (1972) model that medical expenses are positively correlated with health; that the importance of early-life (in utero) health deficits declines with increasing age; and that there exists a stable steady state of constant health (see e.g., Case and Deaton, 2005; Almond and Currie, 2011; and Dalgaard and Strulik, 2015 for a critique).

[11] See, for example, Shephard (1999) for a description from the medical perspective how aging affects work capacity through deteriorating bodily function. Most of our cognitive skills and motor skills start deteriorating around the age of 30 or even earlier (Schaie, 1994; Nair, 2005). Physiologically the phenomenon is explained by an increasing loss of redundancy in the human body and therewith deteriorating reliability (Gavrilov and Gavrilova, 1991). Costa (2000, 2002) provides evidence that the speed of bodily deterioration slowed down since the beginning of the 20th century. The aging-driven decline of bodily function maps into an (occupation-specific) decline of productivity, which maps into a decline of earnings (see Skirbekk, 2004). Case and Deaton (2005) discuss the decline of health during work and retirement in the context of Grossman's (1972) health capital model.

[12] In Strulik (2015) it is shown that the physiological approach to aging can be extended towards uncertain life time with insignificant impact on results albeit with substantial erosion of analytical tractability.

Income can be spent on consumption goods c or on health goods h. The relative price of health goods is p. While consumption goods are directly utility enhancing, health goods are instrumental in repairing or delaying bodily decay and, ultimately, in prolonging the life-span during which consumption goods can be enjoyed. In contrast to our earlier study (Dalgaard and Strulik, 2014), health expenditure also affects the retirement decision. The individual takes all prices and w_0 as given but takes the impact of health and age on wages into account. The law of motion for individual wealth is given by

$$\dot{k} = \ell[w + rk - c - ph] + (1 - \ell)[rk - c - ph]. \tag{4}$$

The problem is to maximize (3) subject to the accumulation equations (1) and (4), the wage schedule (2), the initial conditions $D(0) = D_0$, $k(0) = k_0$, and the terminal conditions $k(T) = \bar{k}$, $D(T) = \bar{D}$. The problem can be solved by employing optimal control theory; the state variables are $k(t)$ and $D(t)$ and the control variables are consumption $c(t)$, health investments $h(t)$, and the work decision (ℓ).

2.4. Optimal aging, retirement, and death

From the first order conditions for consumption, health expenditure and age of retirement R we obtain (see Appendix A for details):

$$g_c \equiv \frac{\dot{c}}{c} = \frac{r - \rho}{\sigma} \tag{5}$$

$$g_h \equiv \frac{\dot{h}}{h} = \begin{cases} \frac{1}{1-\gamma}\left(r - \mu + \nu\mu\gamma h^{\gamma-1}\frac{wA}{pD}\right) & \text{for } \ell = 1 \\ \frac{r-\mu}{1-\gamma} & \text{for } \ell = 0 \end{cases} \tag{6}$$

$$\beta = w(R)c(R)^{-\sigma}. \tag{7}$$

Equation (5) is the standard consumption Euler equation. When the individual is retired (for $\ell = 0$) we obtain the "Health Euler" equation (6) as established in Dalgaard and Strulik (2014). The Health Euler implies that a higher real rate of interest will induce individuals to allow health expenditure to rise over the life cycle; with a higher marginal rate of transformation it becomes attractive to postpone consumption by prolonging life (i.e., more consumption along the "extensive margin", in this case). This growth is tempered however by the natural rate of aging, μ. The intuition is that if μ is high, health deficits accumulate fast at the end of life, which make late-in-life health investments relatively ineffective in prolonging life; instead the optimal strategy is to invest early in life (i.e., "prevention"). The optimal path for health expenditures is also influenced by the degree of diminishing returns in investments γ: intuitively, the greater the degree of diminishing returns the more attractive it will be to smooth health investments.

When the individual is working (for $\ell = 1$) the Health Euler is augmented by a third term capturing the impact of health deficits on wages. Compared to retirement, the growth of health expenditure is somewhat steeper during the working ages because of the additional incentive to invest in health in order to mitigate the impact of deteriorating health on wages.

The condition (7) says that at the age of retirement, R, the disutility from supplying labor must equal the marginal utility gain from labor supply, captured by the marginal utility from consuming $(c(R)^{-\sigma})$ times the additional wage income $(w(R))$. At time R the individual is indifferent between work or leisure, at which point he retires.

Now, consider the consequences of a higher wage level; this corresponds to an experiment in which the wage *profile* of the individual is shifted up, implying greater life-time wages.[13] There are two individual channels that influence the individual retirement choice: an opportunity cost channel and a channel which operates through the level of consumption. The first channel is that as the wage profile shifts up the opportunity cost of leisure rises, enticing the individual to stay on a little longer in the labor market. Hence, the opportunity cost channel will work to delay retirement.

The second channel, however, renders the overall link between income and retirement ambiguous. Higher wages work to increase the level of consumption, which implies lower marginal utility from additional work effort and concordant wage income. Consequently, the "enjoyment" of the incremental wage addition from staying in the labor market a little longer, is reduced. This channel then provides individuals with an incentive to retire a little earlier, thereby increasing their overall utility by increasing leisure rather than consumption.

Given these fundamental trade-offs in human behavior it is *a priori* unclear how the age of retirement (and thus years in retirement) will change if income rises. Accordingly, in order to answer this question we need to calibrate the model. This is done below, where the parameters are chosen such that the model matches behavior of the average US male citizen (born in 1940) in several dimensions such as in terms of the evolution of frailty, health investments over life, and more. With the calibrated model in hand it is possible to conduct experiments, whereby parameters are changed after which the consequences for aging, longevity and retirement can be assessed. We return to this below.

Next consider the impact of health and longevity, holding w_0 and the age-earnings profile constant. Suppose life expectancy improves, for example, through advances in health technology A and that $r = \rho$ such that the age-consumption profile is flat. Since life-time consumption is now spread over more years, instantaneous consumption at any time declines

[13] Formally, w_0 in equation (1) is shifted up: wages are thus higher at all points in time during life.

such that marginal utility from consumption $c(R)^{-\sigma}$ goes up. In order to re-balance (7), individuals prefer a higher wage at retirement $w(R)$. Since the optimum age of retirement is on the declining branch of the bell-shaped age-earnings curve, this implies that individuals prefer an earlier age of retirement. In other words, consumption-leisure substitution effects at the margin induce individuals to retire earlier when life expectancy improves for *exogenous* reasons.

At the time of death the boundary conditions $k(T) = \bar{k}$ and $D(T) = \bar{D}$ have to be fulfilled and the Hamiltonian associated with Problem (1)–(4) has to assume the value of zero (indicating that it is constrained-optimal to die at this age). We solve problem (1)–(2) and (4)–(7) by applying the relaxation algorithm of Trimborn et al. (2008). This provides the unique life time trajectory leading from initial health deficit D_0 to terminal health deficits $\bar{D}$, and the optimal ages of retirement and death.

3. Calibration

We calibrate the model for the average male (white) US citizen who retired around the year 2000; our benchmark American thus originates from the cohort born in 1940. For this cohort life expectancy at age 20, that is at the assumed age at entry into the labor force, was 54.3 (death at age 74.3) and the age at retirement was 62.7.

We begin by calibrating the evolution of health deficits. Since the estimate of the rate of aging μ is unavailable for the US we take from Mitnitski et al. (2002a) the estimate of $\mu = 0.043$ for Canadian men. The rate of aging within the USA and Canada appears to be similar enough to justify this as a good approximation (Rockwood and Mitnitski, 2007).

Following Barro et al. (1995), we set the real interest rate $r = 0.06$ in order to fit the long-term average of real rates of return on the stock market (see also Mehra and Prescott, 1985). In Dalgaard and Strulik (2014) we showed, using data from Keehan et al. (2004), that the growth rate of health expenditure by age is about 2.0 percent for the U.S. (with similar slopes of the health expenditure trajectory for Canada, Australia, and New Zealand). Fitting $g_h = 0.02$ provides the estimate $\gamma = 0.19$ (a value close to the 0.2 estimate of Hall and Jones, 2007).

