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

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**R&D-DRIVEN MEDICAL PROGRESS,  
HEALTH CARE COSTS, AND THE  
FUTURE OF HUMAN LONGEVITY**

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# R&D-Driven Medical Progress, Health Care Costs, and the Future of Human Longevity\*

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## Abstract

In this paper we set up an overlapping generations model of gerontological founded human aging that takes the interaction between R&D-driven medical progress and access to health care into account. We use the model to explore potential futures of human health and longevity. 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. Freezing the expenditure share at the 2020 level by rationing access to health care severely reduces potential gains in health, longevity and welfare. These losses are greatest in the long run due to reduced incentives for medical R&D. For example, rationing is predicted to reduce potential gains of life-expectancy at age 65 by about 4 years in the year 2050. Generally, and perhaps surprisingly, young individuals (i.e. those who save the most health care contributions through rationing) are predicted to suffer the greatest losses in terms of life expectancy and welfare.

**Key words:** Longevity; Medical R&D; Morbidity; Health Care; Rationing.

**JEL classification:** H50; I10; C60.

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# 1 Introduction

A salient feature of structural economic development over the last decades is the secular expansion of the health sector and human longevity. In the U.S., health expenditure per capita grew by on average 4.1% annually since 1970 to a level of about 18% of GDP four decades later (Chernew and Newhouse, 2011; Gaynor et al., 2015). Starting at lower levels, other developed countries experienced similar rates of increase of the health sector such that, across the board, health expenditure increased faster than GDP.

Scholars agree that both the rise of health expenditure and improvements in longevity are related to medical technological progress.<sup>1</sup> Recent examples of health innovations include computerized diagnostic tests (e.g. for medical imaging), personalized cancer therapy, and new treatments of virus infections like HIV or Hepatitis C. More generally, Lichtenberg (2007) shows that later vintages of pharmaceuticals are more powerful in the reduction of health deficits. Considering the evolution of 92 potentially lethal diseases he finds that conditions experiencing greater pharmaceutical innovation tend to have greater declines in mortality rates.<sup>2</sup>

Consistent with such evidence, this paper proposes a new approach to study the interdependence of medical R&D, health expenditure, longevity and the health status of an age-structured population. We develop a multi-period overlapping generations model where endogenous medical progress affects morbidity in interaction with access to health goods. In turn, morbidity determines mortality rates.

We employ the *health deficit index* developed by gerontologists (Mitnitski et al., 2002a,

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<sup>1</sup>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, Finkelstein and Notowidigdo, 2013; Baltagi et al., 2016 ).

<sup>2</sup>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 Economist (2016) provides an overview on recent developments in anti-aging research.

2002b, 2005, 2007) and subsequently used in countless empirical studies in the natural sciences for measuring health status and its relation to mortality in a biologically founded way. The approach has two advantages. First, it enables us to calibrate our model. In contrast to health capital (a latent variable popular among economists; Grossman, 1972), health deficits are observed and easily quantifiable. In line with the conceptualization of morbidity and physiological aging in gerontology research, individuals in our model accumulate health deficits, which determine the probability to die at a given age. The individual accumulation process of health deficits endogenously depends on the interaction of health care access (the extent to which individuals are provided with health goods to treat their illnesses) and the available quality of health goods. The quality of health goods is endogenously determined by vertical R&D. Second, the approach enables us to understand the potential effects of changes in the access to health care by putting the empirically established path-dependency of health deficits at the center of the analysis. It implies that improperly treated health deficits lead to new ones (with exponential growth of health deficits) that overall may considerably shorten life-time. By contrast, the health capital model by Grossman (1972) would imply that health capital depreciates faster when the health capital stock is high, which is clearly at odds with the data on health deficit accumulation. The health capital model would thus, by design, underestimate the effects of health care rationing.

We apply the approach to make inferences about the future development of life expectancy and health expenditure, conditional on the extent of future access to health care. Consistent with the established importance of medical progress for health costs and human longevity, our calibrated model suggests substantial future gains in life expectancy that are associated with significant declines in morbidity. Holding the mark-up on prices of health goods constant, 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.<sup>3</sup>

Despite the good news on human health, the entailed increasing utilization of medical goods and services has already in the past raised concerns about fiscal sustainability of health insurance systems and, more generally, the overall desirability of such trends. It motivated the discussion of further rationing access to health care as potential remedy to curb further rising 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 – rations hip replacements and knee surgeries (Edwards, Crump and Dayan, 2015; OECD, 2015) and severely limits coverage of a novel (albeit expensive) drug that for the first time heals Hepatitis C.<sup>4</sup>

Applying our framework to address this important debate suggests that constraining health expenditure growth in advanced countries, where the bulk of medical R&D takes place, has severe “side-effects” 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.<sup>5</sup> We argue that, consequently, 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.

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<sup>3</sup>Depending on whether relative prices of health goods increase or decrease with medical progress, we may underestimate or overestimate the future health expenditure share, respectively.

<sup>4</sup>See <http://www.hepatitisc.uw.edu/page/treatment/drugs/sofosbuvir-drug>. NHS England has decided to provide treatment only to the 10,000 sickest persons of those being infected per year, a rather small fraction of the estimated 215,000 infected persons in the UK (<https://www.theguardian.com/society/2016/jul/28/nhs-abandoning-thousands-by-rationing-hepatitis-c-drugs>). Also other countries severely ration access to Hepatitis C treatment (World Health Organization, 2016) or discourage health spending in more general ways. For instance, in the mandatory German health system, if the amount of external costs attributable to a medical doctor exceeds a threshold per quarter, the doctor has to privately bear the costs above the cap. Contrary to many European health systems, US medicare (health insurance for the elderly) involves a co-insurance rate for pharmaceuticals of 25%. Co-insurance makes demand for pharmaceuticals price-elastic. In fact, in the US prices for pharmaceuticals are little regulated, compared to European health care systems. The fundamental issue of rationed health care provision is nevertheless present as well in the US, albeit in different form.

<sup>5</sup>Although we calibrate the model for a stylized health care system of the UK, we do not take a single-country perspective. We rather think about advanced countries as a whole and the compound effects of reducing market size by pervasive health care rationing for global medical R&D.

This leaves us with the fundamental normative question how to decide on 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 instantaneous utility of surviving individuals depends on their health status and material consumption. Marginal utility from consumption negatively depends on morbidity, in line with empirical evidence (Finkelstein, Luttmer and Notowidigdo, 2013). Our welfare analysis suggests that particularly future generations would incur dramatic welfare losses from rationing measures that stabilize the health expenditure share at its current level, despite increases in their disposable income associated with reduced health care spending. We estimate, for instance, that someone who is currently 20 years old could expect a welfare loss of 14-24 percent from the regime switch.<sup>6</sup> For those aged 20 in 2050 the estimated welfare loss is between 34 and 48 percent and associated with a reduction in remaining life expectancy by about 10 years.

The remainder of the paper is organized as follows. Section 2 discusses our contribution in view of 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 longevity as a function of access to health care. Most empirical studies of the determinants of health expenditure estimate medical technological progress as a residual. The study by the European Union (2010), for example, regresses health ex-

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<sup>6</sup>We measure welfare changes from a regime switch in the health care system by an equivalent variation measure. We ask by how much we would have to multiply material consumption levels under the baseline health policy scenario such that the *ex ante* life-time utility of a member of a given generation is the same as after the policy regime switch that forces the health expenditure share to remain at its current level.

penditure against income, the population share above 65, and a time trend, and interprets the time trend (of on average 2 percent annually) as the rate of medical technological progress. Treating medical technological progress as a time trend, however, is problematic when predictions are made on long-run developments of population health and health expenditure that do not account for potential changes in health care policy. Implicitly these predictions assume that health care reforms do not affect medical progress. In our study we challenge this view by modeling endogenous medical innovation and endogenous population health for different policy scenarios. In particular, we show that limiting the rise in health expenditure has a detrimental effect on health R&D through a market size effect that is associated with declining health care utilization. We thus formalize 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.<sup>7</sup> Our model predicts that rising life expectancy is associated with increases in health costs, capturing an important trade-off stressed in the empirical literature (e.g. Zweifel, Steinmann and Eugster, 2005; Bech et al., 2011; Breyer, Normann and Niebel, 2015).

The interaction of health R&D and health expenditure is also investigated in a couple of related papers, albeit only in “reduced form” by either assuming a direct utility gain from the consumption of pharmaceuticals (as in Garber, Jones and Romer, 2006, and Grossmann, 2013) or by assuming a direct impact of health good consumption on the mortality rate of a representative individual (as in Jones, 2016a). Garber et al. (2006) investigate the interaction between medical R&D of a monopolist and the generosity of the health care system, measured by the degree of coinsurance payment of individuals. New generations of pharmaceuticals are assumed to directly raise utility of individuals

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<sup>7</sup>Testing this hypothesis, Acemoglu et al. (2006) could not show that the introduction of Medicare (the “Social Security Act of 1965” that covered hospital and doctor expenses) increased pharmaceutical demand and pharmaceutical R&D. This finding is not surprising, however, since coverage of pharmaceuticals was not introduced until 2006. Extending the scope of analysis, Acemoglu and Linn (2004), showed large market size effects of the aging baby boomers on the development and market entry of new (age-specific) pharmaceuticals.



with the respective disease. Neither health nor longevity are explicitly modeled. Prices of pharmaceuticals are set in private markets (approximating the US health care system). In this setup, lower co-payments lead to higher demand and higher markups charged by drug producing firms.<sup>8</sup> Consequently, profits of firms may exceed consumer surplus of patients such that, in this sense, there could be too much demand and too much medical R&D. Here, in contrast, we assume that prices are regulated, approximating the British and German health care system, among others. We focus on the interaction between health expenditure, medical R&D, morbidity and longevity in a dynamic macroeconomic model.

