The Journal of the Economics of Ageing 18 (2021) 100286

Contents lists available at ScienceDirect

The Journal of the Economics of Ageing

journal homepage: www.elsevier.com/locate/jeoa

R&D-driven medical progress, health care costs, and the future of human longevity

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ARTICLE INFO

JEL:
H50
I10
C60
O11

Keywords:
Medical R&D
Morbidity
Health care
Health expenditure share

ABSTRACT

We set up a life-cycle model of gerontologically founded human aging with overlapping generations to make quantitative inferences about the future development of morbidity, life expectancy, and the health expenditure share in GDP, conditional on the extent of future access to health care. Importantly, we take into account the endogeneity of medical technology to health good demand. For the baseline policy scenario of health care access, the calibrated model predicts substantial future increases in health and life expectancy, associated with rising shares of health expenditure in GDP. Fixing the expenditure share at the 2020 level severely reduces potential gains in health, longevity and welfare; for example, reducing the gains in life-expectancy at age 65 by about 4 years in the year 2050. Perhaps surprisingly, young individuals (i.e. those who would save the most health care contributions) would suffer the greatest losses in terms of life expectancy and welfare. These results reflect reduced incentives for medical R&D.

1. Introduction

A salient feature of structural economic development over the last decades is the secular expansion of the health sector and human longevity. Period life expectancy at birth increased by about 10 years between 1970 and 2015 in Japan, France, Germany, and the UK (OECD, 2017). At the same time, these countries experienced considerable growth of the health sector such that, across the board, health expenditure increased faster than GDP (Jones et al., 2016).[3]

Scholars agree that both the rise of health expenditure and improvements in longevity are related to medical technological progress.[4] Recent examples of health innovations include computerized diagnostic tests (e.g. medical imaging), personalized cancer therapy, and new treatments of virus infections like HIV or Hepatitis C.[5] More generally, Lichtenberg (2007) shows that later vintages of pharmaceuticals are more effective in reducing health deficits. Considering the evolution of 92 potentially lethal diseases he finds that conditions experiencing greater pharmaceutical innovation tend to have greater declines in

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[3] The increase was even larger in the U.S., albeit life expectancy increased by a somewhat lesser degree. For instance, in the U.S., health expenditure per capita grew by on average 4.1% annually since 1970 (Chernew and Newhouse, 2011; Gaynor et al., 2015).

[4] As argued convincingly by Chernew and Newhouse (2011), the continued increase of health expenditure shares requires at least one other continuously growing explanatory variable (and thus rules out institutional changes like health care reforms and other only occasionally changing variables). Okunade and Murthy (2002) establish a long-run relationship between medical R&D expenditure and health care expenditure. There may be a role for income as a driver of health costs, although some recent studies refute the luxury good hypothesis of health care by estimating an income elasticity of health expenditure below unity (Acemoglu et al., 2013; Baltagi et al., 2017).

[5] A promising example of a potentially powerful future technology is "targeted genome editing" like the *clustered, regularly interspaced, short palindromic repeat* (CRISPR) technology. It gives rise to the development of novel molecular therapeutics for human disease. The The Economist (2016) provides an overview on recent developments in anti-aging research.

https://doi.org/10.1016/j.jeoa.2020.100286

Available online 20 October 2020

mortality rates.[6]

Consistent with such evidence, this paper develops a multi-period overlapping generations model to make quantitative inferences about the future development of morbidity, life expectancy, and the health expenditure share in GDP, conditional on the extent of future access to health care and taking into account the endogeneity of medical technology to health good demand.

Individual health status is measured by the *health deficit index* developed by gerontologists (Mitnitski et al., 2002a, 2002b, 2005, 2007). It is defined as the fraction of bodily impairments possessed by an individual out of a long list of potential health deficits.[7] The health deficit approach conceptualizes morbidity and physiological aging as accumulation of health deficits, displaying positive path dependency. That is, untreated health deficits lead to new ones such that the mortality risk increases. The approach has been used in countless empirical studies in the natural sciences to predict mortality rates in a biologically founded way and is essential for the success of our study for two reasons. First, it allows us to calibrate our model, because health deficits are observed and easily quantifiable. The calibrated model is, *inter alia*, consistent with UK data on levels and evolution over time of age-specific mortality rates and health expenditure shares.[8] Second, capturing the empirically established path-dependency of health deficits is salient to understand the effects of changes in health care access.

In our model, changes in health care access affect the incentive to improve health goods by changing the demand for health goods for given levels of health deficits in the population and by affecting population ageing for given quality of health goods. Thus, endogeneity of medical R&D to health care access (the extent to which individuals are provided with appropriate health goods) will be a key to understand our results.

Our calibrated model suggests substantial future gains in life expectancy that are associated with significant declines in morbidity. The endogenously changing demographic structure and the evolution of age-structured health deficits leads to an increase in the health expenditure share in GDP by about two percentage points until 2080 in our baseline scenario.[9]

Despite the good news on human health, the entailed increasing utilization of medical goods and services has raised concerns about fiscal sustainability of health insurance systems and, more generally, the overall desirability of such trends. It motivated the discussion of curbing further increases in expenditure shares (Aaron and Schwartz, 1990; Ham and Glenn, 2003; Singer, 2009). For instance, the National Health Service (NHS) – managing tax-financed health care in the UK – limits access to hip replacements, knee surgeries (OECD, 2015) and coverage of a novel (albeit expensive) drug that for the first time heals Hepatitis C.[10] The standard reasoning for such measures is that some treatments like hip replacements, despite their positive effects on the quality of life, would be inconsequential for remaining life expectancy. However, this view has been proven wrong by gerontology research (e.g. Mitnitski et al., 2006). For instance, the physical difficulty to move is known to contribute to developing cardiovascular diseases that may considerably shorten life expectancy. The health deficit approach captures the feature of self-reinforcing health conditions and allows us to analyze the effects of pervasively limiting health care access.

Our analysis suggests that fixing the health expenditure share at its current level has severe consequences on future health and longevity. Aside from the obviously detrimental effects on health of the current population it also reduces market size for new medical products, which, in turn, suppresses medical R&D and therefore reduces future life expectancy gains. For example, we show that preventing the moderate increase in the health expenditure share under the baseline calibration of the model would, for instance, reduce remaining life expectancy of an individual who has reached age 65 in year 2050 by almost 4 years. We thus formalize and exploit an idea that goes back to Weisbrod (1991) who argues that the expansion of U.S. health care insurance has induced higher health R&D effort and newly developed technologies in association with increasing health care utilization and costs. Consistent with such a market size effect, Acemoglu and Linn (2004) found that the aging of the baby boomers is related to the development and market entry of new (age-specific) pharmaceuticals.

This leaves us with the fundamental normative question of how to solve the trade-off between promoting longevity and limiting increases in health costs. For this purpose we propose a welfare analysis that compares different future scenarios of health care access. We assume that marginal utility from consumption negatively depends on morbidity, in line with empirical evidence (Finkelstein et al., 2013). Our welfare analysis suggests that particularly future generations would incur dramatic welfare losses from fixing the health expenditure share at its current level, despite increases in their disposable income. We estimate, for instance, that someone who is currently 20 years old could expect a welfare loss of about 16–21 percent from the policy regime switch.[11] For those aged 20 in 2050 the estimated welfare loss is between 33 and 41 percent and associated with a reduction in remaining life expectancy by about 10 years.[12]

The remainder of the paper is organized as follows. Section 2 discusses our contribution in view of the related literature. The model is presented in Section 3. Section 4 provides the positive analysis of the evolution of life expectancy and morbidity under different health care access scenarios. Section 5 presents a comparative welfare analysis of the different policy scenarios. The last section concludes.

2. Contribution to the literature

Our main contribution is to highlight the interaction between endogenous medical technological progress and endogenous longevity as a function of access to health care. Doing so requires setting up a quantifiable model of an age-structured population.

The interaction of health R&D and health expenditure is also at the

[6] More recently, Lichtenberg (2020) shows that increases in the approvals of new cancer drugs in the U.S. in the period 2000–2014 have been associated with larger declines in premature mortality and hospitalization, prolonging life-expectancy of patients. That said, not all pharmaceutical R&D effort is targeted to improving effectiveness of treatments. So-called "me-too" drugs are a prime example. These have similar chemical structures as an original drug and are used for the same therapeutic purposes, but may differ in some respects such as adverse reactions or drug-drug interactions (Gagne and Choudhry, 2011; Aronson and Green, 2020). Examples are the numerous tricyclic antidepressants, beta-blockers and statins that are not "first-in-class". The theoretical model we propose in this paper is calibrated to capture the effect of an average medical innovation on the accumulation of health deficits and the evolution of mortality, assuming that improvements in the average drug effectiveness is similar in the future than in the past.

[7] According to Rockwood and Mitnitski (2007) and Searle et al. (2008), the exact choice of the set of potential deficits is not crucial, provided that the set is sufficiently large. We present a typical list of health deficits from Searle et al. (2008) that serves to compute the health deficit index (often called "frailty index") in the Online Appendix (Table A.1).

[8] Since medical R&D does not remain within national borders, the UK is taken as representative for advanced countries as a whole where the bulk of technological advancements take place.

[9] We assume a constant mark-up on prices of health goods. Thus, our results do not reflect the concern that health expenditure shares may be rising due to higher relative prices for pharmaceuticals.

[10] See http://www.hepatitisc.uw.edu/page/treatment/drugs/sofosbuvir-drug and World Health Organization (2016).

[11] We measure welfare changes from a regime switch in the health care system by an equivalent variation measure; see Section 5 for details.

[12] As the health deficit approach captures ageing as viewed by gerontologists, throughout, we focus on remaining life expectancies at age 20 and older and do not address child mortality.

center of the life-cycle models in Jones (2016) and Koijen et al. (2016).[13] Jones (2016) investigates the optimal allocation of R&D effort directed towards innovations for health and non-health purposes. He shows that non-health technological progress may optimally converge to zero growth such that the health expenditure share optimally converges to 100 percent under mild conditions. The study makes an important, eye-opening contribution to the debate whether there is too much health care expenditure and it paves the way for our research. Our study shifts the focus from a single-agent view to a multi-period, overlapping generations model with an explicit health care system to provide estimates of future longevity of an age-structured population as a function of health care utilization. Koijen et al. (2016) are interested in the premium to the return to health R&D investments which they attribute to the risk of government intervention. They do not consider longevity (assuming infinite planning horizons).

In another closely related strand of literature, Hall and Jones (2007) and Frankovic and Kuhn (2018) investigate the role of per capita income growth for health expenditure growth. Hall and Jones (2007) argue that declining marginal utility of material consumption implies a rapid increase in health expenditure as a rational response that allows individuals to extend life. The reason is that the marginal utility of life extension does not decline. They also compare the actual and socially optimal health spending in the U.S., suggesting that health spending is too low and that future medical progress calls for substantial future increases in health spending. However, they do not endogenize medical progress, whereas the response of medical R&D investment is critical to understand our results on the detrimental effects of limiting health care access for life expectancy and welfare. Closer to our analysis, Frankovic and Kuhn (2018) propose a multi-period OLG model with endogenous health innovations that interact with the demand for health care. Specifically, they examine the role of the expansion of public health insurance for health expenditure growth in the U.S. (in the period 1960–2010) vis-a-vis income growth and find that the former channel is more important than the latter. In contrast, we aim to predict the influence of alternative future health care scenarios on future R&D and longevity. The most important distinguishing features of our model are the micro-foundation of human health over the life-cycle as well as the modeling of R&D as a market activity of firms. Both features are important for our model calibration and for the model outcomes. In particular, the feature of path-dependency of health deficits implies that the dynamic costs of reduced health care access in terms of health and longevity are much larger than the pure direct (static) impact.

Our paper is also related to a strand of recent studies that utilized the health deficit approach to (re-) investigate the Preston curve (Dalgaard and Strulik, 2014), the education gradient (Strulik, 2018), the historical evolution of retirement (Dalgaard and Strulik, 2017), the role of adaptation for health behavior and health outcomes (Schuenemann et al., 2017), and the optimal design of social welfare systems (Grossmann and Strulik, 2019).[14] In contrast to the popular health capital model (Grossman, 1972), health deficits can directly be observed, which is important for our calibration purposes. Moreover, the health capital model would likely underestimate the effects of limiting health care access, because the impact of untreated health problems (conceptualized as a negative shock to health capital) would depreciate away as the individual gets older and eventually become negligible (see Almond and Currie, 2011; Dalgaard et al., 2017). In the health deficit approach, the opposite happens as health deficit accumulate in the course of life, in line with empirical evidence (see e.g. Mitnitski et al., 2002a; Mitnitski et al., 2002b). The methodological innovation of our paper is to incorporate the health deficit approach in a model with an age-structured population and endogenous medical progress.

Finally, there is a large literature outside economics that attempts to forecast future life expectancy by estimating statistical time trends. For instance, in a widely received paper, Kontis et al. (2017) account for model uncertainty with a Bayesian model averaging approach. Their results suggest a high probability of large gains in life expectancy at older ages in advanced countries, that parallel our findings in the case where governments do not aim at curbing increases in the health expenditure share. However, as acknowledged by the authors, their statistical approach has as "key limitation […] the inability to account for […] changes in the social, technological, and health systems determinants of health" (p. 8). These issues are explicitly taken into account by our economic approach that endogenizes health technology and varies health care utilization rates.