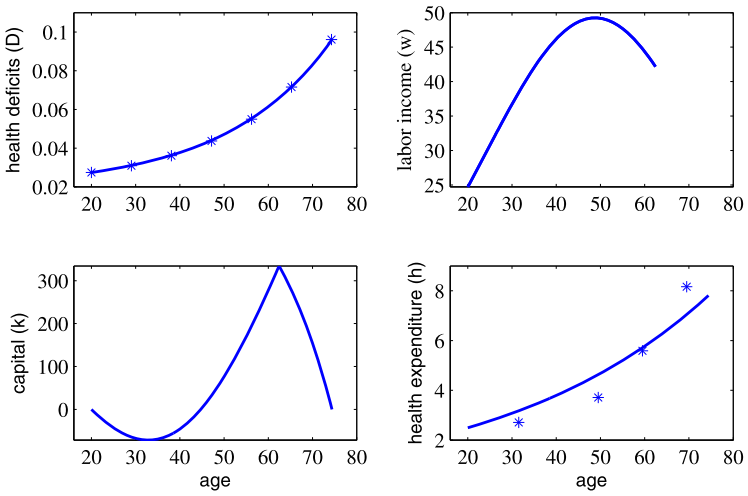
Initial and final health deficits are backed out from Mitnitski et al.'s (2002a) regression analysis. Mitnitski et al. (2002a) show that the quasi-exponential equation $D(t) = E + Be^{\mu t}$ explains around 95% of the variation in the data on the proportion of health deficits by age for Canadians, aged 15 to 79.[14] Using Mitnitski et al.'s regression results $E = 0.02$, $B = 0.0031$, and $\mu = 0.043$, we infer $D_0 = 0.0274$ at age 20, the initial year of our reference American, and $\bar{D} = 0.096$ 54.3 years later at the expected death of the reference American. Before 1840 there was no visible trend for adult life expectancy in the US and we take the average life-expectancy at 20 of cohorts born between 1780 and 1840, which was about 42 years (expected age of death 62; Pole, 1992), as life expectancy without the benefits from medical technological progress. This assumption produces the estimate $a = 0.013$ so that the model predicts a life-expectancy at 20 of 42 years without health investment. The 1850 cohort seems to be the first cohort from which on life expectancy begins to increase permanently (see Section 5).

For the benchmark run we employ data on annual labor income by cohort (see Appendix B for details). From this series we take the wage in 1960; that is, the year when our benchmark citizen (born 1940) is assumed to enter the labor force, and set it equal to w_0. We try to relate the calibration of ν to the empirical literature on the impact of health on wages. This literature, however, has not yet embraced the measure of health deficits (the frailty index) and is instead largely based on self-assessed health (ranging from poor to excellent). Naturally, this prevents a tight calibration of ν and we therefore explore the robustness of our results to alternative assumptions about ν. A study that accounts for unobserved heterogeneity, sample selection, and endogeneity is provided by Jaeckle and Himmler (2010) who estimate that, contingent on the method of estimation, healthy men earn between 1.3 percent and 7.8 percent more than those in poor health. For our benchmark case we calibrate an intermediate value by setting $\nu = 0.1$, which means that an individual who has two percent fewer health deficits enjoys on average 3 percent higher wages over the lifetime (the wage differential is 5 percent at age 20 and then slightly declining with age). In our framework we associate a change in health from "good" health to "poor" health with an increase in health deficits by 2 percentage points. This increase almost doubles health deficits at age 20 and moves health deficits at the age of retirement from 5.2 to 7.2 percent, which is the health of average 75 years old men. The implied (constant) health deficit elasticity of wages from this shift is 10 percent. It is worth noting that a more recent study by Hokayem and Ziliak (2014) suggests a health elasticity of wages of only 5 percent. In Appendix D we therefore explore the robustness of our results to alternative values of ν between 0.03 and 0.3, covering the whole range of empirical estimates on the impact from deficits on wages.

We calibrate ϕ_1 and ϕ_2 to match the return to experience estimated by Lagakos et al. (2012, Fig. 2); the return is measured relative to that of a person with 20 years of experience. In Lagakos et al. (2012, Fig. 2) we see that the return to experience originates from about 0.5, reaches 1 at 20 years of experience continues to rise for bit, assumes a maximum and then declines. It reaches 1 again at 36 years of experience and then declines further until it is about 0.5 again at 52 years of experience. Matching this curve leads to the estimates $\phi_1 = 0.0049$ and $\phi_2 = 0.00081$. The ϕ_1 value coincides with the point estimate of Lagakos et al. (2012, Table 1). The ϕ_2 value is, of course, smaller than their point estimate of 0.001 since we ascribe part of the falling productivity to declining health. Fig. C.1 in the Appendix shows the matched return to experience.

We put $\rho = r$ which renders consumption to be constant across the life cycle, as found by Browning and Ejrnæs (2009) once family size of households has been taken into account. We normalize the relative price of health to unity and adjust

[14] Rockwood and Mitnitski (2007) show that elderly community-dwelling people in Australia, Sweden, and the U.S. accumulate deficits in an exponential way very similar to Canadians (an estimate of precisely the quasi-exponential form of deficit accumulation is unfortunately unavailable for these countries).



Solid lines: model prediction. Stars: data. Labor income is annual labor income in 1000 $. Capital is wealth in 1000 $. Health expenditure is annual health expenditure in 1000 $.

Fig. 1. Age profiles: cohort born 1940.

the remaining parameters of the utility function and the technology parameter A such that (i) death occurs at the moment when $\bar{D}$ health deficits have been accumulated at the age of 74.3; (ii) such that the individual retires at the age of 62.7; and (iii) such that health expenditure approximates average expenditure of American adults at the point of retirement (Keehan et al., 2004). This produces the estimates $A = 0.0015$, $\sigma = 1.04$, and $\beta = 0.853$. The calibrated intertemporal elasticity of substitution for consumption is close to unity as suggested by several recent studies (see e.g. Chetty, 2006). In order to focus on health expenditure and retirement as a motive for savings we assume $k(0) = k(T) = 0$. This rules out intentional bequests, which seem to play an unimportant rule for the savings decision of average Americans (see De Nardi et al., 2010).[15]

Fig. 1 shows the resulting life cycle trajectories. Stars in the upper left panel indicate data according to Mitnitski et al.'s estimate of the age-specific frailty index. We have calibrated the upper and lower end of the frailty trajectory; the trajectory in between, however, is a prediction of the model. It fits the data well. The upper right panel shows the imposed wage trajectory.

The lower right panel shows the resulting trajectory of health expenditure. Stars indicate the actual health expenditure by age-group from Keehan et al. (2004), which can be compared to the solid line representing the prediction from the calibrated model. Admittedly, our calibration predicts a bit too much health expenditure in young ages and fails to match the actual expenditure for the oldest age group. But the model does well on average.

The lower left panel shows how wealth evolves with age. Since individuals wish to consume in retirement and to have health expenditures grow as they age, they save from about age 35 until retirement. The hump shaped wealth trajectory reaches its maximum at the age of retirement after which the individual dissaves until death.

As a further check of our calibration we compute the value of life as a monetary expression of aggregate utility experienced during life until its end, that is period utility is converted by the unit value of an "until", $VOL = \int_0^T \mathrm{e}^{-\rho\tau} u[c(\tau), D(\tau), R(\tau)]\mathrm{d}\tau / u_c[c(0), D(0), R(0)]$. The benchmark calibration predicts a VOL at age 20 of about $ 6.0 million, which is somewhat below Murphy and Topel's (2006, Fig. 3) estimate of the VOL at age 25 of $ 7 million.

4. Comparative statics

In order to understand the mechanics behind the long-run evolution of life expectancy and retirement, which we analyze in detail in Section 5, it is useful to inspect in general the impact from changes in the three key exogenous variables: wages, prices and technology.

Table 1 reports the comparative static results from a change in each of these variables. Table 1.A reports the results from increases in the exogenous variables (A, w_0, p) by a factor 1/3, which could be attained (say) by a constant growth rate of 1.5% per year over two decades; Table 1.B reports comparative statics associated with a decline in each of the variables by 1/3. Since the system is highly non-linear the results need not be symmetrical.