More recently, Jones (2016a) has proposed a dynamic model with horizontal health innovations affecting longevity of a single cohort that privately finances consumption of health goods. By investigating the optimal allocation of R&D effort directed towards innovations for health and non-health purposes, it is shown that, under mild conditions, non-health technological progress may optimally converge to zero growth such that the health expenditure share optimally converges to 100 percent. 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, however, focusses on different research questions and shifts focus from the single-agent view to a multi-period, overlapping generations model with an explicit health care system and a biologically founded relationship between morbidity and mortality. In particular, we provide estimates of the effects of health care (rationing) on health and longevity of an age-structured population and account for the path-dependency of health deficits.

In a development context, higher life expectancy may positively affect per capita income (e.g. Cervellati and Sunde, 2011). In fact, longevity may foster entrepreneurship and investments in human capital because the gains of economic activity are spread over a longer time horizon. In advanced economies, however, longevity is enjoyed by retirees.

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<sup>8</sup>In a similar vein, Grossmann (2013) relates co-insurance rates to medical R&D incentives. By examining an oligopolistic pharmaceutical market with endogenous firm entry, he also shows that entry deregulation may lead to more pharmaceutical R&D despite lowering profits of pharmaceutical firms.

Thus, publicly financed policy interventions to promote health good provision and health R&D do not necessarily raise per capita income and consumption levels. Rather there is a fundamental trade-off between longevity and material well-being that we examine in our welfare analysis.

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, 2016), the historical evolution of retirement (Dalgaard and Strulik, 2017), the role of adaptation for health behavior and health outcomes (Schuennemann, Strulik and Trimborn, 2017), and the optimal design of social welfare systems (Grossmann and Strulik, 2015).<sup>9</sup>

Finally, there is a large literature outside economics that attempts forecasting 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. However, as acknowledged by the authors, such a 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 calibrates 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

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<sup>9</sup>Grossmann and Strulik (2015) 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 or a health good sector, however. Moreover, their analysis is confined to two periods of life (with endogenous lengths).

a tax-financed health care system without coinsurance, like 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 the UK and other 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 health status. Individual health status is measured by the fraction of present health deficits, i.e. the health deficit index.<sup>10</sup> 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

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

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<sup>10</sup>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).

where we assume  $\sigma > 1$  and  $\phi > 1$ .<sup>11</sup> Note that  $\tilde{m}(0) = 0$  and  $\tilde{m}(d_{\max}) = 1$ . 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 rates are related by

$$S_{v,t} = S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u}) \text{ for } t \geq v + 1, \quad (2)$$

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).<sup>12</sup> We thus implicitly assume that, conditional on survival, labor supply is independent of health status.<sup>13</sup> 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 thus first analyze the supply side and introduce life-time utility later to analyze welfare implications of our model.

## 3.2 Production

There is a standard numeraire goods sector, producing a standard final good, and a health sector.

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<sup>11</sup>In the Online Appendix (Figure 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, Bao and Rockwood, 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  $\tilde{m}'' > 0$  if  $\phi \cdot \left(1 - \frac{(d_{\max})^\phi}{\sigma}\right) > 1$ , which will hold in our calibrated model.

<sup>12</sup>These are simplifying assumptions that are hard to relax in a growth context. Allowing instead for a positive elasticity of labor supply with respect to net wages would imply increasing labor supply over time, contrary to the evidence that hours worked have declined over a longer time horizon in many growing economies (e.g. Boppart and Krusell, 2017).

<sup>13</sup>In fact, at the individual level, a decline in health status does not seem to have a large effect on labor supply (see e.g. Jaeckle and Himmler, 2010, as well as Hokayem, and Ziliak, 2014). Introducing age-dependent labor supply or varying the retirement age does not affect the main insights of our analysis.

### 3.2.1 Final Good Sector

The final good is chosen as numeraire. It is produced under perfect competition according to

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

$\alpha \in (0, 1)$ , 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. Its level is initially given by  $A_0 > 0$  and exogenously grows over time with constant rate  $g > 0$ . Physical capital depreciates at rate  $\delta^K \geq 0$ . 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.  $w_t / A_t = (1 - \alpha)(A_t L_t^Y / K_t)^{-\alpha}$ , such that

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

### 3.2.2 Health Sector

The health sector provides patentable health goods (and services) like pharmaceuticals to treat illnesses. Production of one dose of a health good requires  $\chi > 0$  units of labor. Thus, marginal production costs in period  $t$  are  $\chi w_t$ .

There is a continuum of potential illnesses that are represented by the unit interval, indexed by  $j \in [0, 1]$ . For each illness, there is a competitive R&D sector 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. An innovator is formally awarded an infinitely-lived patent. As will become apparent, however, patent holders will frequently be driven out of business by future innovators. The quality of the latest vintage of health good  $j$  available in period  $t$  is denoted by  $q_t(j)$ . The quality of health goods (including older vintages) may deteriorate 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 to treat illness  $j$  that is commercialized in  $t + 1$ . The quality of health good  $j$  then evolves according to

$$q_{t+1}(j) = \begin{cases} (1 - \delta^Q)q_t(j) + \gamma & \text{with probability } \mu_{t+1}(j), \\ (1 - \delta^Q)q_t(j) & \text{otherwise.} \end{cases} \quad (5)$$

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

$$\mathbb{E}[q_{t+1}(j)] = \mu_{t+1}(j) [q_t(j)(1 - \delta^Q) + \gamma] + (1 - \mu_{t+1}(j))q_t(j)(1 - \delta^Q). \quad (6)$$

The innovation probability,  $\mu_{t+1}(j)$ , is determined by R&D investment that affects the perceived innovation probability of a firm  $j$ ,  $\tilde{\mu}_{t+1}(j)$ , and a probability of innovation that is exogenous to firms,  $\bar{\mu}_{t+1}$ ; <sup>14</sup> i.e.,  $\mu_{t+1}(j) = 1 - (1 - \bar{\mu}_{t+1})(1 - \tilde{\mu}_{t+1}(j))$ . 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:

$$\tilde{\mu}_{t+1}(j) = \tilde{\xi}_t l_t(j), \text{ with } \tilde{\xi}_t \equiv \xi \cdot (L_t^Q)^{-\vartheta}, \quad (7)$$

$\xi > 0$ ,  $\vartheta \in (0, 1)$ , where  $L_t^Q$  is the aggregate amount of health R&D labor in  $t$ . The productivity level  $\tilde{\xi}_t$  is taken as given in the decision of R&D firms and captures a negative R&D (“duplication”) externality:  $\vartheta > 0$  implies a wedge between the private and social return 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.<sup>15</sup> In a symmetric equilibrium, where  $l_t(j) = L_t^Q$  for all  $j \in [0, 1]$ , we obtain

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<sup>14</sup>Those innovations may be thought of occurring unintentionally or are commercialized by 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.

<sup>15</sup>The argument is analogous to the one in Jones (1995) made in a non-health R&D context. For pharmaceutical R&D, Miller, Korn and Ross (2015) find that despite legal requirements and ethical

$\tilde{\mu}_{t+1}(j) = \tilde{\mu}_{t+1} = \xi \cdot (L_t^Q)^{1-\vartheta}$  for all  $j$ .<sup>16</sup>

$Q_t \equiv \int_0^1 q_t(j) dj$  denotes the average quality of the latest vintages of health goods (“stock of medical knowledge”). We assume that there is an intertemporal spillover that manifests itself in the probability of an unintentional innovation:

$$\bar{\mu}_{t+1} = \eta Q_t, \quad (8)$$

$\eta \in [0, \delta^Q/\gamma)$ . According to (7) and (8), the total probability of medical progress in any sector is given by

$$\mu_{t+1} = \bar{\mu}_{t+1} + (1 - \bar{\mu}_{t+1})\tilde{\mu}_{t+1} = \eta Q_t + (1 - \eta Q_t) \cdot \xi \cdot (L_t^Q)^{1-\vartheta}. \quad (9)$$

By the law of large numbers, there is no aggregate risk. Thus,  $\int_0^1 \mathbb{E}[q_{t+1}(j)] dj$  is deterministic and equal to  $Q_{t+1}$ . According to (6), it evolves as

$$Q_{t+1} = \gamma \mu_{t+1} + (1 - \delta^Q) Q_t, \quad (10)$$

and given initial level  $Q_0 > 0$ . Substituting (9) into (10), we obtain

$$\frac{Q_{t+1} - Q_t}{Q_t} = \frac{\gamma(1 - \bar{\mu}_{t+1})\tilde{\mu}_{t+1}}{Q_t} - \delta^Q + \gamma\eta = \frac{\gamma(1 - \eta Q_t)\xi(L_t^Q)^{1-\vartheta}}{Q_t} - \tilde{\delta}^Q, \quad (11)$$

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

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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. Thus, duplication of R&D appears to be a common phenomenon.

<sup>16</sup>For  $\vartheta \rightarrow 1$ , social returns to medical R&D investment approach zero.