3. The model

Consider the following multi-period overlapping generations model in discrete time, indexed by t, in which individuals age by accumulating bodily impairments ("health deficits"). In line with the evidence on human aging, on average, individual health deficits correlate exponentially with age and are a highly relevant determinant of the probability of death (e.g. Mitnitski et al., 2002a, 2002b, 2005, 2007). Health goods are provided via a tax-financed health care system without copayments, as in the UK. The government runs a balanced budget. Improved quality of utilized health goods slows down the aging process.

Private firms decide competitively on medical R&D. Also the final good sector and factor markets are perfectly competitive, whereas health good providers charge markup prices. Markup factors can be thought of being determined by negotiations between health care representatives and health good suppliers (like in most advanced countries). There exists a perfect private annuity market and an international capital market that fixes the real interest rate at $\bar{r}$.

3.1. Households

Each period a new cohort is born. Mortality is cohort- and age-specific and determined by the accumulated health deficits at the individual level. Formally, the probability $m_{v,t}$ of a member of cohort v to die between period t and $t+1$, conditional on having reached age $t-v \geq 0$, is increasing in the health deficit index at that age, $d_{v,t} \in [0,1]$. There exists a threshold deficit state $d_{\max} \in (0,1)$ such that no individual survives beyond that state. Moreover, there is a maximum life span (irrespective of health deficits), T. These properties are captured by the parsimonious specification[15]

$$m_{v,t} = \begin{cases} \dfrac{1-e^{-\frac{(d_{v,t})^{\phi}}{\sigma}}}{1-e^{-\frac{(d_{\max})^{\phi}}{\sigma}}} \equiv \widetilde{m}\left(d_{v,t}\right) & \text{if } d_{v,t} < d_{\max} \text{ and } t < v+T-1 \\ 1 & \text{otherwise,} \end{cases} \tag{1}$$

where we assume $\sigma > 1$ and $\phi > 1$. Note that $\widetilde{m}(0) = 0$ and $\widetilde{m}(d_{\max}) = 1$. As will become apparent, specification (1) enables us to capture empirically observed, age-structured survival rates with a small set of parameters. By definition, survival rates $S_{v,t}$ and conditional mortality

[13] By contrast, Garber et al. (2006) and Grossmann (2013) endogenize medical R&D in static models with a health care sector.

[14] Grossmann and Strulik (2019) investigate the interaction between increasing health expenditure, which promotes longevity, and a publicly financed pay-as-you-go pension system that is challenged by (endogenously) changing demography. They do not incorporate health R&D, however.

[15] In the Online Appendix (Fig. A.1) we present an empirical foundation of the close connection between mortality rates and the health deficit index from three survey waves of Canadian cohorts aged 65+ (Mitnitski et al., 2006). The relationship is strictly convex. Less than 4% of the total population had a deficit index above 0.35, implying a very high probability of death above this value. According to (1), we have $\widetilde{m}'' > 0$ if $\phi \cdot \left(1 - \frac{(d_{\max})^{\phi}}{\sigma}\right) > 1$, which will hold in our calibrated model.

rates are related by

$$S_{v,t} = S_{v,v}\prod_{u=v}^{t-1}\left(1-m_{v,u}\right) \text{ for } t \geqslant v+1, \tag{2}$$

i.e., $m_{v,t} \equiv -(S_{v,t+1}-S_{v,t})/S_{v,t}$. The initial size of cohort v is $S_{v,v}$.

Each individual works for R periods and inelastically supplies one unit of labor in working age (and no labor afterwards). We thus implicitly assume that, conditional on survival, labor supply is independent of health status.[16] The total units of labor supplied to the economy in period t are given by $L_t = \sum_{u=t-R+1}^{t} S_{u,t}$.

Households have preferences over material consumption and health status. They choose the consumption path that maximizes expected life-time utility. Because the interest rate is fixed, saving decisions of households do not affect firm decisions. We can thus focus on the supply side for the non-welfare related predictions of the model. Life-time utility is introduced later to analyze the welfare implications of changing health care policy.

3.2. Final good sector

There is a standard final good that is chosen as numeraire. It is produced under perfect competition according to

$$Y_t = \left(K_t^Y\right)^{\alpha}\left(A_t L_t^Y\right)^{1-\alpha}, \tag{3}$$

$\alpha \in (0,1)$, where K_t denotes the physical capital input in period t, L_t^Y is the amount of labor in the consumption goods sector, and A_t is a measure of non-health knowledge with initial level $A_0 > 0$ and exogenous growth rate $g > 0$. Physical capital depreciates at rate $\delta^K \geqslant 0$. Thus, the user cost per unit of capital is given by $\bar{r} + \delta^K$. It is equal to the marginal product of capital, $\bar{r} + \delta^K = \alpha(A_t L_t^Y / K_t)^{1-\alpha}$. The wage rate, w_t, equals the marginal product of labor, i.e. $w_t/A_t = (1-\alpha)(A_t L_t^Y/K_t^Y)^{-\alpha}$, such that

$$\frac{w_t}{A_t} = \left(1-\alpha\right)\left(\frac{\alpha}{\bar{r}+\delta^K}\right)^{\frac{\alpha}{1-\alpha}} \equiv \omega. \tag{4}$$

3.3. Health deficit accumulation and health good utilization

Evidence from modern gerontology, that describes aging as an accumulation of health deficits, suggests that individual health deficits grow exponentially with age (e.g. Mitnitski et al., 2002a; Harttgen et al., 2013). Thus, we assume that the change in the deficit index of a member of cohort v between period t and $t+1$ is increasing in the deficit index accumulated until period t. The accumulation process is slowed down by receiving health input $E_{v,t}$ from the health care provider. The health deficit index evolves according to[17]

$$d_{v,t+1} - d_{v,t} = \begin{cases} \varrho d_{v,t} - \kappa E_{v,t} & \text{if } E_{v,t} < \dfrac{\varrho}{\kappa} d_{v,t}, \\ 0 & \text{otherwise}, \end{cases} \tag{5}$$

$\kappa > 0$, $\varrho > 0$, with initial value $d_{\min} \equiv d_{v,v} > 0$. Parameter ϱ is the growth rate of the health deficit index in absence of health interventions. It can be interpreted as the physiological "force of aging". κ is a shift parameter employed to calibrate the model.

There is a unit mass of health goods (and services), indexed by $j \in [0,1]$, each one targeting a different health deficit. Extending utilization of these goods slows down the accumulation of health deficits. For each member of a given generation, the probability to suffer from a particular health deficit is the same. We conceptualize health input, $E_{v,t}$, as health good consumption index, that equals the quality-weighted sum of consumption of the health goods appropriate for an individual to treat or prevent health deficits.

Consider some examples of health deficits from the set of potential health deficits used in the gerontology literature.[18] For instance, high blood pressure is known to contribute to developing cardiovascular diseases and may require beta blockers. Moreover, if help is needed to climb stairs, a hip replacement may not only be needed to cure the deficit but also to avoid further health deficits that may reduce longevity (like, again, cardiovascular diseases or those implied by dementia). As a final example, feeling lonely or feeling depressed may initiate unhealthy behavior, which, without treatment (like antidepressants), leads to further health deficits.

Formally, the quality of the latest vintage of health good j available in period t is denoted by $q_t(j)$. $Q_t \equiv \int_0^1 q_t(j)\mathrm{d}j$ denotes the average quality of the latest vintages of health goods ("stock of medical knowledge"). An individual born in v utilizes a set $\mathscr{J}_{v,t} \subset [0,1]$ of health goods of average quality with mass equal to the current health deficit index, $|\mathscr{J}_{v,t}| = d_{v,t}$. We normalize the maximally effective consumption per health good to unity capturing the notion of an optimal dose (like for pharmaceuticals and implants). We capture under-utilization of health care by allowing the actual consumption for any health good to be smaller than unity. The "health care provision wedge" in t is parameterized by $\varphi_t \in [0,1]$. One reason of under-utilization is an institutionally caused limit to health care access. Full utilization is reflected by $\varphi = 0$, whereas $\varphi = 1$holds in absence of a health system or full exclusion from it. Thus, an individual born at v receives the health input

$$E_{v,t} = \left(1-\varphi_t\right)\int_{j\in\mathscr{J}_{v,t}} q_t\left(j\right)\mathrm{d}j = \left(1-\varphi_t\right)d_{v,t}Q_t. \tag{6}$$

in period t, that depends on the interaction between the contemporaneous health care utilization $(1-\varphi_t)$, the current deficit state $(d_{v,t})$ and the average quality of health goods (Q_t). Substituting (6) into (5), the growth rate of the health deficit index is deterministic and independent of the deficit state. For $t \geqslant v$ it is given by

$$\frac{d_{v,t+1}-d_{v,t}}{d_{v,t}} = \begin{cases} \varrho - (1-\varphi_t)\kappa Q_t & \text{if } Q_t < \dfrac{\varrho}{\kappa(1-\varphi_t)} \equiv \overline{Q}_t, \\ 0 & \text{otherwise}. \end{cases} \tag{7}$$

Eq. (7) shows that individual morbidity evolves as an interaction of (R&D driven) health care quality and (exogenous) health care access. In line with leading gerontology research (e.g. Mitnitski et al., 2002a, 2002b, 2005, 2007), health deficits grow exponentially with age for given health technology and given health care utilization.

Given the mass of utilized health goods, $|\mathscr{J}_{v,t}| = d_{v,t}$, total health good consumption of surviving members of cohort v in period t (with population size $S_{v,t}$ and health care utilization $1-\varphi_t$) reads as

$$h_{v,t} = \left(1-\varphi_t\right)S_{v,t}d_{v,t} \tag{8}$$

measured in units per health good from the latest vintages. Aggregate demand for recent vintages in period t is obtained by summing up $h_{v,t}$ over all cohorts with living members:

$$H_t = \sum_{v=t-T+1}^{t} h_{v,t} = \left(1-\varphi_t\right)\sum_{v=t-T+1}^{t} d_{v,t}S_{v,v}\prod_{u=v}^{t-1}\left(1-m_{v,u}\right), \tag{9}$$

[16] In fact, at the individual level, a decline in health status typically has a very moderate effect on labor supply (see e.g. Jaeckle and Himmler, 2010; Hokayem and Ziliak, 2014).

[17] Health deficit accumulation would cease if the health input became sufficiently high. Although such a scenario could become conceivable with further biotechnological advances (De Grey and Rae, 2007), it does not arise in our calibrated model.

[18] See Table A.1 in the Online Appendix.

where we used (2) and (8) for the latter equation. As there is a unit mass of health goods, H_t is market demand faced by each health good producer. Sales are financed by the health care system via labor income taxation.

As will become apparent, our calibration strategy involves matching the empirically observed relationship between health inputs and health outcomes and its changes over time. It is important to note that, therefore, our modeling approach does not imply that other factors than health inputs are unimportant for health improvements. To the contrary, we follow other contributions in the macro-health literature (e.g. Hall and Jones, 2007; Jones, 2016) that do not explicitly model prevention behavior (like low-fat diet, avoidance of smoking, and exercise), environmental health factors (like air quality or sanitation) or inefficiencies in health insurance markets (like moral hazard). Like in this literature, by relating health inputs and outputs, our calibrated model implicitly controls for those determinants of health status.

3.4. Health good sector

Production of one dose of a health good requires $\chi > 0$ units of labor. Thus, marginal production costs in period t are χw_t.

There is a competitive R&D sector for each health good aiming to advance the treatment quality. A successful innovator provides a quality level that is by an amount $\gamma > 0$ higher than the quality of the previous vintage. As will become apparent, an innovator drives the incumbent out of business. The quality of health goods (including older vintages) deteriorates over time at rate $\delta^Q \in (0,\gamma)$. In the case of pharmaceuticals, depreciation of quality captures mutations of bacteria and viruses, with resistance of antibiotics being a prime example.

Denote by $\mu_{t+1}(j)$ the probability of a successful innovation of health good j that is commercialized in $t+1$. The quality of health good j then evolves according to

$$q_{t+1}\left(j\right) = \begin{cases} \left(1-\delta^Q\right)q_t(j)+\gamma & \text{with probability } \mu_{t+1}(j), \\ \left(1-\delta^Q\right)q_t(j) & \text{otherwise.} \end{cases} \tag{10}$$

Hence, the expected quality of health good j in period $t+1, \mathbb{E}[q_{t+1}(j)]$, is given by

$$\mathbb{E}\left[q_{t+1}(j)\right] = \mu_{t+1}(j)\left[q_t(j)\left(1-\delta^Q\right)+\gamma\right] + \left(1-\mu_{t+1}(j)\right)q_t(j)\left(1-\delta^Q\right). \tag{11}$$

The innovation probability, $\mu_{t+1}(j)$, is determined by R&D investment that affects the perceived innovation probability of a firm $j, \widetilde{\mu}_{t+1}(j)$, and a probability of innovation that is exogenous to firms, $\overline{\mu}_{t+1}$[19]; i.e., $\mu_{t+1}(j) = 1-(1-\overline{\mu}_{t+1})(1-\widetilde{\mu}_{t+1}(j))$. Let $l_t(j)$ denote the amount of labor devoted to research by a representative R&D firm in health sector j and assume that the perceived probability of a successful innovation is proportional to the employment of researchers:

$$\widetilde{\mu}_{t+1}(j) = \widetilde{\xi}_t l_t(j), \text{ with } \widetilde{\xi}_t \equiv \xi\cdot\left(L_t^Q\right)^{-\vartheta}, \tag{12}$$

$\xi > 0, \vartheta \in (0,1)$, where L_t^Q is the aggregate amount of health R&D labor in t. The productivity level $\widetilde{\xi}_t$ is taken as given in the decision of R&D firms and captures a negative R&D externality: $\vartheta > 0$ implies a wedge between private and social returns to R&D that arises because firms do not take into account that rivals work on the same idea such that, from a social point of view, some of the R&D is duplicated ("duplication externality").[20] In a symmetric equilibrium, where $l_t(j) = L_t^Q$ for all $j \in [0,1]$, we obtain $\widetilde{\mu}_{t+1}(j) = \widetilde{\mu}_{t+1} = \xi\cdot\left(L_t^Q\right)^{1-\vartheta}$ for all j.