To interpret the results we begin with the impact of wages. An increase in w_0 by 1/3 evidently increases longevity by about one and a half year. The reason is easy to grasp. If the individual were to spend his additional income solely on

[15] The model predicts virtually the same life trajectories for consumption and health expenditure and the same age at retirement and death when individuals receive a bequest and leave an inheritance, given that σ and β are adjusted accordingly. For example, for $k(0) = k(T) = 5w(0)$, the adjustment $\sigma = 0.99$ and $\beta = 0.83$ provides benchmark health and consumption trajectories and the benchmark's ages of retirement and death.

Table 1
Comparative statics.

A. Increase in...

	ΔT	ΔR	$(\Delta h/h)$
Income (w_0)	1.51	0.06	40
Price (p)	−1.20	−0.40	−27
Technology (A)	9.71	1.84	4.0

B. Decrease in …

	ΔT	ΔR	$(\Delta h/h)$
Income (w_0)	−1.83	−0.20	−38
Price (p)	1.96	0.40	54
Technology (A)	−6.41	−2.31	−23

Table 1.A shows the results from increasing w_0, A, p by factor 1/3. Table 1.B provides corresponding results for a similar reduction. For the columns on ΔT and ΔR the units are "years" and $(\Delta h/h)$ is measured in percent.

greater per period consumption it would involve sharply diminishing marginal utility. To avoid this situation it is attractive to expand consumption along the extensive margin instead; i.e., via a longer life. But this dictates more health investments so as to postpone the date of death. In the experiment, health spending over the life cycle therefore increases by 40 percent (cf. column 3). From Table 1.B we see the results from a reduction in wages by 1/3: life expectancy and health expenditures decline, as expected.

When wages go up by a third, the age of retirement increases by 0.06 years or about 20 days (Table 1.A, column 2). This minuscule response is to be appreciated in the light of the discussion in Section 2.4, which explains the ambiguity of the impact from wages on retirement: in our calibration the positive effect of income on the age of retirement dominates however. When income is reduced by 1/3, the individual prefers to retire about 2.4 months earlier. The reason for the quantitative difference between the two experiments is due to the fact that the impact of wages is non-linear, due to the curvature of the utility function (i.e., depends on whether ($c^{-\sigma}$ is high or low). Altogether, however, income exerts only a small effect on the preferred age of retirement. This observation will explain why we, in the next section, conclude that income growth has virtually no impact on the preferred age at retirement.

Unsurprisingly, an increase in the relative price of health spending leads to a decrease in health spending. The consequence is a shorter life. Quantitatively, longevity shrinks by about a year, as seen from the table. Meanwhile, the age of retirement also declines but less than longevity, implying a shorter stay in retirement of 0.8 years. Since individuals live a shorter life and spend less on health, they spend more on consumption at any age. In order to counter-balance the marginal utility from consumption with a higher wage at entry into retirement, individuals prefer to retire a bit earlier. This effect is amplified by faster deteriorating health and its effect on wages, which also motivates earlier retirement.

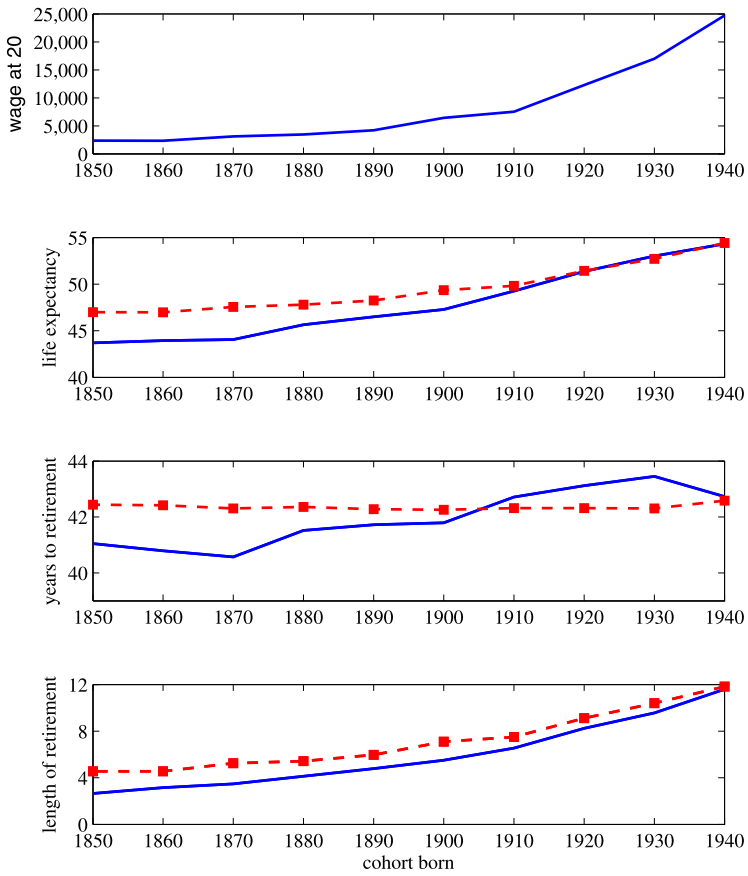
Finally consider the influence from health efficiency. Improvements in health efficiency makes it more attractive to expand utility from consumption along the extensive margin (longer life) relative to the intensive margin (more per period utility). Consequently the level of h increases by 4 percent (and c decreases), which leads to longer life. Quantitatively, the impact of improving health technology is rather substantial: longevity rises by almost 10 years. At the same time the age of retirement also goes up significantly. The reason is that a lower level of per period consumption increases its marginal utility, and thus the marginal benefits from additional wage work. This effect is reinforced by the fact that lower deficits imply higher wages at any age. In order to counter-balance the first order condition, individuals retire later, thereby reducing the wage at entry into retirement. While health efficiency improvements increase the age of retirement (by almost 2 years) they also increase years in retirement by about 8 years. From Table 1.B we note that the results are qualitatively the same for a reduction in A, though the impact on health expenditure and years in retirement is larger while the impact on longevity is smaller. The general take-away from these experiments is that increases in wages, reductions in the relative prices of health spending, or improvements in health efficiency all will work to increase years in retirement.

5. A century of rising health, wealth, and leisure

In this section we examine the model's ability to replicate the movements in longevity, age of retirement and years of retirement across cohorts born 1850–1940 in the US. These cohorts are singled out since they have all (largely) left the labor market as of today, implying that the numbers for age of retirement are based on observation rather than estimation.

We have obtained data on average real wages by cohort, life expectancy by cohort, and age of retirement by cohort for the US. In addition, we have data on the evolution of the relative price of health services from 1928 onwards. Prior to 1928 we assume, inspired by the modest change in CPI, that the relative price of health services remained constant. We use the price series to calculate average prices per life time for the cohorts under scrutiny to proxy p. All data sources and manipulations are described in the Appendix. The data on wages, life expectancy, age of retirement and years in retirement are depicted in Fig. 2 (cf. the solid blue lines).

It is probably worth pausing a moment to comment on the data, since it may seem surprising that the age of retirement (taken from Lee, 2001) is trending upward during most of the period in focus. To be sure, labor force participation rates (LFPR) among older males have declined drastically (and in a monotonic fashion) since the 19th century (Costa, 1998; Lee, 2001). Note, however, that the LFPR only captures individuals who manage to survive until 65; individuals who retired and died *before* the age of 65 do not count. As it turns out people started permanently leaving the labor force already at the age of 50 (Lee, 2001, p. 643). Now, suppose improvements in health allow more people to survive until the age of 65 after which they retire. Then the LFPR will decline, while the expected age of retirement will increase as individuals who in the past retired and died in their 50s manage to survive and retire in their 60s. There is therefore no contradiction between



Solid lines: data. Dashed lines: model prediction with imputed wages and constant medical technology and prices.

Fig. 2. A century of increasing health and leisure: basic run.

Lee's (2001) estimates that show a (slight) increase in age of retirement and the observed dramatic reductions in the LFPR of the old (Costa, 1998). That said, it is also worth observing that the age of retirement apparently starts to decline with cohorts born after 1930. This process continues during the post World War II period (Lee, 2001, Table 1). We return to this fact at the end of this section.