### 3.3 Prices of Health Goods

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.<sup>17</sup> For instance, 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.<sup>18</sup>

Suppose that prices for older vintages are bid down to marginal costs and that these vintages are not supplied anymore, whereas the industry leader can charge a mark up that is increasing in the quality advantage vis-à-vis previous vintages. Denote by  $q > 0$  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  $1 + f(q)$ , 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.  $q = \gamma$ ), 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.  $q = 2\gamma$ ), 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  $f$  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.<sup>19</sup> 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

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<sup>17</sup>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.

<sup>18</sup>Similarly, in Germany and Switzerland, among others, health care suppliers negotiate with pharmaceutical companies the maximum price covered by the mandatory health insurance.

<sup>19</sup>See Grossman and Helpman (1991) for a similar argument in a context of Bertrand competition.



is  $\mathbf{q} = \gamma$ , implying that the price  $p_t$  of each health good is given by

$$p_t = \Gamma \chi w_t = \Gamma \chi \omega A_t, \quad (12)$$

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

### 3.4 Health Deficit Accumulation

We assume that physiological aging starts when individuals become economically active, i.e. consume and supply labor.<sup>20</sup> Modern gerontology describes aging as an accumulation of health deficits, which can be measured by a health deficit index (or frailty index). The frailty index provides the number of health deficits present in person relative to the number of potential health deficits. The evidence suggests that individual health deficits grow exponentially with age in advanced countries (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  $\nu$  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.<sup>21</sup> The health deficit index evolves according to

$$d_{v,t+1} - d_{v,t} = \begin{cases} \varrho d_{v,t} - \kappa E_{v,t} & \text{if } E_{v,t} < \frac{\varrho}{\kappa} d_{v,t}, \\ 0 & \text{otherwise,} \end{cases} \quad (13)$$

$\kappa > 0$ ,  $\varrho > 0$ , with initial value  $d_{\min} \equiv d_{v,v} > 0$ . Parameter  $\varrho$  is the growth rate of the health deficit index in absence of health interventions. It can be interpreted as the physiological “force of aging”.<sup>22</sup>  $\kappa$  is a shift parameter employed to calibrate the model.

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<sup>20</sup>We will calibrate the model such that the initial period for each cohort member is at the age of 20.

<sup>21</sup>In many advanced countries, the bulk of individuals exclusively rely on a highly regulated health system with compulsory contributions (e.g. Germany and Switzerland) or is tax-financed like the National Health Service (NHS) in the UK.

<sup>22</sup>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.

We conceptualize health input,  $E_{v,t}$ , as individual health good consumption to treat illnesses that are caused by existing health deficits, weighted by the quality of the consumed health goods. We thereby capture that health deficits derive from past, not fully cured or not fully curable illnesses. For illustration, consider two health deficits from the set of potential health deficits used in the gerontology literature. The physical difficulty to move is known to contribute to developing cardiovascular diseases. If not treated properly, these diseases lead to further health deficits. Similarly, feeling lonely may cause depression, which, without treatment, triggers further health deficits.

Formally, an individual born in  $v$  acquires a set  $\mathcal{I}_{v,t} \subset [0, 1]$  of illnesses in period  $t \geq v$ . Its measure relates to the current deficit index,  $|\mathcal{I}_{v,t}| = d_{v,t}$ . We normalize the maximally effective individual consumption per health good to unity capturing the notion of an optimal dose of treatment (pharmaceutical intake). 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 institutionally caused health care rationing. Full utilization is reflected by  $\varphi = 0$ , whereas  $\varphi = 1$  holds in absence of a health system or full exclusion from it. By the law of large numbers, suffering from a set of illnesses  $\mathcal{I}_{v,t}$  of measure  $d_{v,t}$  in  $t \geq v$ , an individual born at  $v$  receives health input

$$E_{v,t} = (1 - \varphi_t) \int_{j \in \mathcal{I}_{v,t}} q_t(j) dj = (1 - \varphi_t) d_{v,t} Q_t. \quad (14)$$

The health input 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 (14) into (13), the growth rate of the health deficit index is deterministic and independent of the deficit state. For  $t \geq v$  it is given by

$$\frac{d_{v,t+1} - d_{v,t}}{d_{v,t}} = \begin{cases} \varrho - (1 - \varphi_t) \kappa Q_t & \text{if } Q_t < \frac{\varrho}{\kappa(1 - \varphi_t)} \equiv \bar{Q}_t, \\ 0 & \text{otherwise.} \end{cases} \quad (15)$$

Equation (15) shows that individual morbidity evolves as an interaction of (exogenous) health care access and (R&D driven) health care quality.

Given the set of illnesses,  $\mathcal{I}_{v,t}$ , total health good consumption of surviving members of cohort  $v$  in period  $t$  (with population size  $S_{v,t}$  and dose  $1 - \varphi_t$  per health good) reads as

$$h_{v,t} = (1 - \varphi_t)S_{v,t}d_{v,t} \quad (16)$$

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

$$H_t = \sum_{v=t-T+1}^t h_{v,t} = (1 - \varphi_t) \sum_{v=t-T+1}^t d_{v,t}S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u}), \quad (17)$$

where we used (2) and (16) for the latter equation. Limiting health care access by imposing a higher  $\varphi$  saves health costs by reducing health good consumption. This health care rationing has two detrimental effects on health status and life expectancy. First, according to (15), it speeds up the evolution of health deficits for a given stock of medical knowledge,  $Q$ . Second, according to (17), it lowers market size for health goods,  $H$ , which, in turn, reduces the incentive for health innovations.

## 4 Equilibrium Analysis: The Future of Health and Longevity

Exploring potential futures of human health and longevity does not require explicit consideration of the health care finance side. It is implicitly assumed that the government tax-finances health expenditure  $p_t H_t$ . The trade-off to material consumption of raising health care contributions is examined in the comparative welfare analysis of different policy scenarios (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

$$L_t^Y + L_t^H + L_t^Q = L_t. \quad (18)$$

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

$$\mathfrak{s}_t \equiv \frac{p_t H_t}{GDP_t} = \frac{p_t H_t}{Y_t + p_t H_t}. \quad (19)$$

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

$$DPR_t \equiv \frac{O_t}{L_t} = \frac{\sum_{u=t-T+1}^{t-R} S_{u,t}}{\sum_{u=t-R+1}^t S_{u,t}}. \quad (20)$$

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. (2008)

## 4.2 Calibration

We assume that individuals become economically active at age 20 and die at age 120 at the latest. We dynamically calibrate the model to endogenous observables in the UK whenever available; otherwise we use North American data.

For the baseline calibration, we assume that the health care wedge  $\varphi_t$  is declining and initial cohort size  $S_{v,v}$  is non-decreasing over time (see Figure I in Appendix). The time paths of  $\varphi_t$  and  $S_{v,v}$  are dictated by the data in three ways. First, they ensure that we match the evolutions of the empirical survival function, the health expenditure share, and the employment share in the health sector. Second, the time path of  $S_{v,v}$

reflects the trend of mortality reduction of young individuals. Third, the time path of  $\varphi_t$  is roughly consistent with the historical improvements in the British health care system.<sup>23</sup> 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.” The foundation of the NHS in 1948 speeded up improvements in the access to health care for some time. We assume a particularly fast decline of  $\varphi_t$  in the time period 1997-2010. According to the extensive documentation in Boyle (2011), there have been 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. Waiting times decreased considerably. 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.<sup>24</sup> Recent improvements of the British health care system are also captured by 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 (Murray et al., 2017). In the UK, the HAQ Index improved from 74.3 in 1990 to 82.7 in 2010.

For the baseline calibration, we assume that in the future  $\varphi_t$  decreases moderately, from about 0.15 in 2010 to 0.05 in year 2080. 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.

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<sup>23</sup>Historically, innovations associated with health improvements need not exclusively be interpreted as being associated with the health care sector (that remained limited until the mid 20th century) but include also better access to sanitation and improved environmental conditions.

<sup>24</sup>For further evidence on rationing measures, see OECD, 2015, Fig. 7.11-7.13.

However, some health care rationing measures have been introduced in the UK only recently (Edwards et al., 2015). We thus also investigate a reform scenario on extending rationing (i.e. increasing  $\varphi_t$ ) in the next decades such that the health expenditure share would be stabilized.

The calibration strategy with respect to the time-invariant parameters is relegated to Appendix B. The calibrated model fits UK survival functions quite well, as shown in Figure 1. 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.<sup>25</sup> However, it does not account for changes in access or quality to health care over time that would alter future mortality rates. For life expectancy projections in the numerical analysis we will thus also employ the concept of “cohort life expectancy”.

The health expenditure share in GDP ( $\mathfrak{s}_t$ ) implied by the calibrated model 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 8.5 percent, respectively (OECD, 2016). The implied average rate of change of the health deficit index across cohorts is 3.8 percent. According to Mitnitski et al. (2002a), the estimated rate of change of the health deficit index at a given year in the cross-section of Canadian cohorts is equal to 4.3 percent for men and 3.1 percent for women. Finally, we may approximate  $\ell_t^H$  with the employment share in human health activities, as published by the OECD. For the UK, in 2010, it was 7.3 percent.<sup>26</sup> Including additionally residential care and social work activities (that may include other activities than health care provision) would suggest that  $\ell^H$  was 12.7 percent. Our calibrated model predicts a value in-between, of 10 percent in 2010.