We assume that there is an intertemporal spillover from the existing stock of medical knowledge, Q_t, that manifests itself in the probability of an unintentional innovation:

$$\overline{\mu}_{t+1} = \eta Q_t, \tag{13}$$

$\eta \in [0, \delta^Q/\gamma)$. According to (12) and (13), the total probability of medical progress in any sector is given by

$$\mu_{t+1} = \overline{\mu}_{t+1} + \left(1-\overline{\mu}_{t+1}\right)\widetilde{\mu}_{t+1} = \eta Q_t + \left(1-\eta Q_t\right)\cdot\xi\cdot\left(L_t^Q\right)^{1-\vartheta}. \tag{14}$$

By the law of large numbers, there is no aggregate risk. Thus, $\int_0^1 \mathbb{E}[q_{t+1}(j)]\mathrm{d}j$ is deterministic and equal to Q_{t+1}. According to (11), it evolves as

$$Q_{t+1} = \gamma\mu_{t+1} + \left(1-\delta^Q\right)Q_t, \tag{15}$$

for a given initial level $Q_0 > 0$. Substituting (14) into (15), we obtain

$$\frac{Q_{t+1}-Q_t}{Q_t} = \frac{\gamma(1-\overline{\mu}_{t+1})\widetilde{\mu}_{t+1}}{Q_t} - \delta^Q + \gamma\eta = \frac{\gamma\left(1-\eta Q_t\right)\xi\left(L_t^Q\right)^{1-\vartheta}}{Q_t} - \widetilde{\delta}^Q, \tag{16}$$

where $\widetilde{\delta}^Q \equiv \delta^Q - \gamma\eta > 0$. Thus, the growth rate of the stock of medical knowledge, Q, is a declining function of its level. Since $\widetilde{\delta}^Q > 0$, the growth rate of Q becomes negative without intentional R&D (i.e. $Q_{t+1} < Q_t$ if $L_t^Q = 0$).

3.4.1. Pricing

The price markup of health goods can be thought of as an outcome of negotiations between the health care provider and (a representative body of) health good producers like pharmaceutical companies.[21]

Prices for older vintages are bid down to marginal costs, leaving the suppliers with zero profits. We assume that, therefore, older vintages are not supplied anymore. The industry leader can charge a mark up that is increasing in the quality advantage vis-à -vis previous vintages. Denote by $\mathfrak{q} > 0$ the (absolute) quality advantage of the industry leader over the competitor with the second-highest quality product in the same market. We assume that the mark up factor is given by $1 + f(\mathfrak{q})$, where f is an increasing and strictly concave function that fulfills $f(0) = 0$. It captures the price setting power of health good providers as a function of the quality advantage in the market. If the leading firm is one step ahead of the closest competitor (i.e. $\mathfrak{q} = \gamma$), it realizes profits per unit sold equal to $f(\gamma)\chi w$. If the leading firm is two steps ahead of the closest competitor (i.e. $\mathfrak{q} = 2\gamma$), it realizes profits per unit equal to $f(2\gamma)\chi w$. The profit increase for the industry leader by innovating, i.e. by advancing two steps rather than one step ahead, is $[f(2\gamma) - f(\gamma)]\chi w$. Since strict concavity of f

[19] Those innovations may be thought of occurring unintentionally or being primarily based on ideas of non-profit innovators like public research institutions. The inventions of Penicillin and Viagra are prime examples of major breakthroughs that were not intended to treat the health problems they target today.

[20] The argument is analogous to the one that Jones (1995) made in a non-health R&D context. For pharmaceutical R&D, Miller et al. (2015) find that despite legal requirements and ethical standards a median of 43% of clinical trials per drug were not registered and almost half of all reviewed drugs had at least one undisclosed trial in a later phase, giving rise to duplication of R&D.

[21] Pharmaceutical companies may draw their negotiation power via lobbying and marketing that influences government negotiators and public opinion, respectively, on the merits of pharmaceuticals. For instance, interest groups representing the pharmaceutical sector strongly argue that they need to earn high profits enabling them to conduct R&D and therefore have to charge high prices that should be covered by health insurance. In the UK, prices for pharmaceuticals are regulated and based on a non-contractual agreement between the UK Department of Health and the Association of the British Pharmaceutical Industry. Similarly, in Germany and Switzerland, among others, health care suppliers negotiate with pharmaceutical companies the maximum price covered by the mandatory health insurance.

and $f(0) = 0$ imply $f(2\gamma) < 2f(\gamma)$, we have $[f(2\gamma) - f(\gamma)]\chi w < f(\gamma)\chi w$. Consequently, the incumbent firm would strictly prefer to invest in R&D in a second market rather than advancing its latest vintage.[22] Since it does not pay off for the leader to innovate, the incumbent is driven out of business when there is an innovation in the market it leads. This means that the leader's quality advantage to the closest competitor is $\mathfrak{q} = \gamma$, implying that the price p_t of each health good is given by

$$p_t = \Gamma\chi w_t = \Gamma\chi\omega A_t, \tag{17}$$

where $\Gamma \equiv 1 + f(\gamma)$ is the markup factor.

3.4.2. R&D Incentives

Ruling out bubbles and arbitrage possibilities in the financial market and accounting for the probability $\mu_u(j)$ that health good producers are driven out of business in period $u \geqslant t+1$, the value of a health good innovation in t reads as

$$V_t\left(j\right) \equiv \pi_t\left(j\right) + \sum_{u=t+1}^{\infty} \pi_u\left(j\right) \frac{\prod_{s=t+1}^{u}(1-\mu_s(j))}{(1+\bar{r})^{u-t}}, \tag{18}$$

where $\pi_t(j)$ the instantaneous (i.e. operating) profit of a health good producer in sector j. In equilibrium, R&D workers earn the same wage rate as production workers. Thus, a representative R&D firm searching for a vertical innovation of health good j solves

$$\max_{l_t(j)} \left\{ \widetilde{\mu}_{t+1}\left(j\right) V_{t+1}\left(j\right) - w_t l_t\left(j\right) \right\} = (\widetilde{\xi}_t V_{t+1}(j) - w_t) l_t\left(j\right), \tag{19}$$

according to (12). Eq. (19) implies that there is a symmetric equilibrium where the expected revenue per R&D worker equals the wage rate and expected economic profits from R&D activity equal zero (reflecting free entry). Using (17) and recalling that H_t is the demand for each recent health good vintage, the operating profit per health good producer in symmetric equilibrium is

$$\pi_t(j) = (p_t - \chi w_t)H_t = (\Gamma - 1)\chi w_t H_t. \tag{20}$$

Limiting health care access by imposing a higher φ has two detrimental effects on health status and life expectancy. First, according to (7), it speeds up the evolution of health deficits for a given stock of medical knowledge, Q. Second, according to (9), while saving health costs, it lowers market size for health goods, H. Consequently, according to (18) and (20), an increase in φ reduces the value of a health good innovation, thus depressing R&D incentives.

4. Equilibrium analysis

We now explore potential futures of human health, longevity, and the health expenditure share in GDP, conditional on health care access. The comparative welfare analysis of different policy scenarios is examined in Section 5.

4.1. Preliminaries

Let $L_t^H \equiv \chi H_t$ denote total employment in health goods production. Labor market clearing implies that

$$L_t^Y + L_t^H + L_t^Q = L_t. \tag{21}$$

Defining employment shares by $\ell_t^Y \equiv L_t^Y/L_t, \ell_t^H \equiv L_t^H/L_t$ and $\ell_t^Q \equiv L_t^Q/L_t$, we have $\ell_t^Y + \ell_t^H + \ell_t^Q = 1$. The gross domestic product (GDP) reads as $GDP_t \equiv Y_t + p_t H_t$. Thus, the health expenditure share of the economy is given by

$$\mathfrak{s}_t \equiv \frac{p_t H_t}{GDP_t} = \frac{p_t H_t}{Y_t + p_t H_t}. \tag{22}$$

Finally, denoting the size of the retired (old-aged) population by $O_t \equiv \sum_{u=t-T+1}^{t-R} S_{u,t}$, the "dependency ratio" (ratio of retirees to workers) is given by

$$DPR_t \equiv \frac{O_t}{L_t} = \frac{\sum_{u=t-T+1}^{t-R} S_{u,t}}{\sum_{u=t-R+1}^{t} S_{u,t}}. \tag{23}$$

The dynamical system and the long run equilibrium are summarized in Appendix A. We solve the model numerically using the relaxation method of Trimborn et al. (2006).

4.2. Calibration

Our calibration strategy aims to match the most important long-run trends of health inputs and health outcomes that play a central role in our model: survival rates (i.e. longevity), health expenditures, health sector employment, health deficit accumulation, and the probability of health innovations.

We assume that individuals become economically active at age 20 and die at age 120 at the latest; thus, $T = 101$. In fact, for modern times, 120 years seems to be the maximum life-span, irrespective of increasing life-expectancy in the last decades. The retirement age is reached after $R = 43$ working years (i.e. at age 63).[23] Using Canadian data, Mitnitski et al. (2002a) suggest that the average health deficit index for an individual at the age of 20 is $d_{\min} = 0.03$. The deficit state that leads to death for sure approximately is about two thirds (Rockwood and Mitnitski, 2006), suggesting $d_{\max} = 0.67$.

According to Karabarbounis and Neiman (2014, "CLS KN merged"), the arithmetic average of the corporate labor share in total income for the period 1975–2012 in the U.S. and the period 1987–2011 in the UK has been 62 percent. From (3), $wL^Y/Y = 1-\alpha$. We thus set $\alpha = 0.38$. Also in line with evidence, we choose $\bar{r} = 0.05$ for the real interest rate and $\delta^K = 0.07$ for the depreciation rate of physical capital (Grossmann and Steger, 2017).[24] The wage growth rate, g, is set equal to the annual growth rate of income per capita in the US and UK for period 1960–2011, $g = 0.02$ (Jones et al., 2016).

The main challenge is to dynamically calibrate the model such that we simultaneously match empirical survival rates, health resources data, and observed health deficit states. Since our modeling of the health system is closest to the one in the UK, we match (i) empirical survival rates for ages 20–100 and periods 1950, 1970, 1990, 2010 in the UK, (ii) the ratio of health expenditure to GDP ($\mathfrak{s}_t$) between 1980–2010 in the UK, and (iii) the UK employment share in the health sector (ℓ_t^H). Importantly, as medical R&D activity is central to our analysis, the UK should be interpreted as representing the advanced world as a whole. We also aim to match the average rate of change of the health deficit index ($d_{v,t}$) and the effective patent life (the inverse of the probability of an incumbent to be driven out of the market), $EPL_t \equiv 1/\mu_{t+1}$, for pharmaceuticals.

Doing so, we set the force of aging parameter ϱ to the typical value of 4 percent (Dalgaard and Strulik, 2014). Matching survival rates from year 1950 onwards requires initial conditions for the deficit index of all cohorts with living members in year 1850. Denote the vector of initial deficit states, in year 1850, by $\boldsymbol{d}_0$. We assume that $\boldsymbol{d}_0$ results from a

[22] See Grossman and Helpman (1991) for a similar argument in a context of Bertrand competition.

[23] In the UK, similar to the OECD averages, the average age of withdrawal from the labor market is around 64 for males and slightly below 62 for females in the 2000s (Mitchell and Guled, 2010).

[24] Setting $\bar{r}$ to 5 percent is motivated by the range of net returns for different types of capital investments found by Jorda et al. (2019), by assuming that $\bar{r}$ is between the estimated net return for risky and safe assets. Also note that their evidence suggests no time trend of the real interest rate.

policy regime in which a health care system has never existed, i.e. from the physiological force of ageing (rate of health deficit accumulation in absence of health inputs), $\varrho = 0.04$. The depreciation rate of the health good quality index, δ^Q, is set to the moderate value of 2 percent.[25]

According to (1), (2) and (7), given $\boldsymbol{d}_0$, the evolution of survival functions is exclusively driven by the exogenous time paths $\{\varphi_t\}_{t=0}^{\infty}$ and $\{S_{v,v}\}_{v=0}^{\infty}$ and the endogenous time path of medical knowledge, $\{Q_t\}_{t=0}^{\infty}$, with initial state Q_0. We assume that initial cohort size $S_{v,v}$ is non-decreasing and the health care wedge φ_t has been declining over time.[26] The time path of $S_{v,v}$ reflects the trend of mortality reduction of young individuals. The assumed time path of φ_t is roughly consistent with the historical improvements in the health care system in the UK and elsewhere. First, we assume a moderate decline of φ_t until 1950.[27] The foundation of the NHS in 1948 speeded up improvements in the access to health care for some time, which we capture by a steeper decline in φ_t. We assume a particularly fast decline of φ_t in the time period 1997–2010 to capture a series of health care reforms associated with extending employment in the health sector that halved NHS waiting lists for treatment from 1.3 million people in 1998 to under 600,000 in 2008 (Boyle, 2011).[28] For the future, we assume that φ_t decreases moderately, from about 0.15 in 2010 to 0.05 in year 2080 in the baseline calibration.[29] In addition, we investigate an alternative scenario where the health care wedge is extended after 2020 to fix the health expenditure share at its 2020 level. Q_0 is set to one percent of the steady state value of Q that results for $\varphi = 0.05$.[30]

The relatively fast improvement of health care access in recent times turns out to be critical to match the evolution of the health expenditure share in GDP ($\breve{s}_t$). The calibrated model implies that $\breve{s}_t$ is 5.0 percent in 1980, 5.1 percent in 1990, 6.2 percent in 2000 and 8.3 percent in 2010, compared to the observed UK levels of 5.1, 5.1, 6.3 and 8.6 percent, respectively (OECD, 2015; Tab. A.5). The increase was similar in other advanced countries (Jones et al., 2016). The coincidence of health care reforms in the 1990s and 2000s along with fast rising health expenditure strongly suggests that both are connected via better health care access.