With the data in hand, along with our calibrated parameters, we provide the following cross-cohort analysis. First, we feed our real wage data (displayed in the top panel of Fig. 2) through the model and compare the model's predictions for longevity and age of retirement to the data. As next steps one would ideally like to look at the influence from p and A, individually. Sadly, however, A is unobservable and our data on p is somewhat incomplete. Hence, we do the following instead. We start by filtering our data series for p through the model, and subsequently calibrate the series for A such that we match the evolution of longevity by cohort *exactly*. Notice that measurement error in the series for p (in particular the unobserved part) will be picked up by the calibrated A series. The key check of the model then becomes whether it is able to account for the observed evolution of cohort specific *age of retirement*, conditional on the series for A, w and p. Notice that this procedure also allows us to asses how much of the observed increase in longevity and age of retirement we can account for by either wage changes or changes in p and A, conditional on our calibration.

In Fig. 2 we show the result from the first exercise. As seen from panel 2 from above we cannot fully motivate the evolution of cohort specific life expectancy: the calculated increase due to wage growth is somewhat smaller than what is observed in the data. The model does replicate longevity for the cohort born in 1940 but this is a consequence of the calibration, since it was designed to match life expectancy at age 20 for individuals born precisely in 1940. The sign that wage growth falls short of accounting fully for the path of longevity is thus found in the fact that the model overestimates longevity of the 1850 cohort. That said, the explanatory power of wage growth is substantial in that it can account for roughly 7 additional years in life expectancy, or roughly 70% of the total increase from the 1850 to the 1940 cohort (i.e., 10.7 years). This means that the lions share of the total increase can adequately be accounted for by wage growth over the period in question. However, wage growth is clearly incapable of accounting for the trend in age of retirement. The age of retirement stays almost constant over a century whereas in fact it increased by 1.7 years. This exercise shows that wage growth is unlikely to have been the dominant force in accounting for the evolution of age of retirement between 1850–1940.

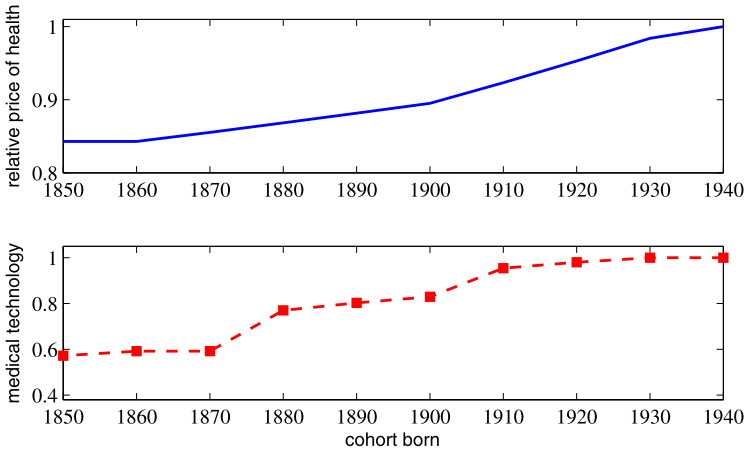


Fig. 3. Imputed prices and predicted medical technology.

Even though the age of retirement evidently rose during most of the period that we consider, years in retirement nevertheless expanded monotonically, as shown in Fig. 2. In this context, however, wage growth tends to overstate the increase slightly. This is reflected in the fact that the simulated number of years in retirement in 1850 is smaller than in the data. Absent changes in technology and prices the model suggest that a larger increase in years in retirement should have occurred.

Accordingly, we next try to gauge the (joint) influence from changes in prices and health efficiency on longevity and age of retirement. The calibrated series for A, which ensures that the model replicates the evolution of longevity, is depicted in Fig. 3 along with our input data on price movements. As noted above, the calibrated series for A is undoubtedly tainted by measurement error if the price series is inaccurate, which it may well be since we have no data prior to 1928. Still, movements in prices have a relatively modest quantitative impact on longevity compared to the impact from A, as documented in the last section. Consequently, as long as the measurement error in p is relatively small the induced bias on the path of A will probably be insignificant. It therefore seems worthwhile to try to assess whether the path of A looks plausible as a representation of the evolution of health efficiency.[16]

It is clear that the series features two major increases: from cohorts born in 1870 to cohorts born 1880–1990, and again between the turn of the century and 1910–1920. Recall that longevity is measured as life expectancy at the age of 20; hence the question is whether major innovations can be said to have occurred during the period 1900–1910 (episode 1) and during the period 1930–1940 (episode 2).[17]

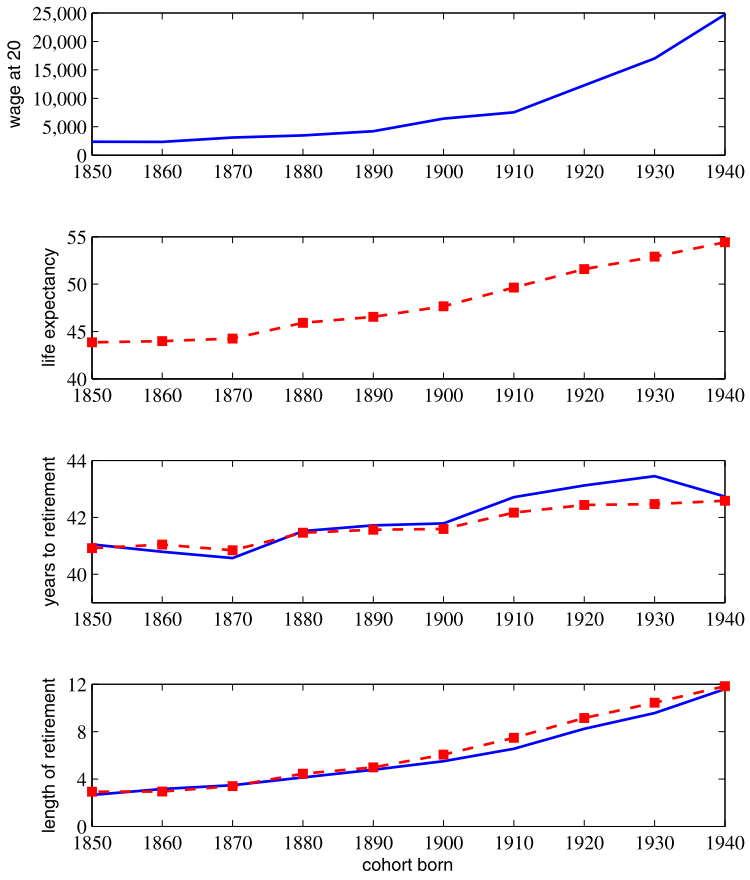
A possible explanation for the first episode could be initiatives associated with the discovery of the germ theory of disease. While this theory, by all accounts, was scientifically accepted around 1880 it is probably not until the beginning of the 20th century that its implications is starting to diffuse in society at large in the US, that is, the value of ventilated rooms, of washing hands, isolating sick individuals etc. (Preston, 1999). If indeed these ideas started to spread at this time one would expect to see an increase in longevity of cohorts that were 20 around the turn of the century. The second episode involving a rising A might be associated with the discovery of penicillin in 1928 and its subsequent application in the 1940s. This is at least broadly consistent with Fig. 3 although the initial upward shift in A seems to be a decade too early. Overall the calibrated series for A does not seem glaringly inconsistent with major health related innovations during the period in question.[18]

Thus reassured, Fig. 4 depicts the impact of changes in A, p and w_0 on our main outcome variables; the panels mimic those from Fig. 2. By construction the model matches life expectancy exactly. The key issue though is the model's fit with regards to the age of retirement, which we now approximate very well in 1850 and all of the 19th century and just a slight underestimation during the early 20th century. Hence, once we also take into account changes in the relative price of health and health efficiency, the model can match 90 years of persistent increases in health and leisure. But there is an important upshot. Since both price increases and wage growth work to lower the age of retirement we have a clear conclusion: the upward sloping path for age of retirement is due to the impact from technological change in health care.

[16] The period under scrutiny also witnessed longevity-influencing events, such at World War I. The impact from such events will inevitably be picked up by A in this calibration.

[17] The major shifts are clearly between generations 1870 and 1880, and between generations 1900 and 1910. But in both cases the increase appears to continue for another generation (to 1890 and 1920, respectively) before it "stalls".