The calibrated model implies a non-profit driven innovation probability of  $\bar{\mu}_{t+1} =$

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<sup>25</sup>Corresponding to Figure 1, Table A.2 in the Online-Appendix compares in detail the remaining “period life expectancy” predicted by the model with the actual life expectancy in the UK.

<sup>26</sup>See [http://stats.oecd.org/Index.aspx?DataSetCode=SNA\\_TABLE3](http://stats.oecd.org/Index.aspx?DataSetCode=SNA_TABLE3), retrieved on January 31, 2016.

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).<sup>27</sup>

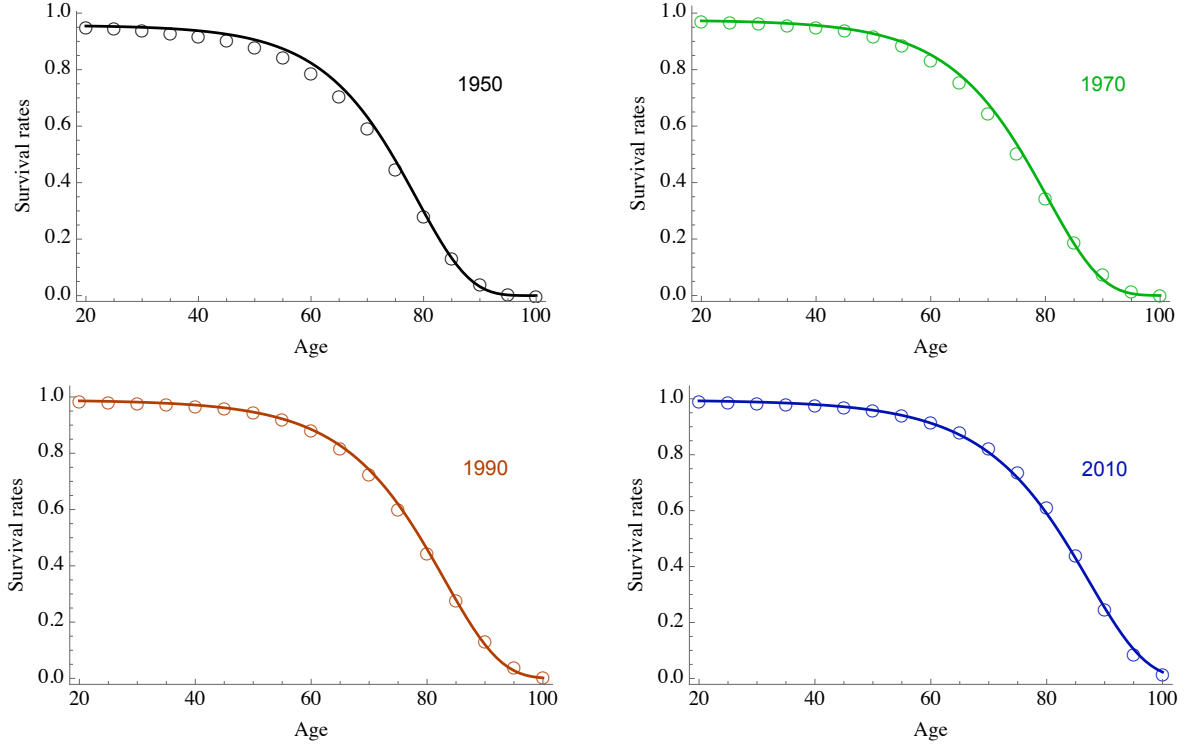


Figure 1: Survival curves for 1950, 1970, 1990 and 2010 based on contemporaneous mortality rates: Calibrated model vs. UK data.

Notes: (1) Calibrated model: solid lines, empirical series: circles. (2) Data source: [www.mortality.org](http://www.mortality.org). (3) Time paths  $\{\varphi_t\}_{t=0}^{\infty}$  and  $\{S_{v,v}\}_{v=0}^{\infty}$  are displayed in in Figure I (Appendix). (4) Initial quality index (in 1850)  $Q_0 = 0.01 \cdot \lim_{t \rightarrow \infty} Q_t$  for  $\lim_{t \rightarrow \infty} \varphi_t = 0.05$ . (5) Other parameters:  $\alpha = 0.38$ ,  $\delta^K = 0.07$ ,  $\sigma = 1.5$ ,  $\phi = 2.65$ ,  $\chi = 0.9$ ,  $\varrho = 0.04$ ,  $\kappa = 0.06$ ,  $\xi = 0.065$ ,  $\eta = 0.12$ ,  $\delta^Q = 0.02$ ,  $\vartheta = 0.6$ ,  $g = 0.02$ ,  $\bar{r} = 0.05$ ,  $d_{\min} = 0.03$ ,  $d_{\max} = 0.67$ ,  $\gamma = 0.1$ ,  $\Gamma = 1.25$ ,  $T = 101$ ,  $R = 43$ .

<sup>27</sup>Hemphill and Sampat (2012) report a standard deviation of three years, in line with an estimated range of 10-15 years for EPL of pharamceuticals in the sample analyzed by Grabowski and Kyle (2007).

### 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 ( $s_t$ ), the employment structure ( $\ell_t^H, \ell_t^Q$ ), 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 two life expectancy concepts.

#### 4.3.1 Baseline Scenario

We start with the implications of the baseline scenario, i.e. for the case of moderately decreasing  $\varphi_t$  decreases from 0.15 in 2010 to 0.05 in year 2080. Panel (a) of Figure 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).<sup>28</sup> For instance, the black line shows the surviving fraction of the 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 people get 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

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<sup>28</sup>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.



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 (16). As displayed in panel (c), total age-specific health care demand is inverted U-shaped as a 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. That is, total health care demand for a given age decreases for younger age-groups and increases for older ones. The shift reflects that, for younger individuals, improvements in the quality of health goods have little effect on survival rates, whereas the opposite holds for older individuals. In fact, survival rates of younger individuals are high and their deficit index is low 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 ( $\mathfrak{s}_t$ ) over time. 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 and our assumption that the health care wedge is declining by less in the future. Panel (e) shows that increases in health expenditure shares are associated with increases in the health employment share ( $\ell_t^H$ ), 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 ( $\ell_t^Q$ ) 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.

As is well known and reflected in (20), demographic change induced by human aging leads to a rising old-age dependency ratio ( $DPR_t$ ). The interesting question, however, is by how much we should expect old-age dependency to rise. Projections in the literature

that do not account for the endogeneity of health care quality and possible changes to health care access are not very informative in this respect. Panel (i) shows the evolution of the ratio of population size aged 63+ (retirement age) to the population size aged 20-62 (working age). It 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 that has yet not been clearly worked out in the literature: 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. For this reason, measures to raise health care rationing are not desirable from a social welfare perspective (as shown in section 5).

### 4.3.2 Rationing Scenario: Stable Health Expenditure Share

We next analyze a health care rationing scheme that stabilizes the health expenditure share from the year 2020 onwards. Rationing requires a substantial increase in the health care provision wedge ( $\varphi_t$ ) over time, from 11 percent in 2020 to 17 percent in year 2050 and 27.2 percent in year 2080. The implications can be seen in Figure 3.<sup>29</sup>

The thin lines in panels (a)-(c) of Figure 3 repeat the results for the baseline scenario shown in Figure 2, whereas the thick lines correspond to the rationing scenario. Panel (a) shows that survival rates in the rationing 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 in the rationing scenario, as shown in panel (b). Panel (c) shows that age-specific health care demand ( $h_{v,t}$ ) is lower compared to the baseline scenario, particularly for older age-groups. This outcome

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<sup>29</sup>The rationing scheme is displayed in Figure A.2 of the Online-Appendix.