The employment share in the health sector critically depends on the labor requirement per unit of health good (χ). We could approximate ℓ_t^H with the employment share in human health activities, as published by the OECD. For the UK, in 2010, it was 7.3 percent.[31] Including additionally residential care and social work activities (that may include other activities than health care provision) would suggest that ℓ^H was 12.7 percent. We set $\chi = 0.9$ to obtain an intermediate value of ℓ_t^H of 10 percent in 2010.Survival rates depend on the link between health deficits and mortality rates, as driven by the curvature parameters (σ, ϕ) in (1), and on the evolution of health deficits in (7) that is affected by κ. Medical R&D technology parameters (ξ, ϑ), innovation step size (γ), and the strength of the intertemporal innovation spillover (η) jointly govern R&D incentives via the (intentional) probability to innovate, $\widetilde{\mu}_{t+1}$. R&D incentives are, in addition, determined by the per period profit stream $\{\pi_t\}$ that, according to (20), depends on the mark up for health goods (Γ), labor requirement per unit of health good output (χ), and the time path of market size for health goods, $\{H_t\}$. According to (9), H_t critically depends on health care access, φ_t. Calibrating the model is thus involved with complex interactions between health innovations and market size. Fortunately, the exact determination of the probability to innovate is not critical for our results, which mitigates concerns of remaining degrees of freedom. Appendix B clarifies this point further by providing a steady state analysis that highlights the relationship between endogenous observables that we match.

Fig. 1 shows that the calibrated model fits the historical survival functions for the UK quite well. The most important deviation of the calibrated model (solid lines) from the data (circles) is for middle-aged individuals in 1950 and to a lesser degree in 1970. Importantly, we use the cross-section of mortality rates for a given year rather than those for a given cohort over time. This procedure is consistent with the standard way of computing "period life expectancy", but different to $S_{v,t}$ in the theoretical model.[32] However, period life expectancy does not account for future decreases in mortality rates that in our model are implied by changes in access or quality to health care over time. Thus, for life expectancy projections in the numerical analysis we will also employ the concept of "cohort life expectancy".

The implied average rate of change of the health deficit index across cohorts is 3.8 percent. According to Rockwood and Mitnitski (2007), the estimated rate of change of the health deficit index in the cross-section of cohorts is similar among advanced countries and around 4 percent. We consider the possibility to calibrate the model to a summary measure of observable health status jointly with mortality rates a major advantage of employing the health deficit approach.

The calibrated model implies a non-profit driven innovation probability of $\overline{\mu}_{t+1} = 0.034$ in 2010. The total innovation probability is $\mu_{t+1} = 0.08$, implying an effective patent life (the inverse of the probability of an incumbent to be driven out of the market) of $EPL_t \equiv 1/\mu_{t+1} = 12.5$. This is close to the median (average) EPL of 12.6 (12.2) years for pharmaceuticals in the sample of Hemphill and Sampat (2012).[33] Finally, the implied ratio of population size aged 63+ (retirement age) to the population size aged 20–62 (working age), DPR_t, is 40 percent for 2010.[34]

[25] Recall that Q captures the average quality of health goods rather than physical health equipment, which justifies assuming a considerably lower depreciation rate than for physical capital (δ^K). As previously discussed, depreciation of health good quality may be caused by mutations of viruses and bacteria, for instance.

[26] See Fig. A.2 in Online-Appendix for details.

[27] In the UK, the public health care sector remained limited until the mid 20th century, albeit health improvements occurred via better access to sanitation. According to Light (2003, p. 26): "In 1911, Parliament passed a very limited national health insurance act that covered workers (but not dependents) for primary care, pharmaceutical drugs, and cash benefits during sickness and disability. Provident societies, doctors' clubs, and fraternal organizations offered varying degrees of voluntary insurance coverage. Otherwise, health care was financed by private fees, charity, or through public hospitals."

[28] For instance, the median average waiting times for elective treatment like hip replacements and heart surgery fell from 12.7 weeks in 2002 to 4.3 weeks in 2010. For other advanced countries, see OECD (2015, Figs. 7.11–7.13)). Recent improvements of the British health care system are also reflected in a newly created "Healthcare Access and Quality (HAQ) Index" based on measuring mortality that should not be fatal in the presence of effective medical care (Barber, 2017). In the UK, the HAQ Index (with a range from 0 to 100) improved from 74.3 in 1990 to 82.7 in 2010.

[29] For instance, the density of physicians is much lower in rural areas than in urban areas, suggesting that access to health care is still severely limited in rural regions (OECD, 2015, Fig. 7.10). The trend towards urbanization and better information about treatment possibilities of patients could thus continue to improve health care utilization in the future.

[30] Our calibrated model leads to the case where steady state quality of health goods $\widehat{Q} \equiv \lim_{t\to\infty} Q_t < \overline{Q} = \frac{\varrho}{0.95\kappa}$. We can verify that the steady state equilibrium of the calibrated model is saddle-point stable.

[31] See http://stats.oecd.org/Index.aspx?DataSetCode=SNA_TABLE3, retrieved on January 31, 2016.

[32] Corresponding to Fig. 1, Table A.2 in the Online-Appendix compares in detail the remaining "period life expectancy" predicted by the model with the empirical one for the UK.

[33] Hemphill and Sampat (2012) report a standard deviation of three years, in line with an estimated range of 10–15 years for EPL of pharmaceuticals in the sample analyzed by Grabowski and Kyle (2007).

[34] This is considerably higher than the level in the data (33.1 percent); see Office for National Statistics (2016). The deviation mainly reflects our neglect of immigration into the UK labor market that was primarily enabled by the free movement of labor within the European Union. Notably, we are interested in changes of DPR_t over time rather than its level.

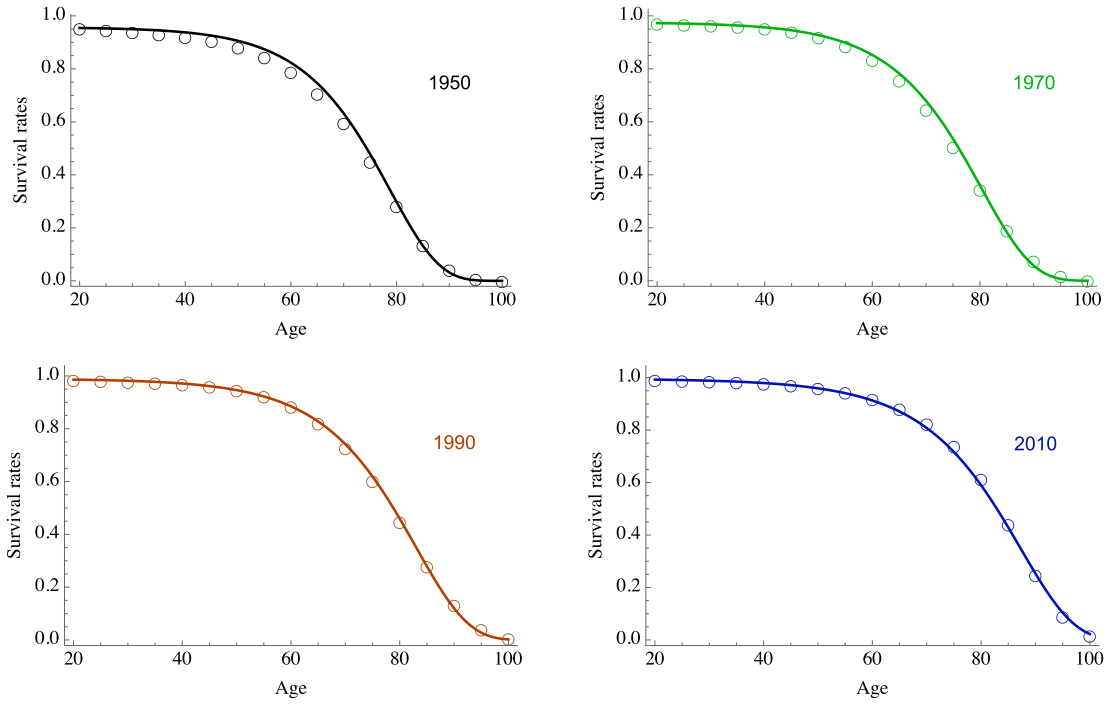

Fig. 1. Survival curves for 1950, 1970, 1990 and 2010 based on contemporaneous mortality rates: Calibrated model vs. UK data. (Notes: (1) Calibrated model: solid lines, empirical series: circles. (2) Data source: www.mortality.org. (3) Time paths $\{\varphi_t\}_{t=0}^{\infty}$ and $\{S_{v,v}\}_{v=0}^{\infty}$ are displayed in Fig. A.2 (Online Appendix). (4) Initial quality index (in 1850) $Q_0 = 0.01 \cdot \lim_{t\to\infty} Q_t$ for $\lim_{t\to\infty}\varphi_t = 0.05$. (5) Other parameters: $\alpha = 0.38$, $\delta^K = 0.07$, $\sigma = 1.5$, $\phi = 2.65$, $\chi = 0.9$, $\varrho = 0.04$, $\kappa = 0.06$, $\xi = 0.065$, $\eta = 0.12$, $\delta^Q = 0.02$, $\vartheta = 0.6$, $g = 0.02$, $\bar{r} = 0.05$, $d_{\min} = 0.03$, $d_{\max} = 0.67$, $\gamma = 0.1$, $\Gamma = 1.25$, $T = 101$, $R = 43$).

4.3. Results

We now examine from the year 2020 onwards the evolution of cohort-specific survival rates ($S_{v,t}$), age-specific morbidity ($d_{v,t}$), age-specific health care demand ($h_{v,t}$), the total health expenditure share (ς_t), the employment structure (ℓ_t^H, ℓ_t^Q), and the old-age dependency ratio (DPR_t) for two scenarios of future health care access. We also investigate the implications for age-specific life expectancies in these scenarios, distinguishing period and cohort life expectancy.

4.3.1. Baseline scenario

We start with the implications of the baseline scenario, i.e. for the case of moderately decreasing φ_t from 0.15 in 2010 to 0.05 in year 2080. Panel (a) of Fig. 2 displays the predicted cohort-specific survival rates ($S_{v,t}$) for 2020 (solid black line), 2050 (dashed blue line) and 2080 (dotted green line).[35] The black line, for instance, shows the surviving fraction of a cohort born in year 2020 minus the age shown on the horizontal axis. For example, at age 80 we read off the size of the cohort born in 1940 whose surviving members are 80 years old in the year 2020. The figure shows that there are considerable upward shifts of survival rates over time. For instance, whereas only 57.8 percent of those born in 1940 survive to age 80 (black line), 77.7 percent of those born in 1970 survive until age 80 (dashed blue line). In 2080, 87.1 percent of those born in 2000 are still alive (dotted green line).

Rising survival rates are driven by declining morbidity, displayed in panel (b). Age-specific mortality decreases over time because individuals become healthier at any given age, i.e. health deficits ($d_{v,t}$) are accumulated at lower rates with increasing age. For instance, the health deficit index for 80 years old individuals is 18.5 percent in the year 2020, 12.9 percent in the year 2050, and 9.7 percent in 2080. The aging process is slowed down because the stock of medical knowledge (Q_t) is increasing and because there is better access to health care.

The evolution of health deficits ($d_{v,t}$) determines, in interaction with survival rates ($S_{v,t}$), the evolution of age-specific health care demand ($h_{v,t}$), according to (8). As displayed in panel (c), total age-specific health care demand is a hump-shaped function of age, reflecting that health deficits are increasing with age (determining individual health care demand) whereas survival rates (and thus cohort sizes) are decreasing with age.

Over time, the curve shifts to the right, i.e. total health care demand for a given age decreases for younger age-groups and increases for older ones. For younger individuals, the shift reflects that improvements in the quality of health goods have little effect on survival rates, as these are high to begin with. By contrast, total health care demand for older age-groups is rising over time because of considerable increases in survival rates.

Consequently, despite declining morbidity and declining mortality at any age, population aging may result in increasing health expenditure shares (ς_t). In fact, according to panel (d), the health expenditure share increases from 8.4 percent in 2020 to 9.2 percent in 2050 and 10.3 percent in 2080. The kinks in 1997 and 2010 result from the fact that improvements in health care access were particularly large in the period 1997–2010, as captured by our calibrated model. Panel (e) suggests that increases in health expenditure shares will be associated with increases in the health employment share (ℓ_t^H), albeit not as fast as before 2020. Importantly, increasing health expenditure raises the incentive for health innovations through increased market size. This implies that the medical R&D labor share (ℓ_t^Q) is rising over time as well, as shown in panel (f). The increasing R&D effort leads to improvements in the quality of health care (Q_t) that drives the trend of declining morbidity and mortality.