[18] Medicaid was introduced via the Social Security Amendments of 1965, which provided federal health insurance for people above the age of 65 and for poor families and was funded by a separate earnings tax. In principle this could affect cohorts born in 1900 and later. Insofar as the collective insurance provided efficiency gains this will also be absorbed in our calibrated series for productivity, A. A rigorous analysis of the Medicaid system, which would require a general equilibrium model, is beyond the scope of the present paper.



Solid lines: data. Dashed lines: model prediction with imputed wages and prices and estimated medical technological progress.

Fig. 4. A century of increasing health and leisure: imputed prices.

Finally, the fit for years in retirement is also good. In part, of course, because we match longevity exactly. Nevertheless we now see that the model no longer underestimates years in retirement for the cohort born in 1850. This suggests that while technology (and prices) do serve to increase years in retirement (cf. Table 1), their quantitative impact is more modest than that of income, and have therefore served to stabilize the evolution of years in retirement over the period in question.

In the analysis above we have focused on cohorts born between 1850 and 1940. As noted in the Introduction, we focus on these cohorts as they have all (largely) retired from the labor market. Consequently, we can rely on observations (cohort estimates) on age of retirement rather than (cross sectional) period estimates. Nevertheless, it seems worth commenting on more recent developments. The evidence reported in Lee (2001) shows that years in retirement have continued to expand with cohorts born after 1940: it rose from 11.6 years to 16.3 years for the cohort born in 1990. The further rise of retirement is driven by increasing life expectancy but also by a declining age of retirement: whereas the expected age of retirement was 63.5 for the cohort born in 1930 (the recorded "peak"), it declined to 61.5 in 1990. Naturally, these are all projections which may turn out to be inaccurate. But if we take them at face value they suggest, seen through the lens of our model, that income and technological progress have been unable to off-set the impact from an accelerated rate of increase in the medical consumer price index (MCPI); the relative rate of increase in the MCPI went progressively up from 0.4% per year to 2.8% per year at the end of the century (Berndt et al., 2000, Fig. 1). This, at least, can account for the declining age at retirement, combined with the continued increase in years in retirement, within the context of the model developed above. At the same time other mechanisms might well have been at work, encouraging earlier retirement (e.g., Gruber and Wise, 1998; Kalemli-Ozcan and Weil, 2011).

The genesis of the golden age had a tremendous impact on the value of life. Fig. 5 shows the evolution of the value of life predicted by the computational experiment of Fig. 4. The value of life is estimated to have climbed from 0.44 million dollars for the cohort born 1850 to 6.0 million dollars for the cohort born 1940. The greatest part of the increase was experienced in the 20th century; from 1850 to 1890 the value of life "merely" doubles to 0.84 million dollars. Rising longevity can be identified as the most important driver behind the increasing value of life. The intuition behind this result has been

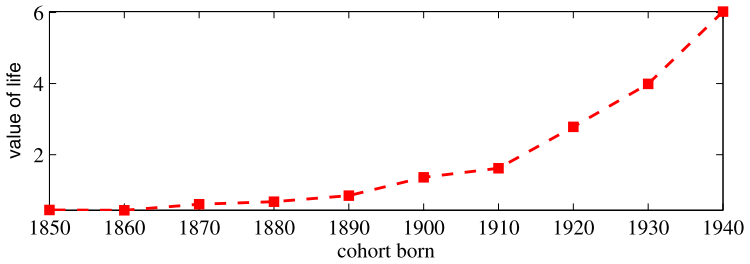


Fig. 5. Value of life.

developed above: period utility is subject to decreasing marginal returns due to strict concavity, life time utility is linearly increasing in longevity.[19]

Finally we note that the average health of workers improved substantially over the century, with particularly large improvements at older ages. The predicted frailty at age 50 is 6 percentage points for the cohort born in 1850 and 4.7 percentage points for the cohort born in 1940, an improvement by 21 percent. At age 60, the frailty index improved from 8.4 percentage points to 6.1 percentage points, an improvement by 27 percent. These predictions are consistent with Costa's (2000, 2002) observation of long-run trends of health (deficits) of older American workers. For example, the share of elderly US American men (50–64 years old) having difficulties bending declined from 44 percent in 1900 to 8 percent in 1994. The share of men having difficulties walking declined from 28 percent in 1900 to 8 percent in 1994. Costa (2000, 2002) observes similar trends for a host of other health conditions potentially affecting labor productivity.

In Appendix D we perform a sensitivity analysis with respect to the parameters we know perhaps less about. Fig. D.1 replicates Fig. 4 when $\nu = 0.03$ (instead of 0.1) and ϕ_2 is adjusted to 0.00084 in order to match the same age-earnings curve (of Fig. C.1). Thus falling wages of older workers are to a smaller degree explained by rising health deficits. For $\nu = 0.03$ the health deficits elasticity of wages is three percent as suggested by Hokayem and Ziliak (2014). In this case the model slightly underestimates the increase of the age of retirement while the overall fit of the long-run historical evolution is still good. In Fig. E.1 we show results for the opposite case when $\nu = 0.3$ (and ϕ_2 adjusted to 0.00072), which attributes much more of falling wages in old age to increasing frailty (with a health deficit elasticity of wages of 30 percent). Naturally, in this case, the model overpredicts the rise of the age of retirement by about a year and thus underpredicts the increase of years in retirement by about one year.

Finally we have also examined the consequences of allowing for a "health motive" for retirement; i.e., the situation where the disutility of work is increasing in deficits. In this case the model predicts too steep an increase the age of retirement, and consequently ends up underpredicting the evolution of years in retirement. A further discussion is found in Appendix E.

6. Extensions and applications

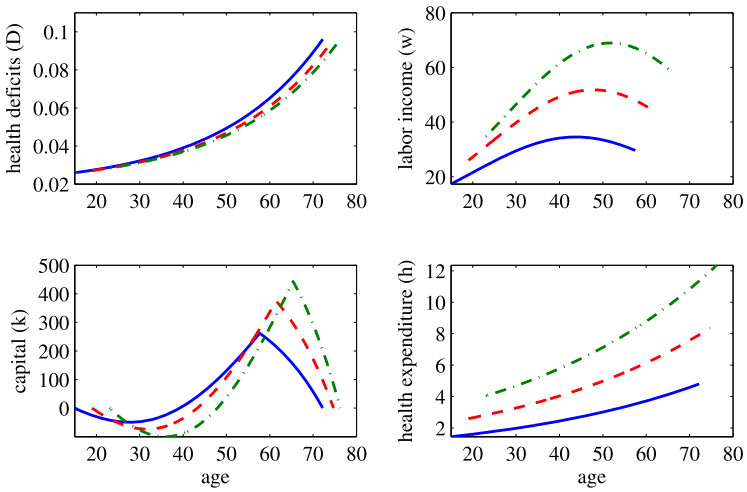
6.1. Education

We next investigate the impact of alternative age-earning profiles on the age of retirement and longevity. This exercise could also be understood as exogenously feeding into the model three different educational statuses. The case of endogenous education in the context of health deficit accumulation is discussed in detail elsewhere (Strulik, 2013). Here, we take education into account by shifting the age of entry into the labor market (the initial age of our analysis) as well as the initial wage earned at entry.

In order to compare with Card (1991, Fig. 4) we discuss the outcomes from 8, 12 and 16 years of education. Assuming individuals start attending school at age seven, this means that individuals begin their working life at age 15, 19 (close to our benchmark assumption) and 26, respectively (see Table 2). From Card we infer that the initial wages for 12 years of schooling are about 25% higher than the initial wage for 8 years of schooling and about 25% lower than the initial wage for 16 years of schooling. We insert the initial wages into the health deficit law from Mitnitski et al. (2002a) and obtain the initial health deficits; better educated individuals are marginally less healthy at entry into the labor force due to (mild) aging, see Table 2.