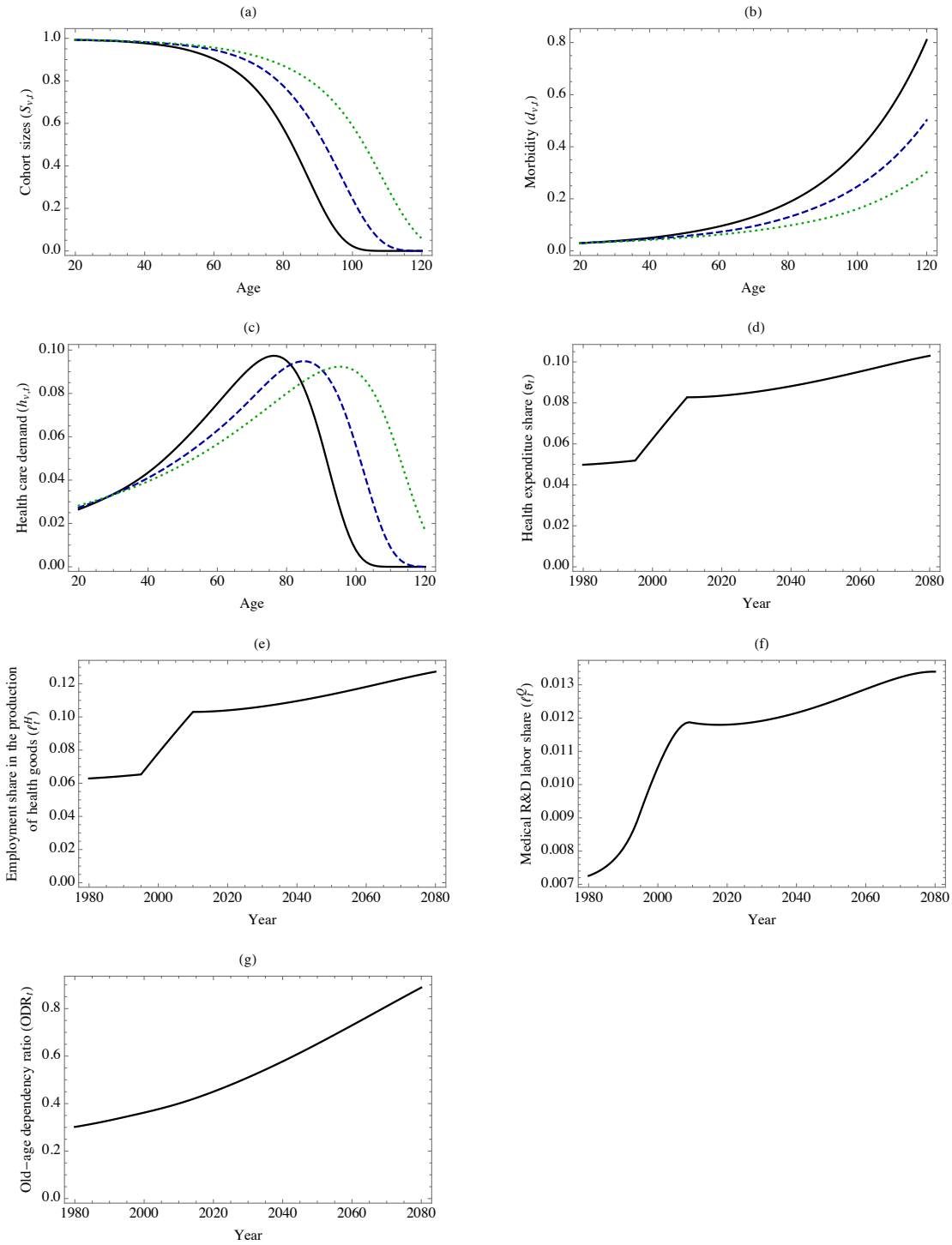


Figure 2: The future of human health, longevity and health costs for the baseline policy scenario.

Notes: (1) Panels (a)-(c): Solid (black) line for 2020, dashed (blue) line for 2050, dotted (green) line for 2080. (2) Parameters as for Figure 1.

reflects the fact that survival rates of older cohorts improve by less over time in the rationing scenario, which dominates the effect from higher morbidity at any age in the rationing scenario.

In panels (d)-(g), the solid lines reflect results from the baseline scenario whereas dashed lines reflect results from the rationing scenario. Panel (d) displays the, by design, time-invariant health expenditure share ( $\mathfrak{s}_t$ ) in the rationing scenario. Consequently, the employment share in the production of health goods ( $\ell_t^H$ ) stays basically constant as well, as shown in panel (e). Panel (f) shows that the medical R&D labor share ( $\ell_t^Q$ ) is lower than in the baseline scenario and even decreases slightly over time. This dynamic incentive effect of health care rationing 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 Comparing Life Expectancy Effects

We compare 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”, as it is usually done in the statistical literature (e.g. Kontis et al., 2017). For this, like for the survival rates displayed in Figures 1-3, we use the contemporaneous mortality rates from the cross-section of cohorts and pretend they stay constant over time. As will become apparent shortly, this dramatically underestimates life expectancy when access to health care or the quality of health care improves over time. We therefore also compute “cohort life expectancy”, based on future age-specific mortality rates.

**Period Life Expectancy** Figure 4 displays period life expectancy at a given year for 20 and 65 years old individuals for the baseline scenario (solid line) and the rationing scenario (dashed line). Circles indicate the evolution of the respective empirical period

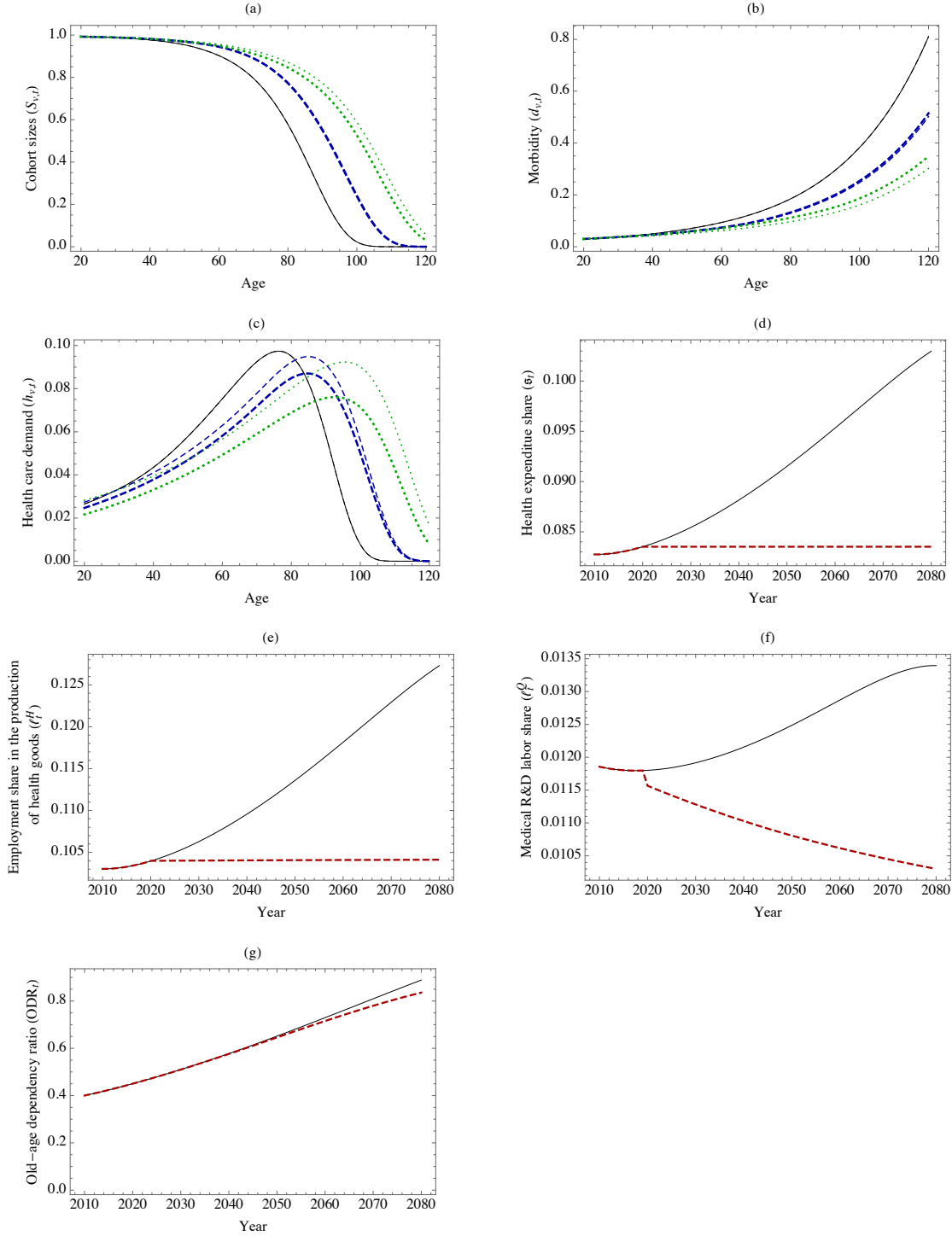


Figure 3: Effects of extending health care rationing from year 2020 onwards in order to stabilize the health expenditure share (rationing scenario).

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 rationing scenario. (2) Panels (d)-(g): Solid (black) lines repeat the baseline scenario, dashed (red) lines show the rationing scenario. (3) Time paths for  $\{\varphi_t\}$  in the rationing scenario as displayed in Figure A.2 (Online-Appendix). (4) Other parameters as for Figure 1.

life expectancies in the UK until 2010. For the baseline scenario, we observe 20 years old individuals in the year 2020 (born in 2000) expect to live until age 83.6 under the standard 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 the in 2050 and 2080.<sup>30</sup> Individuals aged 65 in 2020, 2050 and 2080 expect to live until age 86.9, 96.2 and 106.2, respectively.

Under the rationing scenario, period life expectancy increases by less than in the baseline scenario. The difference across scenarios is 0.8 years and 4.6 years 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.<sup>31</sup> In sum, the model suggests for both scenarios considerable gains in period life expectancy over time. In the shorter run, implementing the cost-saving health care reform is less detrimental than in the longer run. Over time, however, rationing induces a sizable reduction of the potential gain in life expectancy.

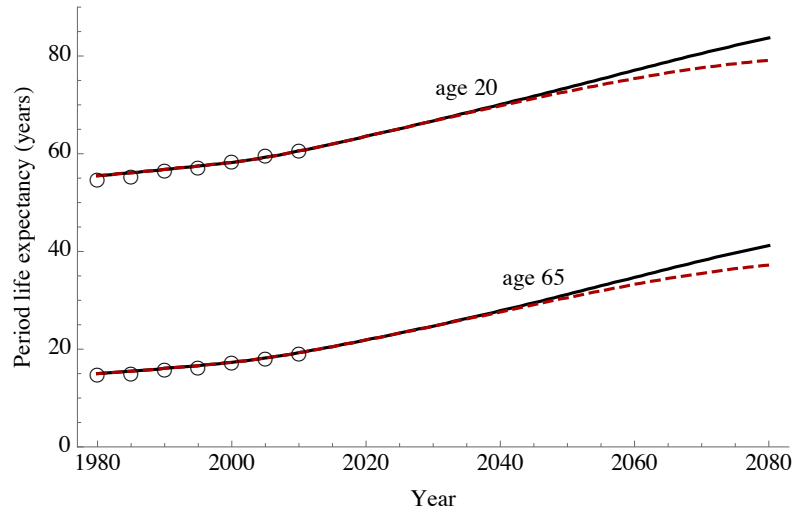


Figure 4: Implied remaining period life expectancies at age 20 and age 65: baseline vs. rationing scenario.