Demographic change induced by human aging leads to a rising old-

[35] Strictly speaking, the figure shows remaining cohort sizes. However, according to Figure I in Appendix, cohort sizes at age 20 ($S_{v,v}$) are close to one from 1950 onwards. It is thus innocuous to implicitly assume that cohort sizes at birth are all normalized to unity, such that $S_{v,t}$ can be interpreted as survival rates.

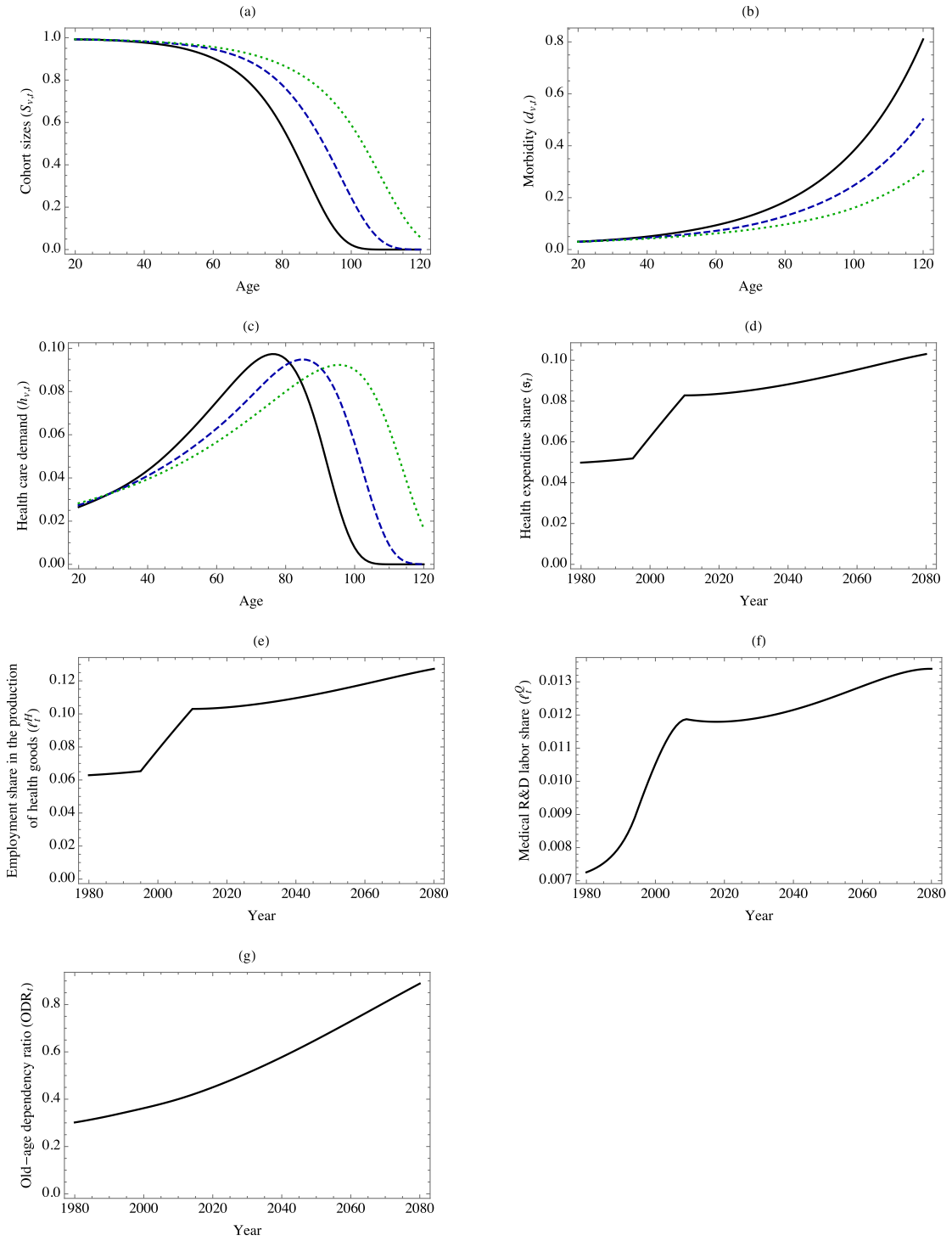

Fig. 2. The future of human health, longevity and health costs for the baseline policy scenario. (Notes: (1) Panels (a)–(c): Solid (black) line for 2020, dashed (blue) line for 2050, dotted (green) line for 2080. (2) Parameters as for Fig. 1). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

age dependency ratio (DPR_t), see (23). The interesting question, however, is by how much we should expect the old-age dependency ratio to rise. One advantage of our projection is to account for endogeneity of health care quality and possible changes to health care access. Looking at the evolution of the ratio of retired population (aged 63+) working-age population (aged 20–62), panel (g) suggests that *DPR* rises from 45 percent in 2020 to 65.2 percent in 2050 and 88.9 percent in 2080. Thus, our model implies that the ratio of retirees to workers will be doubling over the next 60 years, when the retirement age remains at its current level.

In sum, our model gives rise to an important insight: population aging that is associated with health improvements at any age may be associated with rising health expenditure shares even if prices of health goods grow at the same rate as income. In this sense, rising health costs are good news: they indicate that people live on average a longer *and*-healthier life.

4.3.2. *Constant health share scenario*

The increase in health expenditure shares has sparked an intensive

debate on health care access in many advanced countries. We now evaluate the effects of fixing the health expenditure share from the year 2020 onwards ("constant health share scenario"), by adjusting health care utilization. It turns out that achieving this goal requires an increase in the health care provision wedge, φ_t, from 11 percent in 2020 to 17 percent in year 2050 and 27.2 percent in year 2080.[36] The implications can be seen in Fig. 3.

The thin lines in panels (a)–(c) of Fig. 3 repeat the results for the baseline scenario shown in Fig. 2, whereas the thick lines correspond to the constant health share scenario. Panel (a) shows that survival rates in the constant health share scenario are predicted to improve by less than in the baseline scenario. The differences across policy regimes are particularly visible for the year 2080. Likewise, morbidity ($d_{v,t}$) improves by less, as shown in panel (b). Panel (c) shows that age-specific health care demand ($h_{v,t}$) is lower compared to the baseline scenario despite higher morbidity at any age, particularly for older age-groups. This outcome reflects the fact that survival rates of older cohorts improve by less over time in the alternative scenario.

In panels (d)–(g), the solid lines reflect results from the baseline scenario whereas dashed lines reflect results from the constant health share scenario. Panel (d) displays the health expenditure share ($\breve{s}_t$), which is, by design, constant in the latter scenario. Consequently, the employment share in the production of health goods (ℓ_t^H) stays basically constant as well, as shown in panel (e). Panel (f) shows that the medical R&D labor share (ℓ_t^Q) is lower than in the baseline scenario and even decreases slightly over time. This dynamic incentive effect of curbing future increases in the health expenditure share adds to the static effect of reduced health care usage to jointly slow down both demographic change and health improvements in the population. Consequently, as shown in panel (g), the old-age dependency ratio (DPR_t) rises somewhat more moderately than in the baseline scenario, from 45 percent in 2020 to 64.6 percent in 2050 and 83.6 percent in 2080.

4.3.3. *Life expectancy effects*

We derive age-specific (remaining) life expectancies from the age-specific mortality rates for the two scenarios in two ways. First, we calculate for both scenarios the "period life expectancy", that uses the contemporaneous mortality rates from the cross-section of cohorts (pretending they stay constant over time; see e.g. Kontis et al., 2017). As will become apparent, this may dramatically underestimate future increases in life expectancy. We therefore also compute "cohort life expectancy", based on future age-specific mortality rates.

Period Life Expectancy. Fig. 4 displays period life expectancy at a given year for 20 and 65 years old individuals for the baseline scenario (solid line) and the constant health share scenario (dashed line). Circles indicate the evolution of the respective empirical period life expectancies in the UK until 2010. For the baseline scenario, we observe that 20 years old individuals in the year 2020 (born in 2000) expect to live until age 83.6 under the (incorrect) assumption that age-specific mortality rates in a given year will not improve over time. Analogous figures are 93.5 years and 103.7 years for 20 years old individuals in 2050 and 2080.[37] Individuals aged 65 in 2020, 2050 and 2080 expect to live until age 86.9, 96.2 and 106.2, respectively.

Under the constant health share scenario, period life expectancy increases by less than in the baseline scenario. The difference across scenarios is 0.8 years and 4.6 years for 20 years old individuals in year 2050 and 2080, respectively, and 0.7 and 4.0 years for 65 years old individuals in 2050 and 2080.[38] In sum, implementing the cost-saving health care reform is particularly detrimental in the longer run, characterized by a sizable reduction of the potential gain in life expectancy.

Cohort Life Expectancy. Remaining cohort life expectancy of a member of a cohort born in v is computed as follows. We use the number of persons surviving to age $t-v$, $S_{v,t}$, to calculate the "person-years lived" between ages $t-v$ and $t-v+1$ for individuals born in v as $\mathscr{P}_{v,t} \equiv S_{v,t+1} + 0.5 \cdot S_{v,t} m_{v,t}$, where $S_{v,t} m_{v,t}$ is the number of persons dying between age $t-v$ and $t-v+1$. The total number of years lived after attaining age $t-v$ is given by $\mathscr{N}_{v,t} \equiv \sum_{u=t}^{v+T-1} \mathscr{P}_{v,u}$. Remaining life expectancy at age $t-v$ is then obtained as $\mathscr{N}_{v,t}/S_{v,t}$.

Fig. 5 displays the predicted evolution of cohort life expectancy at age 20 and 65. We see that in both scenarios cohort life expectancy is considerably higher than period life expectancy (cf. Fig. 4). For example, in the year 1980, 20 years old individuals could have expected to live until age 91.1 in the baseline scenario and until 90.3 in the constant health share scenario. The static period life expectancy concept employed for Fig. 4 thus underestimates remaining life expectancy for this cohort by more than 14 years. In the baseline scenario, those who are 20 years old in year 2020 expect to die at age 106.2 (whereas period life expectancy is 22.6 years shorter). 65 years old individuals in 1980 expect to live 16.4 additional years in both scenarios (whereas according to period life expectancy it was 15 years). This means that the error made by considering period life expectancy rather than cohort life expectancy is smaller for higher ages, reflecting the fact that the elderly have less time left to benefit from improvements in the quality of health goods and in the access to health care.

The difference in the evolution of life expectancy across scenarios is considerably higher in Fig. 5 compared to Fig. 4. For example, 20 years old individuals in the year 2050 expect to live until age 111 in the baseline scenario and until age 100.6 in the constant health share scenario.[39] The concept of period life expectancy severely underestimates potential gains in life expectancy and losses from limiting health care access. Individuals aged 65 in 2050 expect to live until age 106.1 in the baseline scenario and 3.7 less in the constant health share scenario. Hence, like for period life expectancy, the loss in remaining life expectancy from stabilizing the health expenditure share is lower for older persons.

5. Normative analysis

In this section, we examine the welfare implications of fixing the health expenditure share to its 2020 level rather than staying in the baseline scenario.

5.1. *Expected lifetime utility*

We first need to define an appropriate welfare criterion. Facing uncertain death, rational individuals calculate (under rational expectations) the expected utility from life-time consumption by multiplying instantaneous utility (u) experienced in a given period with the probability to be alive in that period ($S_{v,t}$). Instantaneous utility depends positively on the consumption level of the numeraire and negatively on the health deficit index.

Formally, with maximum life span T, a member of cohort v has preferences that are represented by the intertemporal utility function

$$U_v = \sum_{t=v}^{v+T-1} \beta^{t-v} S_{v,t} u\left(c_{v,t}, d_{v,t}\right), \tag{24}$$

where $\beta \geqslant 0$ is the discount factor and $c_{v,t}$ denotes the consumption level

[36] See Fig. A.2 in the Online-Appendix.

[37] Table A.3 in Online-Appendix (left columns) displays the complete remaining period life expectancies according to age.

[38] Again, see Table A.3 in Online-Appendix (right columns).

[39] For the complete remaining life expectancies according to age, see Table A.4 in Online-Appendix. For comparison, the Office for National Statistics (2015) suggests that a 20 years old woman in the year 2050 can expect to live until age 109.1 in the most optimistic of three considered scenarios (2.1 years longer than a comparable male), but only until age 85.5 in the most pessimistic scenario (2.9 years longer than a comparable male).