The implied age-earning curves are shown in the upper right panel of Fig. 6. Notice that the model predicts a steeper increase of earnings for better educated individuals. As shown in the lower right panel, better educated individuals are predicted to invest substantially more in health such that their productivity declines less due to aging. This means that their productivity rises faster at young ages because increasing experience is to a lesser degree compensated by declining health than for individuals of low education. Moreover, higher health investments allow better educated individuals to live substantially longer. Empirically, the education gradient between high school or less education and college education was about 7 years of life expectancy at age 25 in the year 2000 (Meara et al., 2008). Our model rationalizes 20–50% of

[19] The depicted increase amounts to an annual average growth rate of the value of life by roughly two percent, which includes gains from both increasing income (i.e., consumption) and life expectancy. Murphy and Topel (2006) estimate that over the 20th century the value of life increased by about 0.2 percent per year due to longevity alone.



Solid lines: 8 years education ($T = 72.1$, $R = 57.8$, length of retirement 14.3). Dashed lines: 12 years education ($T = 74.7$, $R = 61.9$, length of retirement 12.8). Dash-dotted lines: 16 years education ($T = 76.2$, $R = 65.7$, length of retirement 10.5).

Fig. 6. Life cycle for different education strata.

Table 2
Results: education strata.

Case	Years edu.	Init. age	Init. deficits	Age death	Age retire	Length retirement
1	8	15	2.6	72.1	57.8	14.3
2	12	19	2.7	74.7	61.9	12.8
3	16	23	2.8	76.2	65.7	10.5

the education gradient: the predicted life expectancy gap is 4.1 years between 8 and 16 years of education and 1.5 years between 12 and 16 years of education. This reduced-form estimate is close to the estimates in Strulik (2013) who concludes that endogenous education in the Dalgaard–Strulik model can motivate about half of the observable education gradient of life expectancy.

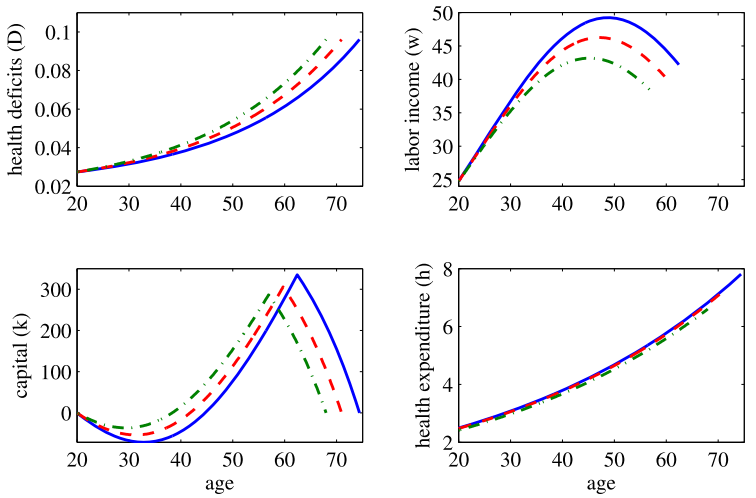
By construction, our reduced-from approach shuts off reverse causality (from health to education) and thus estimates the causal effect of better education through higher income and a steeper earnings profile on health. Empirically, it is difficult to identify the education gradient (because of reverse causality) as well as the channels through which it operates. According to one much-cited study (Cutler and Lleras-Muney, 2010), 20–30 percent of the education gradient are motivated through the income channel. Quantitatively, our results are in the same ballpark.

As shown in Table 2, the model predicts that better educated individuals tend to retire later, in line with the empirical evidence (Burtless, 2013). In total the education gradient motivates almost 8 years of later retirement. The years spent in retirement are shorter for better educated individuals. In total, the education gradient predicts a difference of about four years in retirement. Another way to look at these results is that individuals at all levels of education spend about 42.7 years working. The prospect of a longer life, given exogenous education, hardly influences the optimal length of the worklife (see the discussion in Hazan, 2009 and Strulik and Werner, 2016).

The income change through education has a level effect and a growth effect on health expenditure, i.e. it shifts the h-trajectory upwards and makes it steeper. The shift originates from decreasing marginal utility of current consumption. It occurs because richer individuals enjoy an additional unit of current consumption less than poor individuals and thus spend more on life-prolonging health investments. The age-expenditure curve gets steeper because better educated individuals are motivated to spend more on health in order to prevent the deterioration of productivity through declining health. In order to estimate the contribution of level and growth effects we run the experiment of Table 2 again and set $\nu = 0$ (no impact of health on wages). This shuts off the growth channel (see (6)). As a result, the age of retirement increases markedly to 59.9 for 8 years of education and to 67.8 for 16 years of education. The predicted age of death, however, increases only mildly to 72.4 for 8 years of education and 76.4 for 16 years of education, implying a marginal reduction of the gradient from 4.1 to 4.0 years. The growth rate effect thus seems to be of second order for the education gradient of life expectancy, which largely originates from the level effect of rising income on health investments of the better educated.

6.2. Occupation-specific aging

We next investigate (in reduced-form) the impact of job-specific characteristics on health deficit accumulation and retirement. Since it can be argued that the force of aging should be treated as a natural rate (Dalgaard and Strulik, 2014), the



Solid lines: benchmark case: ($T = 74.4$, $R = 62.7$, length of retirement 11.7). Dotted lines: $a_w = 0.9a$, $\nu = 0.2$: ($T = 70.9$, $R = 59.9$, length of retirement 11.0). Dash-dotted lines: $a = 0.8a_w$, $\nu = 0.3$: ($T = 68.0$, $R = 56.9$, length of retirement 11.1).

Fig. 7. Occupation-specific aging.

gateway for job-specific stress on the human body is the "environmental parameter" a. Notice that a higher value of a slows down the aging process. Particularly strenuous or stressful occupations are thus characterized by a low value of a. We also assume that the extra strain on the body is constant over time and is experienced only during the working ages. Equation (1) is thus modified as (8).

$$\dot{D}(t) = \mu\left(D(t) - a(t) - Ah(t)^{\gamma}\right), \quad D(t) \leq \bar{D}, \qquad a(t) = \begin{cases} a_w \geq a_n & \text{if working} \\ a_n & \text{otherwise.} \end{cases} \tag{8}$$

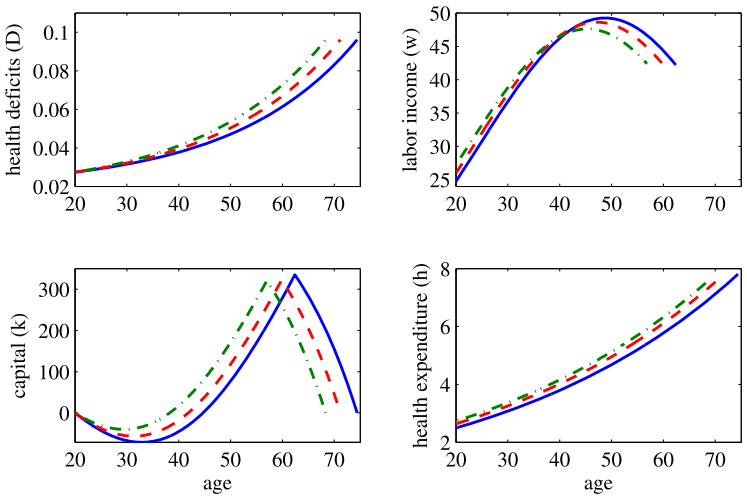
A low value of a_w would thus characterize a strenuous job of manual labor. Another aspect of manual labor is that it relies on bodily functions that deteriorate earlier in life (muscle function and motor skills vs. mental capabilities). It will be reflected by a higher value of ν.

Fig. 7 shows the life cycle trajectories for the benchmark case from Fig. 1, in which $a_w = a_n = a$ (solid lines), for an individual with a more strenuous job where $a_w = 0.9a$ and $\nu = 0.2$ (dashed lines) and an individual with a severely unhealthy job where $a_w = 0.8a$ and $\nu = 0.3$ (dash-dotted lines). All other parameter values are kept from our benchmark case. In the upper right panel we see that a strenuous job implies a slower increase of wages at young ages and an earlier peak of maximum wages and maximum productivity. Structural change, which increasingly moves individuals from physically stressful manual jobs in hazardous environments (like mining) to non-manual office work, may thus be another explanation for why, on average, the age of peak productivity increases over time (Burtless, 2013; Strulik and Werner, 2016).