Notes: (1) Solid (black) lines for baseline, dashed (red) lines for reform, circles according to UK data. (2) Data source: [www.mortality.org](http://www.mortality.org). (3) Parameters as for Figure 2 (baseline) and Figure 3 (reform).

<sup>30</sup>See Table A.3 in Online-Appendix (left columns) for the remaining age-specific period life expectancies.

<sup>31</sup>Again, see Table A.3 in Online-Appendix (right columns).

**Cohort Life Expectancy** Remaining cohort life expectancy of a member of a cohort born in  $v$  is computed as follows. Noting that the number of persons surviving to age  $t - v$  is denoted by  $S_{v,t}$  as given by (2), we calculate the “person-years lived” between ages  $t - v$  and  $t - v + 1$  for individuals born in  $v$  as  $\mathcal{P}_{v,t} \equiv S_{v,t+1} + 0.5 \cdot S_{v,t}m_{v,t}$ , 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 then obtained as  $\mathcal{N}_{v,t} \equiv \sum_{u=t}^{v+T-1} \mathcal{P}_{v,u}$ . Finally, remaining life expectancy at age  $t - v$  is obtained as  $\mathcal{N}_{v,t}/S_{v,t}$ .

Figure 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. Figure 4). For example, 20 years old individuals in the year 1980 can expect to live until age 91.1 in the baseline scenario and until 90.3 in the rationing scenario. The static period life expectancy concept employed for Figure 4 thus underestimates remaining life expectancy by more than 14 years. In the baseline scenario, 20 years old individuals in 2020 can 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 much 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 Figure 5 compared to Figure 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 rationing scenario, i.e. rationing reduces life expectancy by more than one decade.<sup>32</sup> The concept of period life expectancy severely underestimates potential gains in life

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<sup>32</sup>See Table A.4 in Online-Appendix. Our predictions may be compared to the estimates of cohort life expectancy by the Office for National Statistics (2015) for the UK. It suggests that a female who has reached age 20 in year 2050 can expect to live until age 109.1 in the most optimistic of three scenarios (2.1 years longer than a comparable male), but only until age 85.5 years in the most pessimistic scenario (2.9 years longer than a comparable male).

expectancy and losses from health care rationing. Individuals aged 65 in 2050 expect to live until age 106.1 in the baseline scenario and 3.7 less in the rationing scenario. Hence, like for period life expectancy, the loss in remaining life expectancy from stabilizing the health expenditure share is lower for older persons.

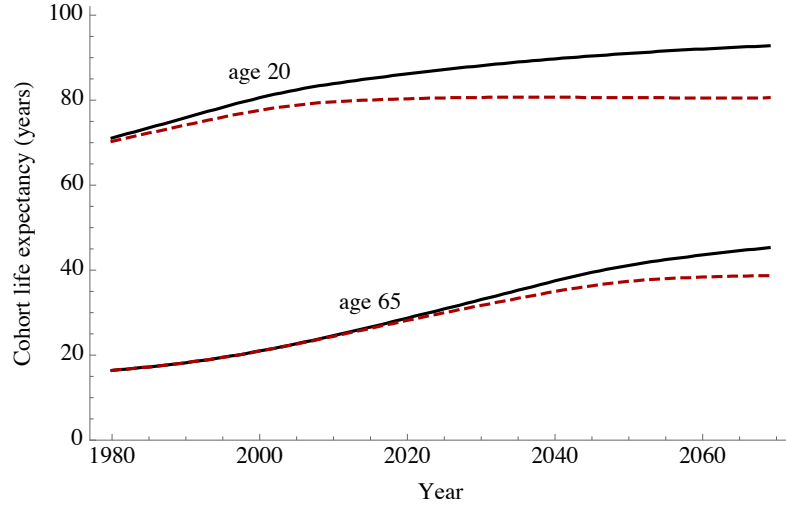


Figure 5: Implied remaining cohort life expectancies at age 20 and age 65: baseline vs. rationing scenario.

Notes: (1) Solid (black) lines for baseline, dashed (red) lines for reform. (2) Parameters as for Figure 2 (baseline) and Figure 3 (reform).

## 5 Normative Analysis of Health Care Rationing

In this section, we examine the welfare implications of a switch in health policy from the baseline scenario (analyzed in Figure 2) to the rationing 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

$$U_v = \sum_{t=v}^{v+T-1} \beta^{t-v} S_{v,t} u(c_{v,t}, d_{v,t}), \quad (21)$$

where  $\beta \geq 0$  is the discount factor and  $c_{v,t}$  denotes the consumption level in  $t$ . Instantaneous utility is specified as

$$u(c_{v,t}, d_{v,t}) \equiv \frac{\log c_{v,t}}{(1 + d_{v,t})^\zeta} + \bar{u}, \quad (22)$$

where  $\zeta > 0$  measures to which extent a higher deficit state reduces the marginal utility of consumption and  $\bar{u} \geq 0$  ensures that instantaneous utility is non-negative.<sup>33</sup> 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. Given log-utility, the intertemporal elasticity of substitution is unity, as supported by Chetty (2006), among others.

We assume that the health care system is financed by a constant contribution rate out of wage income, denoted by  $\tau_t$  for period  $t$ .<sup>34</sup> 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 (12), the health contribution rate equals the markup factor for health goods ( $\Gamma$ ) times the share of labor ( $\ell^H$ ) allocated for producing health goods and services:

$$\tau_t = \Gamma \ell_t^H. \quad (23)$$

Let asset holding (“wealth”) of a member of cohort  $v$  in  $t$  be denoted by  $a_{v,t}$ . Initial asset holding  $a_{v,v} = 0$ , since there is no bequest motive and the annuity market is perfect. We assume fair insurance within a cohort in the annuity market. That is, zero-profit

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<sup>33</sup>Otherwise, individuals could prefer to live shorter for given consumption levels, see Jones (2016a).

<sup>34</sup>Assuming that health insurance is paid exclusively by workers greatly simplifies the analysis. If health insurance were also financed by capital income, we would have to keep track of aggregate asset holdings in the economy. Recall that these are unrelated to investments as the interest rate is fixed at  $\bar{r}$ .

insurance companies pay a rate of return above  $\bar{r}$  but keep the individuals' wealth after death. The corresponding law of motion for individual wealth for a member of cohort  $v$  can be written as<sup>35</sup>

$$a_{v,t+1} = (1 - \tau_t)w_t + (1 + r_{v,t})a_{v,t} - c_{v,t}, \quad (24)$$

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

$$1 + r_{v,t} = \frac{1 + \bar{r}}{1 - m_{v,t-1}}. \quad (25)$$

Individuals of each generation  $v$  choose their consumption paths  $\{c_{v,t}\}_{t \geq v}$  to maximize utility  $U_v$  s.t. (24) and the non-negativity constraint  $a_{v,v+T} \geq 0$ . 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 in Figure I (Appendix) as long as there is no policy switch. When the rationing 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.

Welfare effects of policy reforms are evaluated as follows. Let superscript 0 on consumption levels, deficit states and survival rates denote the values of these variables in the baseline policy regime and superscript 1 the values in the policy reform regime. Let

$$\tilde{U}_v^i(\psi) \equiv \sum_{t=v}^{v+T-1} \beta^{t-v} S_{v,t}^i u(\psi c_{v,t}^i, d_{v,t}^i) \quad (26)$$

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<sup>35</sup>For simplicity, we do not consider the possibility of “out-of-pocket” health payments or coinsurance. Although the absence of these features would imply a limitation of our analysis considering countries like the U.S., it captures the health system of the UK reasonably well. The NHS does not demand copayments and many important health goods, like surgeries treating orthopedic deficits or drugs for treating cancer and virus infections, are unaffordable for most individuals if not covered by NHS. Private health insurance coverage is at a modest level (10.5 percent in the year 2014) and out-of-pocket health expenditures as fraction of total UK health expenditure was only around 10 percent in the 2000s (OECD, 2016).

denote life-time utility of cohort  $v$  when consumption levels in policy scenario  $i \in \{0, 1\}$  are multiplied with factor  $\psi > 0$ . By definition of (26), life-time utility in the rationing scenario is  $\tilde{U}_v^1(1)$ . We report cohort-specific welfare losses  $1 - \psi_v$  of switching from the baseline scenario to the rationing scenario, where  $\psi_v$  is the factor by which consumption levels of the baseline scenario are multiplied such that cohort  $v$  experiences the same utility as in the rationing scenario (equivalent variation); formally,<sup>36</sup>

$$\tilde{U}_v^0(\psi_v) = \tilde{U}_v^1(1). \quad (27)$$

## 5.2 Calibration

We choose a typical value for the subjective discount rate,  $\beta$ , such that  $\beta(1 + \bar{r}) > 1$ ,<sup>37</sup> setting  $\beta = 0.98$  and (like for the positive analysis)  $\bar{r} = 0.05$ . Next, we calibrate  $\zeta$ , 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. 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  $\mathcal{STD}(d)$ , the estimate of Finkelstein et al. (2013) then suggests that  $\zeta$  is given by

$$\frac{[1 + \mathbb{E}(d) + \mathcal{STD}(d)]^{-\zeta}}{[1 + \mathbb{E}(d)]^{-\zeta}} = 1 - LOSS. \quad (28)$$

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

Our calibrated model delivers non-negative instantaneous utility even for a utility constant  $\bar{u} = 0$  which we thus safely assume. Finally, recall that labor efficiency  $A_t$  in

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<sup>36</sup>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.