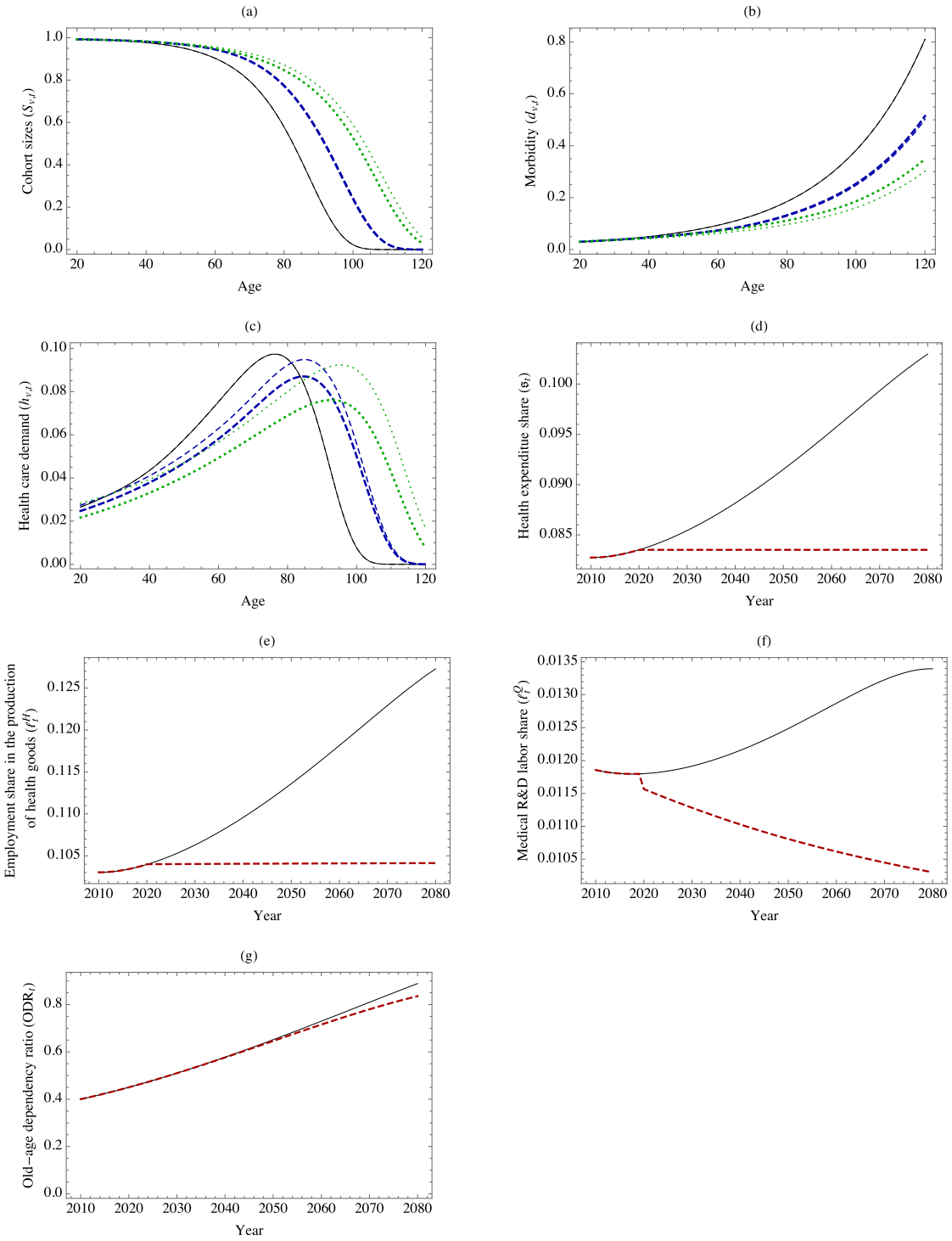

Fig. 3. Effects of limiting health care access from year 2020 onwards to fix the health expenditure share. (Notes: (1) Panels (a)–(c): Solid (black) line for 2020, dashed (blue) lines for 2050, dotted (green) lines for 2080. Thin lines repeat the baseline scenario, thick lines show the constant health share scenario. (2) Panels (d)–(g): Solid (black) lines repeat the baseline scenario, dashed (red) lines show the constant health share scenario. (3) Time paths for $\{\varphi_t\}$ in the alternative scenario as displayed in Fig. A.2 (Online-Appendix). (4) Other parameters as for Fig. 1). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

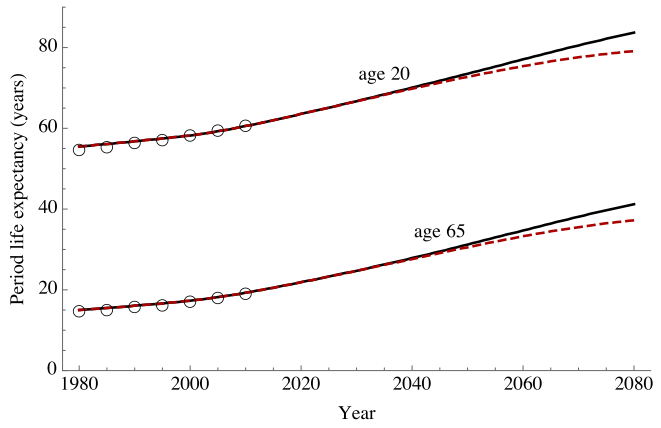

Fig. 4. Implied remaining period life expectancies at age 20 and age 65: baseline vs. constant health share scenario. (Notes: (1) Solid (black) lines for the baseline scenario, dashed (red) lines for the constant health share scenario, circles according to UK data. (2) Data source: www.mortality.org. (3) Parameters as for Fig. 2 (baseline scenario) and Fig. 3 (constant health share scenario)). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

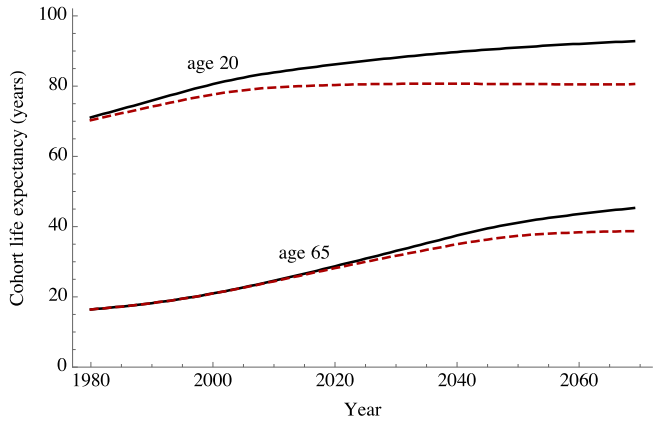

Fig. 5. Implied remaining cohort life expectancies at age 20 and age 65: baseline vs. constant health share scenario. (Notes: (1) Solid (black) lines for the baseline scenario, dashed (red) lines for constant health share scenario. (2) Parameters as for Fig. 2 (baseline scenario) and Fig. 3 (constant health share scenario)). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

in t. Instantaneous utility is specified as

$$u\left(c_{v,t}, d_{v,t}\right) \equiv \frac{\log c_{v,t}}{\left(1+d_{v,t}\right)^{\zeta}} + \overline{u}, \tag{25}$$

where $\zeta > 0$ measures to what extent a higher deficit state reduces the marginal utility of consumption and $\overline{u} \geqslant 0$ is used to obtain a reasonable expected value of life in the calibrated model.[40] For an individual without health deficits ($d_{v,t} = 0$) or in the case where $\zeta = 0$, we are back to a standard instantaneous utility function.[41]

We assume that the health care system is financed by a constant contribution rate out of wage income, denoted by τ_t for period t. The health care budget is balanced at each point in time; that is, revenue, $\tau_t w_t L_t$, equals expenses, $p_t H_t$. Consequently, recalling (17), the health contribution rate equals the markup factor for health goods (Γ) times the share of labor (ℓ^H) allocated for producing health goods and services:

$$\tau_t = \Gamma \ell_t^H. \tag{26}$$

Let asset holding ("wealth") of a member of cohort v in t be denoted by $a_{v,t}$. Initial asset holding equals zero, $a_{v,v} = 0$, since there is no bequest motive and the annuity market is perfect. We assume fair insurance within a cohort on the annuity market, which means that zero-profit insurance companies pay a rate of return above $\overline{r}$ and keep the wealth of the deceased. The corresponding law of motion for individual wealth for a member of cohort v can be written as[42]

$$a_{v,t+1} = \left(1-\tau_t\right) w_t + \left(1+r_{v,t}\right) a_{v,t} - c_{v,t}, \tag{27}$$

$t \geqslant v$, where the cohort-specific interest factor between date t and $t+1$ is given by

$$1 + r_{v,t} = \frac{1+\overline{r}}{1-m_{v,t-1}}. \tag{28}$$

Individuals of each generation v choose their consumption paths $\{c_{v,t}\}_{t \geqslant v}$ to maximize utility U_v s.t. (27) and the non-negativity constraint $a_{v,v+T} \geqslant 0$. Individuals take into account the future health contribution rate and health deficit states (including implied mortality risks in (1)) that result from the baseline health care wedge as long as there is no policy switch. When the constant health share scenario is introduced in period t_0 (i.e. year 2020), living members of generations $v < t_0$ (i.e. those already born) re-optimize by taking into account the new policy regime from t_0 onwards. The optimization problems of consumers without and with a policy switch are solved in Appendix C.

We report cohort-specific welfare losses $1 - \psi_v$ of switching from the baseline scenario to the constant health share scenario, where ψ_v is the factor by which consumption levels of the baseline scenario are multiplied such that cohort v experiences the same utility as in the constant health share scenario (equivalent variation).[43]

5.2. Calibration

We choose a typical value for the subjective discount rate, $\beta = 0.98$, such that $\beta(1+\overline{r}) > 1$.[44] Initial labor efficiency level A_0 (in the year 1850) is normalized to unity.[45] Next, we calibrate ζ, which determines the loss in marginal utility from consumption caused by health deficits. Finkelstein et al. (2013) find that, starting at the mean, a one-standard deviation increase of chronic diseases is associated with a decline in the marginal utility of consumption, denoted by *LOSS*, of 11.2 percent.

[40] A positive constant $\overline{u}$ can also be used to ensure that instantaneous utility is non-negative. Otherwise, individuals could prefer to live shorter for given consumption levels, see Jones (2016).

[41] Log-utility in consumption implies that the intertemporal elasticity of substitution is unity, as supported by Chetty (2006).

[42] For simplicity, we do not consider the possibility of "out-of-pocket" health payments or coinsurance. The NHS does not demand copayments. Moreover, many important health goods, e.g. treatment for orthopedic deficits, cancer medication, antiviral drugs etc., are unaffordable for most individuals if not covered by NHS. The fraction of population with private health insurance has been stable over time at a moderate level (11 and 10.5 percent in the year 2000 and 2016, respectively; see OECD, 2016). Also health expenditures from voluntary schemes and out-of-pocket as fraction of total UK health expenditure has shown no upward trend since 2000 and was at a moderate level of 20.5 percent in 2016 (consisting of 6.4 percent for curative and rehabilitative care, 6.2 percent for long term health care, 5.5 percent for medical goods, 1.3 percent for preventive care, and 1.1 percent for other purposes), according to https://stats.oecd.org, retrieved December 23, 2018.

[43] See Jones and Klenow (2016) for a similar way to measure welfare differences of randomly chosen individuals in a cross-country context rather than across policy regimes.

[44] Recall that $\overline{r} = 0.05$. If we assumed $\beta(1+\overline{r}) = 1$, then the complementarity of consumption and health in utility would imply that consumption monotonically declines with age, which is inconsistent with the evidence.

[45] A_0 does not enter the dynamical system for the positive analysis (Appendix A).

Marginal consumption utility reads as $(1+d_{v,t})^{-\zeta}/c_{v,t}$. Evaluated at the mean deficit index, $\mathbb{E}(d)$, and denoting the standard deviation by $\mathscr{STD}(d)$, the estimate of Finkelstein et al. (2013) then suggests that $\zeta$is given by

$$\frac{[1+\mathbb{E}(d)+\mathscr{STD}(d)]^{-\zeta}}{[1+\mathbb{E}(d)]^{-\zeta}}=1-LOSS. \tag{29}$$

According to Mitnitski et al. (2002a,b), the mean deficit index in the population is $\mathbb{E}(d)=0.054$ and the standard deviation is $\mathscr{STD}(d)=0.024$. Hence, $\zeta=-44.42\cdot\log(1-0.112)=5.1$.

Denote the expected value of life, VoL (sometimes called the value of a statistical life), of an individual born in v by W_v and assume it is given by expected (indirect) life-time utility in the baseline policy scenario, normalized by the marginal instantaneous (indirect) utility in the initial period of life:

$$W_v\equiv\frac{U_v}{\frac{\partial u\left(c_{v,v},d_{\min}\right)}{\partial c}}=U_v(1+d_{\min})^{\zeta}c_{v,v}. \tag{30}$$

We calibrate the constant $\overline{u}$ such that the expected value of life (VoL) of the cohort starting out in year 2010 has a plausible value according to empirical studies based on "wage differences on jobs with varying probabilities of accidental death or from market prices for products that reduce the likelihood of fatal injury" (Murphy and Topel, 2006; p. 884; see also Hall and Jones, 2007). Given that GDP per person employed, $y\equiv GDP/L$, in the UK was about 75,000 US$ (PPP) in 2010, we alternatively set $\overline{u}=0.34,1.32$ and 2.31 to match $W_v/y_v=60,80$ and 100 in 2010, corresponding to a VoL of 4.5, 6 and 7.5 million US$, respectively.

5.3. Results

Fig. 6 displays the cohort-specific welfare losses $(1-\psi_v)$ of switching from the baseline scenario (analyzed in Fig. 2) to the constant health share scenario (Fig. 3). We see that stabilizing the health expenditure share to its 2020 level is almost welfare-neutral for older cohorts. On the one hand, individuals close to retirement age at the time of the reform do not save much health care contributions (assumed to be entirely paid by workers). On the other hand, the detrimental effects from the policy switch on longevity and morbidity are small for elderly individuals because for them slower future medical progress is of little importance.