Because the worker in strenuous manual labor earns less income, he also spends less on health when young, irrespective of his quickly deteriorating body (see the lower right panel of Fig. 7). As a result the worker in the most strenuous occupation retires 5.7 years earlier than the worker in the least strenuous occupation and he expires about 6.4 years earlier, implying that he altogether experiences about nine months less in retirement. Structural change from strenuous manual labor to light office work may thus serve as another explanation for the rise of the "golden years".

From the upper left panel in Fig. 7 we see that the distance of health deficits between individuals in strenuous work and those in light work increases with age, even after retirement, when environmental effects on aging are assumed to be equal across individuals. This predicted outcome is in line with the stylized facts, as shown by Case and Deaton (2005). Case and Deaton use this observation (among others) in order to discard the health capital model (Grossman, 1972). It is easy to see why. A basic assumption in the Grossman is that health capital at age t is accumulated as $\dot{H}(t) = f(h) - d(t)H(t)$, in which $f(h(t))$ is health investment and $d(t)$ is the (potentially age-dependent) rate of health capital depreciation. This means that the model predicts that at any age individuals with much health capital (i.e. healthy individuals) lose more health capital through depreciation than unhealthy ones. This produces the counterfactual prediction that any initial gap between healthy and unhealthy individuals gets smaller with increasing age. The health deficit model, in contrast, assumes that unhealthy individuals, i.e. those who have already many health deficits, age faster, i.e. develop further health deficits more rapidly (see equation (1) or (8)). This means that the model predicts, in line with the observations, that any initial gap between healthy and unhealthy individuals gets larger over time.

The study by Case and Deaton (2005) also discusses that individuals may be motivated to accept a strenuous job because this type of work is more highly rewarded at young ages. We capture this case (in reduced-form) by assuming that initial labor income is a positive function of environmental stress, i.e. a negative function of a_w:



Solid lines: benchmark case: ($T = 74.4$, $R = 62.7$, length of retirement 11.7). Dotted lines: 10% reduction of a: ($T = 71.2$, $R = 59.9$, length of retirement 11.3). Dash-dotted lines: 10% reduction of a: ($T = 68.3$, $R = 57.0$, length of retirement 11.3).

Fig. 8. Occupation-specific aging and financially rewarding strenuous jobs.

$$w(t) = \theta(a_w)\exp\left(\phi_1 t - \phi_2 t^2\right)\left(\frac{D_0}{D(t)}\right)^{\phi}, \qquad \theta'(a_w) < 0. \tag{9}$$

In Fig. 8 we show results for this case. Solid lines reiterate the life cycle trajectories for our benchmark American. Dashed lines show life cycle trajectories predicted for a worker with $a_w = 0.9a, \theta(a_w) = 1.05\theta$ and dash-dotted lines represent the case $a_w = 0.9a, \theta(a_w) = 1.1\theta$. In contrast to the previous numerical experiment, workers in strenuous jobs are now financially better off. The discounted life time income for the most strenuous job (dash-dotted lines) exceeds the life time income of the least strenuous job (solid lines) by 12 percent. Consequently, individuals in strenuous occupations now spend more on their health at any give age (see bottom right panel of Fig. 8). Notwithstanding their greater health expenditure the health of workers in strenuous jobs declines faster, before and after retirement (see the upper left panel of Fig. 8). The main result of the widening health gradient is robust against the extension of financial rewards in strenuous jobs.[20]

7. Conclusion

In the present paper we have developed a life cycle model with an optimal retirement choice where individuals are subject to physiological aging. We have used a calibrated version of the model to study the origins of the remarkable rise in adult life expectancy as well as the increase in the age of retirement, for cohorts born 1850–1940.

We find that the bulk of the increase in adult longevity appears to have been generated via an income-cum-health investment channel; about 2/3 of the total increase seems to be due to income growth. Technological progress also contributed, but income appears to have been more important. Conversely, technological knowledge appears to have been largely responsible for the increase in age of retirement in the US, 1850–1940; income and rising prices work to drive the age of retirement in the other direction during this period.

From a policy angle the difference between longevity and age of retirement is perhaps of greatest interest. The model predicts that increases in (relative) health care prices, technological progress in health care and rising income all contribute to more years in retirement. Since there is no particular reason to expect that either one of these factors will stall, this suggests that desired length of retirement will continue to grow in the years to come. This could be seen as bad news from the point of view of fiscal sustainability.

Since governments are unlikely to wish for technological regress and declining income, it would appear that the only option left to policy is to target relative prices of health care. Our calibration suggest that *declining* relative prices of health investments will serve to increase both longevity and the age of retirement, but that the impact on the latter appears to be larger, which would thus imply fewer desired years in retirement. Yet caution is warranted as these results might be specific to the US calibration. Moreover, the quantitative impact from even substantial reductions in prices appear to be modest.

[20] Eventually, of course, if the income gap gets wide enough, the result will be overturned. For our estimated parameters of medical technology, life expectancy of workers in the unhealthy environment begins to exceed that of the benchmark American, if their life time work income is four times larger. Such income gaps may be realized by professional rock musicians and athletes but are less likely in "ordinary" strenuous occupations like coal mining or chemical plant operation.

Appendix A. Derivation of (5)–(7)

The current-value Hamiltonian associated with problem (1)–(4) is given by:

$$H = \frac{c^{1-\sigma}-1}{1-\sigma} - \beta\ell + \lambda_k\left[w_0\exp(\phi_1 t-\phi_2 t^2)(D_0/D)^{\nu}\ell + rk - c - ph\right] + \lambda_D\mu\left[D - Ah^{\gamma} - a\right] \tag{A.1}$$

The first order conditions and co-state equations are:

$$c^{-\sigma} - \lambda_k = 0 \tag{A.2}$$

$$-\lambda_k p - \lambda_D\mu\gamma A h^{\gamma-1} = 0 \tag{A.3}$$

$$-\beta + \lambda_k w = 0 \tag{A.4}$$

$$\lambda_k r = \lambda_k\rho - \dot{\lambda}_k \quad \Rightarrow \quad \dot{\lambda}_k/k = \rho - r \tag{A.5}$$

$$\lambda_k(-\nu)w\ell/D + \lambda_D\mu = \lambda_D\rho - \dot{\lambda}_D. \tag{A.6}$$

Differentiating (A.2) wrt time and inserting (A.5) provides (5) in the text. Inserting (A.2) into (A.4) provides (7) in the text. Differentiating (A.3) with respect to time provides:

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left(\frac{\dot{\lambda_D}}{\lambda_D} - \frac{\dot{\lambda}_k}{\lambda_k}\right). \tag{A.7}$$

Inserting (A.3) into (A.6) provides:

$$\frac{\dot{\lambda}_D}{\lambda_D} = \rho - \mu + \nu\mu\gamma\frac{Ah^{\gamma-1}w\ell}{pD}. \tag{A.8}$$

Finally, inserting (A.6) and (A.8) into (A.7) provides (6) in the text.

Appendix B. Data appendix

- Life expectancy at age 20 by cohort. Source: Lee (2001, Table 1).
- Expected age of retirement at age 20 by cohort. Source: Lee (2001, Table 1).
- Average years of schooling by cohort. Source: Hazan (2009).
- Nominal wage index for unskilled workers for the US. Source: Historical statistics for the US millennium edition. Table Ba4218: Index of money wages for unskilled labor: 1774–1974.[21]
- CPI for the US. Source: Historical statistics for the US millennium edition. Table CC1-2: Consumer price indices for all items: 1774–2003.[22]
- Real wages 1850–1940 for individual cohorts: The real wage index for unskilled workers, at time t, x_t, is calculated as the nominal wage (index = 100 in 1860) divided by the CPI (index = 100 in 1860). In practise the average wage reflects educational attainment of the cohort. Hence, for cohorts born at time c, with u_c years of recorded schooling, the associated real wage index was calculated as