<sup>37</sup>If we assumed  $\beta(1 + \bar{r}) = 1$ , then the complementarity of consumption and health in utility implies, that consumption monotonically declines with age, which is inconsistent with the evidence.

(3) grows annually at a constant rate ( $g = 0.02$ ; see Appendix B). The challenge is that, for the welfare analysis, we need to set an initial level  $A_0$  (in the year 1850) because productivity levels affect the value of life and potentially its change when switching from the baseline scenario to the rationing scenario. For this purpose, denote the value of life of an individual born in  $v$  by  $W_v$  and assume it is given by expected (indirect) life-time utility of a cohort of age 20 in the year 2010, normalized by the marginal instantaneous (indirect) utility in the initial period of life:

$$W_v \equiv \frac{\tilde{U}_v^0(1)}{\frac{\partial u(c_{v,v}, d_{\min})}{\partial c}} = \tilde{U}_v^0(1) (1 + d_{\min})^\zeta c_{v,v}. \quad (29)$$

We calibrate  $A_0$  such that, for the year 2010, the ratio of the value of life to GDP per worker  $W_v/y_v$  of the cohort  $v = 2010$  equals 80 in the baseline scenario. Given that GDP per person employed in the UK was about 75,000 US\$ (PPP) in 2010, this corresponds to a value of life of 6 million US\$ – 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). It leads to the estimate of  $A_0 = 6.0$ . In a sensitivity analysis, we also analyze the calibrated model for values of life of 4.5 and 7.5 million US\$ (i.e.  $W_v/y_v = 60$  and  $W_v/y_v = 100$  in 2010, leading to  $A_0 = 1.55$  and  $A_0 = 23$ , respectively).

### 5.3 Results

Figure 6 displays the cohort-specific welfare losses  $1 - \psi_v$  (i.e. the absolute value of the percentage change in permanent consumption) of switching from the baseline scenario (analyzed in Figure 2) to the rationing scenario (Figure 3), where  $\psi_v$  solves (27). We see that rationing 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 (that we assumed to be entirely paid by workers) because of the reform. On the other hand, the detrimental effects from the reform 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 from the reform 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 reform 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 rationing-induced welfare loss of 20.4 percent when calibrating  $A_0 = 6$  (corresponding to a value of life of 6 million US\$ for the cohort aged 20 in the year 2010). Targeting alternatively a lower (4.5 million US\$) or higher (7.5 million US\$) value of life, the losses are 16.1 and 24.4 percent, respectively. Welfare losses are even higher for future generations. Individuals aged 20 in 2050 experience a welfare loss of in the range of 34.4-47.8 percent (41.5 percent in the medium case). These drastic welfare losses from health care rationing reflect the losses in cohort life expectancy (displayed in Figure 5) as well as increased morbidity (displayed in panel (c) of Figure 3).

## 6 Concluding Remarks

In this paper we made a first attempt to predicting future health expenditure, longevity and morbidity from an interaction of endogenous medical R&D and future health care access, proposing a novel, multi-period overlapping generations model with an age-structured population. 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 future increases in human longevity and significant reductions in morbidity especially for higher ages.

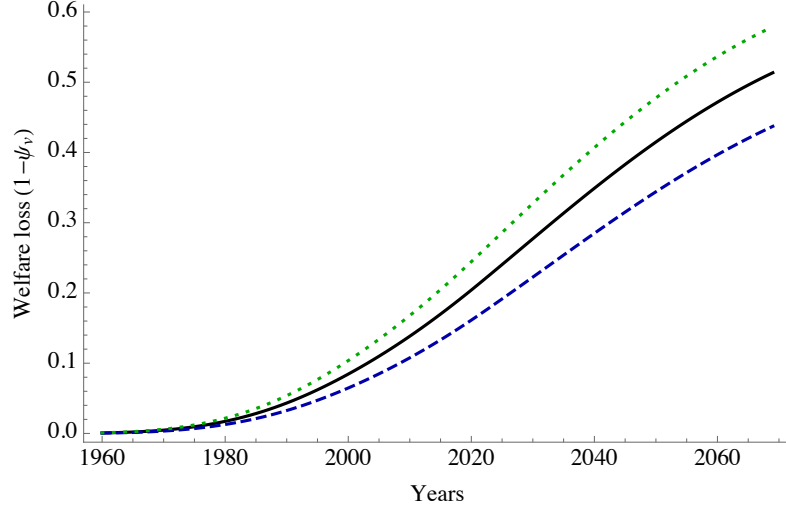


Figure 6: Cohort-specific welfare losses of extending health care rationing for stabilizing the health expenditure share for three alternative labor efficiency levels.

Notes: (1) The displayed value for year  $t$  corresponds to the welfare loss of the cost-saving reform for someone who is 20 years old in year  $t$ . (2) Solid (black) line:  $A_0 = 6$ , dashed (blue) line:  $A_0 = 1.55$ , dotted (green) line:  $A_0 = 23$ . (3)  $\beta = 0.98$ ,  $\zeta = 5.1$ . (4) Time paths  $\{\varphi_t\}$  in baseline scenario 0 and rationing scenario 1 are displayed in Figure A.3 (Online-Appendix). (5) Other parameters as for Figure 1.

The key to perform such an analysis is to capture biologically founded aging based on gerontology research. Our approach has two advantages. First, it enables us to calibrate the model by using the health deficit index as a simple and observable measure of health status and a powerful determinant of mortality. Second, our approach captures the empirically established path-dependency of health deficits. It implies that improperly treated health deficits lead to new ones that, overall, may considerably shorten life-time.

The path-dependency of health deficits has important consequences for the desirability of both extending health care access to medical advances and the implied gains in longevity. The standard reasoning in the debate on health care rationing was that some treatments like hip replacements may affect the quality of life but are typically inconsequential for remaining life expectancy and thus would be expendable. Such view has proven utterly wrong by gerontology research. For instance, the physical difficulty to move is known to contribute to developing cardiovascular diseases that may considerably

shorten life expectancy.

Our analysis suggests that stabilizing a moderately increasing health expenditure share by extending health care rationing 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 through rationing) 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 health care rationing on the accumulation of health deficits, long-run implications also work through reduced medical R&D incentives. This is so because population aging and rising health costs interact with each other through the market size effect of increased life expectancy on medical technological progress. By taking the endogeneity of medical R&D into account we arrive at a new view on the secular expansion of the health sector. A rising health expenditure share, inefficiencies in the health system notwithstanding, should not be regarded as a curse but as a blessing for human health and longevity.

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# Appendix

## A. Dynamical System and Long Run Equilibrium

- **Dynamical System:** Denote by  $\pi_t(j)$  the instantaneous profit of a health good producer in sector  $j$ . 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  $u \geq t + 1$ , the value of a vertical innovation in  $t$  reads as

$$V_t(j) \equiv \pi_t(j) + \sum_{u=t+1}^{\infty} \frac{\prod_{s=t+1}^u (1 - \mu_s(j)) \pi_u(j)}{(1 + \bar{r})^{u-t}}. \quad (30)$$

A representative R&D firm searching for a vertical innovation to treat illness  $j$  solves

$$\max_{l_t(j)} \{ \mu_{t+1}(j) V_{t+1}(j) - w_t l_t(j) \} = \left( \tilde{\xi}_t V_{t+1}(j) - w_t \right) l_t(j), \quad (31)$$

according to (7). Thus,  $\tilde{\xi}_t V_{t+1}(j) = w_t$  for all  $j$ , i.e. R&D firms do not earn profits in equilibrium and health good producers are identical in all sectors, i.e.,  $l_t(j) = L_t^Q$  and  $V_{t+1}(j) = V_{t+1}$  for all  $j \in [0, 1]$ . Using  $\tilde{\xi}_t = \xi \cdot (L_t^Q)^{-\vartheta}$ , the zero-profit condition for R&D firms reads as

$$V_{t+1} \xi (L_t^Q)^{-\vartheta} = w_t. \quad (32)$$

With mark up  $\Gamma$ , the profit per health good producer is

$$\pi_t = (p_t - \chi w_t) H_t = (\Gamma - 1) \chi w_t H_t, \quad (33)$$

according to (12). According to (30),

$$\begin{aligned} V_t = & \pi_t + \frac{1 - \mu_{t+1}}{1 + \bar{r}} \pi_{t+1} + \frac{(1 - \mu_{t+1})(1 - \mu_{t+2})}{(1 + \bar{r})^2} \pi_{t+2} + \\ & \frac{(1 - \mu_{t+1})(1 - \mu_{t+2})(1 - \mu_{t+3})}{(1 + \bar{r})^3} \pi_{t+3} + \dots, \end{aligned} \quad (34)$$

$$V_{t+1} = \pi_{t+1} + \frac{1 - \mu_{t+2}}{1 + \bar{r}} \pi_{t+2} + \frac{(1 - \mu_{t+2})(1 - \mu_{t+3})}{(1 + \bar{r})^2} \pi_{t+3} + \dots = \frac{1 + \bar{r}}{1 - \mu_{t+1}} (V_t - \pi_t). \quad (35)$$