For later cohorts, however, the welfare loss is substantial. This is a remarkable result since younger cohorts save health contributions over a long working period. Those who start working life after the reform year 2020 benefit from reduced contributions for the entire working life, whereas reductions in survival rates in response to the policy switch are minor for working-aged individuals. However, reduced survival rates during retirement and reduced instantaneous utility from higher health deficits by far outweigh the utility increases from higher disposable income for younger generations. We estimate that 20 years old individuals in 2020 experience a welfare loss from the policy switch of 15.7, 18.6 and 21.4 percent when calibrating $\overline{u}$ to correspond to a VoL of 4.5, 6 and 7.5 million US$ for the cohort aged 20 in the year 2010, respectively. Welfare losses are even higher for future generations. Individuals aged 20 in 2050 experience a welfare loss of 33.4, 37.3 and 41.1 percent for the three alternative benchmark VoLs considered. These drastic welfare losses from the policy switch reflect the considerable losses in cohort life expectancy (displayed in Fig. 5) as well as increased morbidity (displayed in panel (c) of Fig. 3).

6. Conclusion

This paper attempts to predict future health expenditure, longevity and morbidity by employing a multi-period overlapping generations model with an age-structured population. In order to facilitate a calibration of the model and to derive quantitative implications, we employed the concept of health deficits as a simple and observable measure of health status that has proven to be a powerful determinant of mortality. The gerontologically founded health deficit model exhibits positive path dependence and implies that improperly treated health deficits lead to new ones and (considerably) reduces life expectancy. The health deficit approach is thus particularly appropriate to address the implications of changes in health care utilization for the future evolution of morbidity and longevity. The calibrated model is consistent, *inter alia, with observed health expenditures, morbidity and longevity, notably including existing inefficiencies in the health system. Most importantly, we capture that age-specific health deficit accumulation and mortality is driven by the interaction between endogenous medical R&D and health care access.*

In the baseline scenario with slightly improving health care access over time, our calibrated model suggests that the health expenditure share in GDP will moderately rise along with substantial increases in human longevity and significant reductions in morbidity especially for higher ages in the more distant future. The main reason is that good health care access maintains R&D incentives that lead to medical advances.

We also consider the effects of stabilizing the health expenditure share to its 2020 level by limiting health care utilization. Our analysis suggests that such policy switch has sizable negative effects on morbidity and longevity, particularly in the long run. Generally, and perhaps surprisingly, young individuals (i.e. those who save the most health care contributions from the policy switch) are predicted to suffer the greatest losses in terms of life expectancy and welfare. Whereas short-run effects can mainly be attributed to the direct effects of reduced health care utilization on the accumulation of health deficits, long-run implications mainly work through reduced medical R&D incentives.

CRediT authorship contribution statement

Sebastian Böhm: Software, Formal analysis, Visualization, Investigation. **Volker Grossmann:** Conceptualization, Methodology, Formal analysis, Funding acquisition, Visualization, Writing - original draft, Writing - review & editing. **Holger Strulik:** Conceptualization, Methodology, Funding acquisition, Visualization, Writing - original draft, Writing - review & editing.

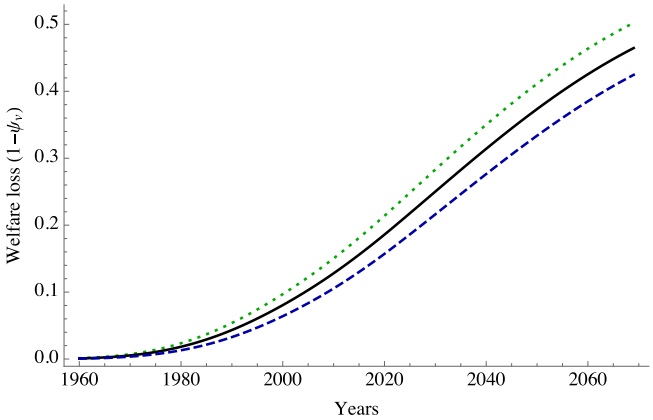

Fig. 6. Cohort-specific welfare losses of fixing the health expenditure share to the 2020 level for three alternative labor efficiency levels. (Notes: (1) The displayed value for year t corresponds to the welfare loss of the policy switch for someone who is 20 years old in year t. (2) Dashed (blue) line: $\overline{u}=0.34$, solid (black) line: $\overline{u}=1.32$, dotted (green) line: $\overline{u}=2.31$. (3) $\beta=0.98,\zeta=5.1$. (4) Time paths $\{\varphi_t\}$ in the baseline scenario and the constant health share scenario are displayed in Fig. A.2 (Online-Appendix). (5) Other parameters as for Fig. 1). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

We are grateful to an anonymous reviewer, Mark Dusheiko, Chad Jones, and seminar participants at the ifo Institute in Munich, University of Bielefeld, Leipzig University, University of Fribourg, the Munich Center for the Economics of Aging (MEA), the Annual Congress of the International Institute of Public Finance (IIPF) in Tokyo, the Econometric Society European Meeting (ESEM) in Lisbon, the Annual Congress of the Association for Public Economic Theory (PET) in Paris, the Annual Congress of the German Economic Association in Vienna, the Swiss Health Economics Workshop in Lausanne, and the CESifo Area Conference on Public Sector Economics in Munich for valuable comments and suggestions. Financial support of the Swiss National Fund (SNF) and Deutsche Forschungsgemeinschaft (DFG) through the co-financed project "Optimal Health and Retirement Policy under Biologically Founded Human Ageing" (SNF Grant No. 100018L 150096) is gratefully acknowledged.

Appendix A. Dynamical System

According to (19), R&D firms do not earn profits in equilibrium and health good producers are identical in all sectors, i.e., $l_t(j) = L_t^Q$, $\pi_t(j) = \pi_t$ and $V_{t+1}(j) = V_{t+1}$ for all $j \in [0,1]$. Using $\widetilde{\xi}_t = \xi \cdot (L_t^Q)^{-\vartheta}$ in (19), the zero-profit condition for R&D firms reads as

$$\xi \left(L_t^Q\right)^{-\vartheta} V_{t+1} = w_t. \tag{31}$$

According to (18),

$$\begin{aligned} V_t \;&= \pi_t + \frac{1-\mu_{t+1}}{1+\overline{r}}\pi_{t+1} + \frac{(1-\mu_{t+1})(1-\mu_{t+2})}{(1+\overline{r})^2}\pi_{t+2} + \\ &\quad \frac{(1-\mu_{t+1})(1-\mu_{t+2})(1-\mu_{t+3})}{(1+\overline{r})^3}\pi_{t+3} + \ldots, \end{aligned} \tag{32}$$

$$V_{t+1} = \pi_{t+1} + \frac{1-\mu_{t+2}}{1+\overline{r}}\pi_{t+2} + \frac{(1-\mu_{t+2})(1-\mu_{t+3})}{(1+\overline{r})^2}\pi_{t+3} + \ldots = \frac{1+\overline{r}}{1-\mu_{t+1}}(V_t - \pi_t). \tag{33}$$

Using (20) in (33), we get the following no-arbitrage condition in the market that finances health R&D:

$$\frac{1-\mu_{t+1}}{1+\overline{r}}\frac{V_{t+1}}{V_t} + \frac{(\Gamma-1)w_t\chi H_t}{V_t} = 1. \tag{34}$$

Now let us define $\mathscr{V}_t \equiv V_t/A_t$. Observing $A_{t+1}/A_t = 1+g$, we thus have $V_{t+1}/V_t = (1+g)\,\mathscr{V}_{t+1}/\mathscr{V}_t$. Also recall $w_t/A_t = \omega$. Denote by $\mathfrak{d}_{\mathfrak{a},t}$ the health deficit index of a surviving individual of age $\mathfrak{a}$ in period t and $\widetilde{\mathfrak{a}}_t$ as the highest age in period t such that $\mathfrak{d}_{\mathfrak{a},t} \leqslant d_{\max}$. Thus, $\overline{\mathfrak{a}}_t \equiv \min(\widetilde{\mathfrak{a}}_t, T)$ is the age at which an individual dies for sure. Neglecting the household side (which is relevant for the welfare analysis only), the dynamical system can be summarized as follows:

$$\mathfrak{d}_{1,t+1} = [1 + \varrho - (1-\varphi)\kappa Q_t]d_{\min}, \tag{35}$$

$$\mathfrak{d}_{2,t+1} = [1 + \varrho - (1-\varphi)\kappa Q_t]\mathfrak{d}_{1,t}, \tag{36}$$

$$\mathfrak{d}_{3,t+1} = [1 + \varrho - (1-\varphi)\kappa Q_t]\mathfrak{d}_{2,t}, \tag{37}$$

$$\vdots$$

$$\mu_{t+1} = \eta Q_t + \left(1 - \eta Q_t\right)\cdot\xi\cdot\left(L_t^Q\right)^{1-\vartheta}, \tag{38}$$

$$Q_{t+1} - Q_t = \gamma(1-\eta Q_t)\xi\left(L_t^Q\right)^{1-\vartheta} - \left(\delta^Q - \gamma\eta\right)Q_t, \tag{39}$$

$$\frac{1-\mu_{t+1}}{1+\overline{r}}\mathscr{V}_{t+1}(1+g) + \left(\Gamma - 1\right)\omega_t\chi H_t = \mathscr{V}_t, \tag{40}$$

$$\mathscr{V}_{t+1}(1+g)\xi\cdot\left(L_t^Q\right)^{-\vartheta} = \omega, \tag{41}$$

$$\begin{aligned} H_t \;&= \left(1-\varphi_t\right)S_{t,t}d_{\min} + \left(1-\varphi_t\right)\left(1-\widetilde{m}(d_{\min})\right)\times \\ &\quad \left\{S_{t-1,t-1}\mathfrak{d}_{1,t} + \mathfrak{d}_{2,t}S_{t-2,t-2}\left(1-\widetilde{m}\left(\mathfrak{d}_{1,t-1}\right)\right) + \right. \\ &\quad \mathfrak{d}_{3,t}S_{t-3,t-3}\left(1-\widetilde{m}\left(\mathfrak{d}_{2,t-1}\right)\right)\left(1-\widetilde{m}\left(\mathfrak{d}_{1,t-2}\right)\right) + \ldots + \\ &\quad \left.\mathfrak{d}_{\overline{\mathfrak{a}}_t,t}S_{t-\overline{\mathfrak{a}}_t,t-\overline{\mathfrak{a}}_t}\left(1-\widetilde{m}\left(\mathfrak{d}_{\overline{\mathfrak{a}}_t-1,t-1}\right)\right)\left(1-\widetilde{m}\left(\mathfrak{d}_{\overline{\mathfrak{a}}_t-2,t-2}\right)\right)\times\ldots\times\left(1-\widetilde{m}\left(\mathfrak{d}_{1,t-\overline{\mathfrak{a}}_t+1}\right)\right)\right\}, \end{aligned} \tag{42}$$

$$L_t^Y + \chi H_t + L_t^Q = L_t, \tag{43}$$

according to (7), (14), (16), (34), (31), (9), (21), respectively, where we used $L_t^H = \chi H_t$ for the latter. Initial quality index $Q_0 > 0$ and the vector of current deficit states of the cohorts living in period 0, $\boldsymbol{d}_0 \equiv (\mathfrak{d}_{1,0}, \mathfrak{d}_{2,0}, \mathfrak{d}_{3,0}, \ldots, \mathfrak{d}_{\overline{\alpha}_0,0})$, are given.[46]

Appendix B. Long run equilibrium analysis

A steady state analysis is instructive to understand the relationship between endogenous observables. It has also helped us to calibrate the model, as discussed below.

First, setting $Q_{t+1} = Q_t$ in (10) and omitting the time index implies

$$\mu = \frac{\delta^Q}{\gamma} Q. \tag{44}$$

Thus, in the long run, the total innovation probability $\mu$is proportional to the medical knowledge stock, Q. Second, according to (13) and (38),

$$\frac{\left(L^Q\right)^{\vartheta}}{\xi} = \frac{(1-\overline{\mu})L^Q}{\mu - \overline{\mu}}. \tag{45}$$

Using $\mathscr{V}_{t+1} = \mathscr{V}_t = \mathscr{V}$ and $L^H = \chi H$ in (40) implies

$$\mathscr{V} = \frac{(\Gamma - 1)(1+\overline{r})\omega L^H}{\overline{r} - g + \mu(1+g)}. \tag{46}$$

Moreover, according to (41),

$$\mathscr{V} = \frac{\omega\left(L^Q\right)^{\vartheta}}{(1+g)\xi}. \tag{47}$$

Combining (46) and (47) implies

$$\frac{\left(L^Q\right)^{\vartheta}}{\xi} = \frac{(\Gamma - 1)(1+\overline{r})L^H}{\frac{\overline{r}-g}{1+g} + \mu}. \tag{48}$$

Combining (45) with (48) and using (44) implies that

$$\ell^Q = \frac{\frac{\delta^Q}{\gamma\eta} - 1}{\frac{1}{\overline{\mu}} - 1} \frac{(\Gamma - 1)(1+\overline{r})\ell^H}{\frac{\overline{r}-g}{1+g} + \mu} \tag{49}$$

holds in the long run (recall that $\delta^Q > \gamma\eta$). Third, according to (22), the health expenditure share can be written as

$$\mathfrak{s} = \frac{pH}{Y + pH} = \frac{1}{\frac{Y}{pH} + 1} = \frac{1}{\frac{L^Y}{\Gamma\omega L^H}\left(\frac{K^Y}{AL^Y}\right)^{\alpha} + 1} = \frac{1}{\frac{\ell^Y}{(1-\alpha)\Gamma\ell^H} + 1}, \tag{50}$$

where we used (3) and (17) for the third equation and $\omega = (1-\alpha)(AL^Y/K^Y)^{-\alpha}$ for the final one.