$$y_c = x_{c+20}\cdot e^{\theta\cdot u_c}. \tag{B.1}$$

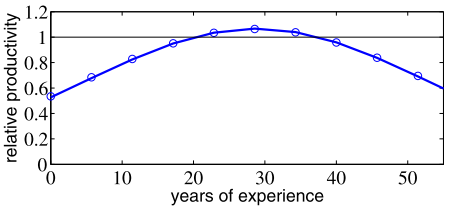
Hence, we assume cohort c enters the labor market at age 20. The parameter θ is the return to a year of schooling. We assume $\theta = 0.1$ in all years. Finally, to get the wage *level*, we did the following. First, we obtained GDP per worker, rgdpl2wok, for 1970 from Penn World Tables 7.0 (1970 is the last year for which we have data on cohort specific years of schooling from Hazan, 2009). Second, we define z_t as $2/3\cdot rgdpl2wok_{1970}$, where "2/3" proxies for the labor share. Third, for all cohorts c their entry real wage level is then calculated as

$$w_c = z_t\cdot(y_c/y_{1970}). \tag{B.2}$$

• The relative price, p. We constructed p at an annual frequency (1850–2000) in the following way: Until 1927 $p = 1$. After 1927 the p is allowed to rise with the relative rate of inflation (CPI vs MCPI) in the period intervals reported in Berndt et al (2000, Fig. 1). For instance, between 1927 and 1946 the relative rate of increase in MCPI was 0.4 pct per year, rising progressively to 2.81 pct per year in the last period 1986–1996. The relative speed of inflation from 1996 to 2016 (the terminal year of the 1940 cohort) is assumed identical to the period 1986–1996.

[21] Web source: http://hsus.cambridge.org/HSUSWeb/essay/showtableessay.do?id=Ba4218&swidth=1366.
[22] Web source: http://hsus.cambridge.org/HSUSWeb/search/searchTable.do?id=Cc1-2.

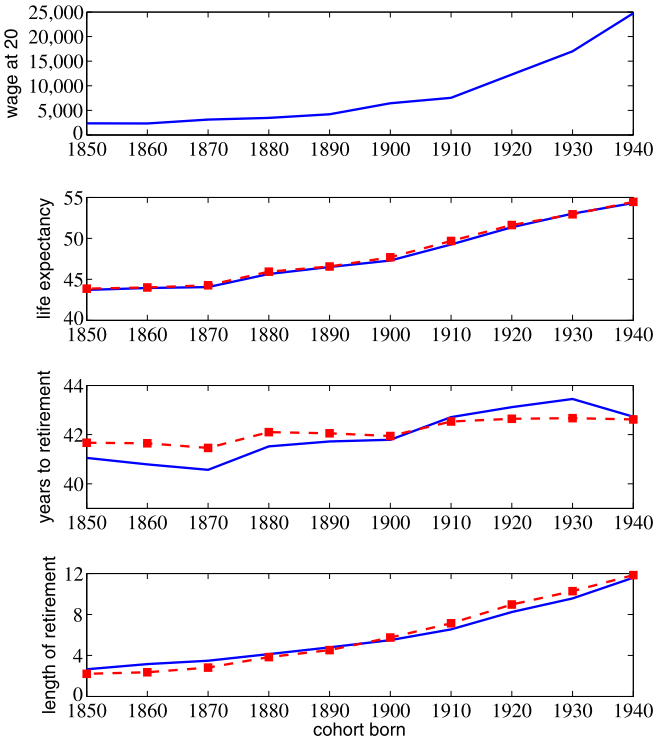
Appendix C. Age-experience curve



Solid line: continuous approximation of Lagakos et al. (2012, Figure 2). Dotted line: model prediction with $\phi_1 = 0.049$, $\phi_2 = 0.00081$, and $\nu = 0.1$. Return measured relative to return at 20 years of experience.

Fig. C.1. Estimated age-experience curve.

Appendix D. Sensitivity analysis

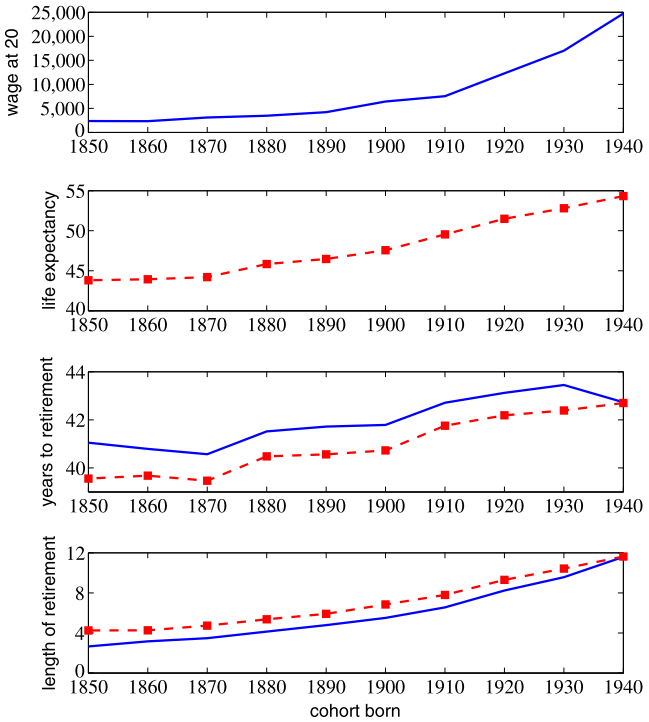


Solid lines: data. Dashed lines: model prediction with imputed wages and prices and estimated medical technological progress. Parameters as for benchmark run except $\nu = 0.03$ and $\phi = 0.00084$.

Fig. D.1. A century of increasing health and leisure: imputed prices: $\nu = 0.03$.

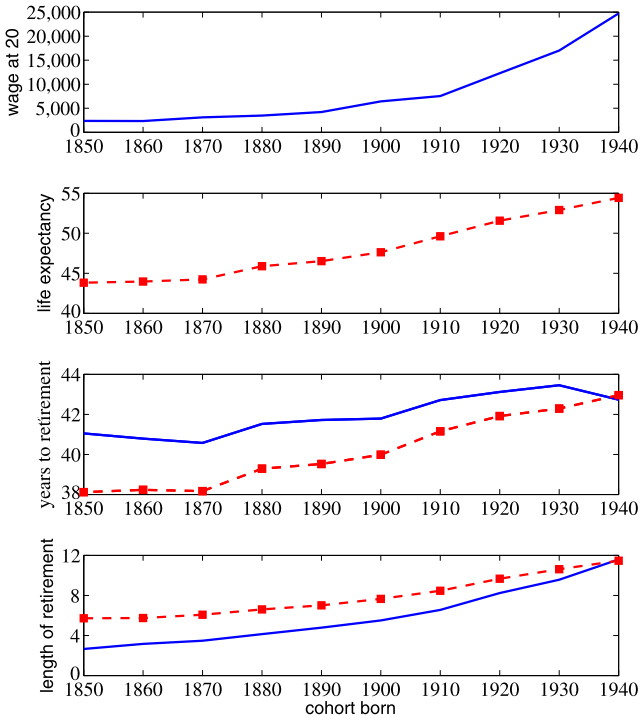
Appendix E. Health motive for retirement

The disutility from work is now measured by βD^{ψ} and the first order condition (7) is replaced by $\beta D(R)^{\psi} = w(R)c(R)^{-\sigma}$. The disutility from work generates a third channel through which health deficits matter for retirement. It serves to delay retirement. Notice that ψ affects only the age of retirement and that the same life trajectories (as shown in Fig. 1) can be generated by different combinations of β and ψ. Fig. E.2 replicates Fig. 4 for $\psi = 0.5$, i.e. a health deficit elasticity of leisure of 50 percent, and $\beta = 3.3$ such that the cohort born in 1940 continues to retire at age 62.7. The model predicts now a too steep increase of the age of retirement by about 2.5 years and consequently it underestimates the length of retirement.



Solid lines: data. Dashed lines: model prediction with imputed wages and prices and estimated medical technological progress. Parameters as for benchmark run except $\nu = 0.3$ and $\phi_2 = 0.00072$.

Fig. E.1. A century of increasing health and leisure: imputed prices: $\nu = 0.3$.



Solid lines: data. Dashed lines: model prediction with imputed wages and prices and estimated medical technological progress. Parameters as for benchmark run except $\beta = 3.3$.

Fig. E.2. A century of increasing health and leisure: imputed prices: $\psi = 0.5$.

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