We do neither have good data for the UK employment share of medical R&D workers (ℓ^Q) nor for the health good price markup factor (Γ). Fortunately, however, (49) and (50) show that given the observable employment share in health goods production (ℓ^H), the total innovation probability (μ) and the unintentional innovation probability ($\overline{\mu}$), the health R&D productivity parameter ξ does neither affect (long run) levels of the employment share of medical R&D workers, ℓ^Q, nor the health expenditure share, $\mathfrak{s}$. This points to the possibility that many combinations of $\Gamma$and ξ allow us to match observables (i)–(iv). Importantly, we confirmed that our results are not sensitive to changing the calibration as long as it matches the data. We assume a plausible markup factor $\Gamma = 1.25$ that along with the other parameters matches observables (i)–(iv) and is associated with a reasonable value $\ell_t^Q = 0.012$ for the employment share of medical R&D workers in the year 2010 (widely interpreted to include managers and professionals organizing R&D in addition to medical scientists and engineers).

Appendix C. Consumption paths (Normative analysis)

We first derive the consumption paths before the policy switch to the constant health share scenario is introduced in period t_0. Recall that before the policy switch, an individual born in v takes as given the paths of the health deficit state, $\{d_{v,t}\}_{t=v}^{v+T-1}$ (including implied mortality risks, $m_{v,t} = \widetilde{m}(d_{v,t})$) and the health contribution rate, $\{\tau_t\}_{t=v}^{v+R-1}$, that result from the baseline scenario 0 (i.e. they do not anticipate the policy switch). Those who currently alive in period t_0 re-optimize in t_0, which we consider afterwards. Those born in $v \geqslant t_0$ always live in the new policy regime and the analysis is analogous to that without policy switch, i.e. without reoptimization.

- **Before reoptimization:** We start with the case before reoptimization, which is also the case without policy switch. Using $S_{v,t} = S_{v,v}\prod_{u=v}^{t-1}(1-m_{v,u})$ in (24), the Lagrangian $\mathscr{L}_v$ associated with maximizing U_v subject to (27) and $a_{v,v+T} \geqslant 0$ is

[46] Assuming that a health system has not existed initially (like in our calibrated model with 1850 as the initial year), such that $\varphi_0 = 1$, we have $\mathfrak{d}_{\mathfrak{a},0} = d_{\min}(1+\varrho)^{\mathfrak{a}}$ for all $\mathfrak{a} \in [0, \overline{\alpha}_0]$, where $\overline{\alpha}_0$ is the maximum age in period 0.

$$\begin{aligned}\mathscr{L}_v &= \ldots + \beta^{t-v} S_{v,v} \prod_{u=v}^{t-1}\left(1-m_{v,u}\right)\frac{\log c_{v,t}}{\left(1+d_{v,t}\right)^{\zeta}} + \\ &\beta^{t+1-v} S_{v,v} \prod_{u=v}^{t}\left(1-m_{v,u}\right)\frac{\log c_{v,t+1}}{\left(1+d_{v,t+1}\right)^{\zeta}} + \ldots + \\ &\lambda_{v,t}\left[\left(1-\tau_t\right)w_t + \left(1+r_{v,t}\right)a_{v,t} - c_{v,t} - a_{v,t+1}\right] + \\ &\lambda_{v,t+1}\left[\left(1-\tau_{t+1}\right)w_{t+1} + \left(1+r_{v,t+1}\right)a_{v,t+1} - c_{v,t+1} - a_{v,t+2}\right] + \ldots\end{aligned} \tag{51}$$

where $\lambda_{v,t}$, $\lambda_{v,t+1}$, etc. denote the multipliers for period $t, t+1$, etc. The first-order conditions $\partial\mathscr{L}_v/\partial c_{v,t} = \partial\mathscr{L}_v/\partial c_{v,t+1} = \partial\mathscr{L}_v/\partial a_{v,t+1} = 0$ can be written as

$$\frac{\beta^{t-v} S_{v,v} \prod_{u=v}^{t-1}\left(1-m_{v,u}\right)}{\left(1+d_{v,t}\right)^{\zeta} c_{v,t}} = \lambda_{v,t}, \tag{52}$$

$$\frac{\beta^{t+1-v} S_{v,v} \prod_{u=v}^{t}\left(1-m_{v,u}\right)}{\left(1+d_{v,t+1}\right)^{\zeta} c_{v,t+1}} = \lambda_{v,t+1}, \tag{53}$$

$$\lambda_{v,t} = \lambda_{v,t+1}\left(1+r_{v,t+1}\right). \tag{54}$$

Combining (52)–(54) leads to

$$\frac{\left(1+d_{v,t+1}\right)^{\zeta} c_{v,t+1}}{\left(1+d_{v,t}\right)^{\zeta} c_{v,t}} = \beta\left(1-m_{v,t}\right)\left(1+r_{v,t+1}\right). \tag{55}$$

Using (28) in (55) implies

$$c_{v,t+1} = \left(\frac{1+d_{v,t}}{1+d_{v,t+1}}\right)^{\zeta}\beta\left(1+\bar{r}\right)c_{v,t}. \tag{56}$$

Iterating and using $d_{v,v} = d_{\min}$, we obtain

$$c_{v,t} = \left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^{\zeta}\beta^{t-v}(1+\bar{r})^{t-v}c_{v,v}. \tag{57}$$

From (27), $a_{v,v} = 0$ and $a_{v,v+T} = 0$ (reflecting that it is optimal not to hold wealth after certain death), we find that the intertemporal budget constraint of a member of cohort v is given by

$$c_{v,v} + \sum_{t=v+1}^{v+T-1}\left(\frac{c_{v,t}}{\prod_{u=v+1}^{t}\left(1+r_{v,u}\right)}\right) = \left(1-\tau_v\right)w_v + \sum_{t=v+1}^{v+R-1}\left(\frac{\left(1-\tau_t\right)w_t}{\prod_{u=v+1}^{t}\left(1+r_{v,u}\right)}\right). \tag{58}$$

Using (28) and (57), we obtain for the left-hand side of (58) that

$$c_{v,v} + \sum_{t=v+1}^{v+T-1}\left(\frac{c_{v,t}}{\prod_{u=v+1}^{t}\left(1+r_{v,u}\right)}\right) = c_{v,v}\left(1 + \sum_{t=v+1}^{v+T-1}\beta^{t-v}\left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^{\zeta}\prod_{u=v}^{t-1}\left(1-m_{v,u}\right)\right). \tag{59}$$

Equating the right-hand sides of (58) and (59), and using (2), (28), $w_t = \omega A_t$ and $A_t = A_v(1+g)^{t-v}$, implies that the initial consumption level, $c_{v,v}$, is given by

$$c_{v,v} = \omega A_v \frac{\sum_{t=v}^{v+R-1}\left(1-\tau_t\right)\left(\frac{1+g}{1+\bar{r}}\right)^{t-v}\frac{S_{v,t}}{S_{v,v}}}{\sum_{t=v}^{v+T-1}\beta^{t-v}\left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^{\zeta}\frac{S_{v,t}}{S_{v,v}}}. \tag{60}$$

For the welfare analysis, for each cohort we feed in the consumption path (57) with initial level (60).

- **With reoptimization in period when the policy regime switches:** We now turn to the case where currently living individuals experience the switch to the constant health share scenario in period t_0. Since the policy switch is not anticipated, for $t < t_0$, individuals follow the same consumption path $\{c_{v,t}\}_{t=v}^{t_0-1}$ as computed in the previous case and re-optimize in t_0. According to (56), knowing c_{v,t_0}, the path of consumption of any living member of generation v for future dates $t \geqslant t_0$ evolves as

$$c_{v,t} = \left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^{\zeta}\beta^{t-t_0}(1+\bar{r})^{t-t_0}c_{v,t_0}. \tag{61}$$

We thus need to derive c_{v,t_0}. For this, we need to know the individual wealth level of someone born in v that prevails in t_0, i.e. we need to know a_{v,t_0}. We distinguish the case when the individual is still in working age and when already retired at the time where the policy shock occurs, $t_0 < v+R$ and $t_0 \geqslant v+R$, respectively.

– Case $t_0 < v+R$: Using (27) and $a_{v,v} = 0$, for $t_0 < v+R$ we have

$$\frac{a_{v,t_0}}{\prod_{u=v+1}^{t_0-1}(1+r_{v,u})} = (1-\tau_v)w_v - c_{v,v} + \sum_{t=v+1}^{t_0-1} \frac{(1-\tau_t)w_t - c_{v,t}}{\prod_{u=v+1}^{t}(1+r_{v,u})} \tag{62}$$

Using (2), (28) and (57), we obtain

$$c_{v,v} + \sum_{t=v+1}^{t_0-1}\left(\frac{c_{v,t}}{\prod_{u=v+1}^{t}(1+r_{v,u})}\right) = c_{v,v}\sum_{t=v}^{t_0-1}\beta^{t-v}\left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^{\zeta}\frac{S_{v,t}}{S_{v,v}}. \tag{63}$$

Using (28) and (2), we also get

$$\prod_{u=v+1}^{t}(1+r_{v,u}) = \frac{S_{v,v}(1+\bar{r})^{t-v}}{S_{v,t}}, \text{ i.e. } \prod_{u=v+1}^{t_0-1}(1+r_{v,u}) = \frac{S_{v,v}}{(1+\bar{r})^{v+1-t_0}S_{v,t_0-1}}. \tag{64}$$

Substituting (63), (64), $w_t = \omega A_t$ and $A_t = A_v(1+g)^{t-v}$ into (62), the wealth holding of a member of generation v in $t_0 < v+R$ is given by

$$a_{v,t_0} = \frac{A_v\omega\left(\sum_{t=v}^{t_0-1}(1-\tau_t)\left(\frac{1+g}{1+\bar{r}}\right)^{t-v}\frac{S_{v,t}}{S_{v,t_0-1}}\right) - c_{v,v}\sum_{t=v}^{t_0-1}\left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^{\zeta}\beta^{t-v}\frac{S_{v,t}}{S_{v,t_0-1}}}{(1+\bar{r})^{v+1-t_0}}. \tag{65}$$

Next, use (27) and $a_{v,v+T} = 0$ to obtain

$$c_{v,t_0} + \sum_{t=t_0+1}^{v+T-1}\frac{c_{v,t}}{\prod_{u=t_0+1}^{t}(1+r_{v,u})} = \left(1+r_{v,t_0}\right)a_{v,t_0} + (1-\tau_{t_0})w_{t_0} + \sum_{t=t_0+1}^{v+R-1}\frac{(1-\tau_t)w_t}{\prod_{u=t_0+1}^{t}(1+r_{v,u})}. \tag{66}$$

Using (61) and

$$\prod_{u=t_0+1}^{t}(1+r_{v,u}) = \frac{(1+\bar{r})^{t-t_0}}{\prod_{u=t_0}^{t-1}\left(1-m_{u,t}\right)} = (1+\bar{r})^{t-t_0}\frac{S_{v,t_0}}{S_{v,t}}, \tag{67}$$

according to (28) and (2), implies

$$c_{v,t_0} + \sum_{t=t_0+1}^{v+T-1}\frac{c_{v,t}}{\prod_{u=t_0+1}^{t}(1+r_{v,u})} = c_{v,t_0}\sum_{t=t_0}^{v+T-1}\left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^{\zeta}\rho^{t-t_0}\frac{S_{v,t}}{S_{v,t_0}}. \tag{68}$$

Equating the right-hand sides of (66) and (68) and using (28), (67), $w_t = \omega A_t$ and $A_t = A_{t_0}(1+g)^{t-t_0}$ implies, for $t_0 < v+R$, the consumption level:

$$c_{v,t_0} = \frac{\frac{1+\bar{r}}{1-m_{v,t_0-1}}a_{v,t_0} + \omega A_{t_0}\sum_{t=t_0}^{v+R-1}\frac{1-\tau_t}{(1+\bar{r})^{t-t_0}}\left(\frac{1+g}{1+\bar{r}}\right)^{t-t_0}\frac{S_{v,t}}{S_{v,t_0}}}{\sum_{t=t_0}^{v+T-1}\left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^{\zeta}\rho^{t-t_0}\frac{S_{v,t}}{S_{v,t_0}}} \tag{69}$$

with a_{v,t_0} given by (65) and $A_{t_0} = A_v(1+g)^{t_0-v}$.

– Case $t_0 \geqslant v+R$: Analogously to (65) and (69), for $t_0 \geqslant v+R$ (i.e. the policy switch occurs after retirement), we have

$$a_{v,t_0} = \frac{A_v\omega\sum_{t=v}^{v+R-1}(1-\tau_t)\left(\frac{1+g}{1+\bar{r}}\right)^{t-v}\frac{S_{v,t}}{S_{v,t_0-1}} - c_{v,v}\sum_{t=v}^{t_0-1}\left(\frac{1+d_{\min}}{1+d_{v,t}}\right)^{\zeta}\beta^{t-v}\frac{S_{v,t}}{S_{v,t_0-1}}}{(1+\bar{r})^{v+1-t_0}}, \tag{70}$$

$$c_{v,t_0} = \frac{\frac{1+\bar{r}}{1-m_{v,t_0-1}}a_{v,t_0}}{\sum_{t=t_0}^{v+T-1}\left(\frac{1+d_{v,t_0}}{1+d_{v,t}}\right)^{\zeta}\beta^{t-t_0}\frac{S_{v,t}}{S_{v,t_0}}} \tag{71}$$

with a_{v,t_0} given by (70).

Appendix D. Supplementary data

Supplementary data associated with this article can be found, in the online version, athttps://doi.org/10.1016/j.jeoa.2020.100286.

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