Using (33) in (35), we get the following no-arbitrage condition in the market that

finances health R&D:

$$\frac{1 - \mu_{t+1}}{1 + \bar{r}} \frac{V_{t+1}}{V_t} + \frac{(\Gamma - 1)w_t \chi H_t}{V_t} = 1. \quad (36)$$

Now let us define  $\mathcal{V}_t \equiv V_t/A_t$ . Denote by  $\mathfrak{d}_{\mathfrak{a},t}$  the health deficit index of a surviving individual of age  $\mathfrak{a}$  in period  $t$  and  $\tilde{\mathfrak{a}}_t$  as the highest age in period  $t$  such that  $\mathfrak{d}_{\mathfrak{a},t} \leq d_{\max}$ . Thus,  $\bar{\mathfrak{a}}_t \equiv \min(\tilde{\mathfrak{a}}_t, T)$  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:

$$\mathfrak{d}_{1,t+1} = [1 + \varrho - (1 - \varphi)\kappa Q_t] d_{\min}, \quad (37)$$

$$\mathfrak{d}_{2,t+1} = [1 + \varrho - (1 - \varphi)\kappa Q_t] \mathfrak{d}_{1,t}, \quad (38)$$

$$\mathfrak{d}_{3,t+1} = [1 + \varrho - (1 - \varphi)\kappa Q_t] \mathfrak{d}_{2,t}, \quad (39)$$

$\vdots$

$$\mu_{t+1} = \eta Q_t + (1 - \eta Q_t) \cdot \xi \cdot (L_t^Q)^{1-\vartheta}, \quad (40)$$

$$Q_{t+1} - Q_t = \gamma (1 - \eta Q_t) \xi (L_t^Q)^{1-\vartheta} - (\delta^Q - \gamma \eta) Q_t, \quad (41)$$

$$\frac{1 - \mu_{t+1}}{1 + \bar{r}} \mathcal{V}_{t+1} (1 + g) + (\Gamma - 1) w_t L_t^H = \mathcal{V}_t, \quad (42)$$

$$\mathcal{V}_{t+1} (1 + g) \xi \cdot (L_t^Q)^{-\vartheta} = \omega, \quad (43)$$

$$\begin{aligned} H_t = & (1 - \varphi_t) S_{t,t} d_{\min} + (1 - \varphi_t) (1 - \tilde{m}(d_{\min})) \times \\ & \{ S_{t-1,t-1} \mathfrak{d}_{1,t} + \mathfrak{d}_{2,t} S_{t-2,t-2} (1 - \tilde{m}(\mathfrak{d}_{1,t-1})) + \\ & \mathfrak{d}_{3,t} S_{t-3,t-3} (1 - \tilde{m}(\mathfrak{d}_{2,t-1})) (1 - \tilde{m}(\mathfrak{d}_{1,t-2})) + \dots + \\ & \mathfrak{d}_{\bar{\mathfrak{a}}_t,t} S_{t-\bar{\mathfrak{a}}_t,t-\bar{\mathfrak{a}}_t} (1 - \tilde{m}(\mathfrak{d}_{\bar{\mathfrak{a}}_t-1,t-1})) (1 - \tilde{m}(\mathfrak{d}_{\bar{\mathfrak{a}}_t-2,t-2})) \times \dots \times (1 - \tilde{m}(\mathfrak{d}_{1,t-\bar{\mathfrak{a}}_t+1})) \} \end{aligned} \quad (44)$$

$$L_t^Y + L_t^H + L_t^Q = L_t, \quad (45)$$

$$L_t^H = \chi H_t,$$

according to (15), (9), (11), (36), (32), (17), (18) and the assumption of a unit mass of health good producers (i.e., the total and per firm amount of labor allocated to the



production of health goods is the same), respectively. Initial quality index  $Q_0 > 0$  and the vector of current deficit states of the cohorts living in period 0,  $\mathbf{d}_0 \equiv (\mathfrak{d}_{1,0}, \mathfrak{d}_{2,0}, \mathfrak{d}_{3,0}, \dots, \mathfrak{d}_{\bar{\mathfrak{a}}_0,0})$ , are given.

- **Long Run Equilibrium:** We next derive the long run equilibrium (focussing on the case where  $Q_{t+1} = Q_t$  holds for  $t \rightarrow \infty$  only). Setting  $Q_{t+1} = Q_t = Q$  in (10) and omitting the time index, we obtain

$$L^Q = \left( \frac{\frac{\delta^Q}{\gamma} Q - \bar{\mu}}{(1 - \bar{\mu})\xi} \right)^{\frac{1}{1-\vartheta}}. \quad (46)$$

Using  $\mathcal{V}_{t+1} = \mathcal{V}_t = \mathcal{V}$  in (42) implies

$$\mathcal{V} = \frac{(\Gamma - 1)(1 + \bar{r})\omega L_t^H}{\bar{r} - g + \mu(1 + g)}. \quad (47)$$

Moreover, according to (43) and (46),

$$\mathcal{V} = \frac{\omega(L^Q)^\vartheta}{(1 + g)\xi}. \quad (48)$$

Combining (47) and (48) implies

$$\frac{(L^Q)^\vartheta}{\xi} = \frac{(\Gamma - 1)(1 + \bar{r})L^H}{\frac{\bar{r}-g}{1+g} + \mu}. \quad (49)$$

Let  $\hat{\mathfrak{d}}_{\mathfrak{a}}$  denote the long run health deficit index of individual of age  $\mathfrak{a} \geq 0$ , associated with the steady state quality index  $\hat{Q} < \bar{Q}$ . Moreover, let  $\hat{\varphi} \equiv \lim_{t \rightarrow \infty} \varphi_t$  and suppose that  $\hat{S} \equiv \lim_{v \rightarrow \infty} S_{v,v} = 1$ . According to (15),

$$\hat{\mathfrak{d}}_{\mathfrak{a}+1} = \left[ 1 + \varrho - (1 - \hat{\varphi})\hat{Q} \right] \hat{\mathfrak{d}}_{\mathfrak{a}}, \quad (50)$$

with initial condition  $\hat{\mathfrak{d}}_0 = d_{\min} > 0$ . The solution of difference equation (50) provides the steady state age-path of the health deficit index conditional on  $\varphi$  and  $\hat{Q}$ , denoted by  $\mathfrak{D}(\mathfrak{a}, \varphi, \hat{Q})$ ,  $\mathfrak{a} \geq 0$ . Function  $\mathfrak{D}(\mathfrak{a}, \varphi, Q)$  is increasing in age,  $\mathfrak{a}$ , increasing in  $\varphi$ , and decreasing in quality index,  $Q$ . Let  $\tilde{\mathfrak{a}}_\infty$  denote the largest age

$\mathfrak{a}$  such that  $\mathfrak{D}(\mathfrak{a}, \varphi, Q) \leq d_{\max}$  and define function

$$\tilde{H}(\varphi, Q) \equiv (1 - \varphi) \left[ \sum_{\mathfrak{a}=0}^{\bar{\mathfrak{a}}_{\infty}} \mathfrak{D}(\mathfrak{a}, \varphi, Q) \prod_{u=0}^{\mathfrak{a}} [1 - \tilde{m}(\mathfrak{D}(u, \varphi, Q))] \right], \quad (51)$$

where  $\bar{\mathfrak{a}}_{\infty} \equiv \min(\tilde{\mathfrak{a}}_{\infty}, T)$ . Substituting (46) into (40), we have

$$\mu = \frac{\delta Q}{\gamma} Q. \quad (52)$$

Substituting (52) into (49) and using  $\bar{\mu} = \eta Q$ ,  $L^H = \chi \tilde{H}(\varphi, Q)$  and (46) we obtain

$$\frac{\delta Q}{\gamma} Q = \left( \frac{\frac{1}{Q} - \eta}{\frac{\delta Q}{\gamma} - \eta} \right)^{\frac{\vartheta}{1-\vartheta}} \xi^{\frac{1}{1-\vartheta}} (\Gamma - 1)(1 + \bar{r}) \chi \tilde{H}(\varphi, Q) - \frac{\bar{r} - g}{1 + g}, \quad (53)$$

which implicitly defines  $\hat{Q}$ . We see that  $\hat{Q}$  is unique when  $\tilde{H}(\varphi, Q)$  is non-increasing in  $Q$ . The other long run values follow.

## B. Calibration (Equilibrium Analysis)

We first consider the output elasticity of labor,  $1 - \alpha = wL^Y/Y$ . According to Karabarbounis and Neiman (2014, “CLS KN merged”), the arithmetic average for the period 1987-2011 of the UK corporate labor share in total income has been 62 percent (which is also the 2011 value). Thus, we set  $\alpha = 0.38$ . For the real interest rate we choose the typical value  $\bar{r} = 0.05$ . For the depreciation rate of physical capital we follow Grossmann and Steger (2017) who argue that  $\delta^K = 0.07$ . The growth rate of wage rates is set equal to the annual growth rate of income per capita in the UK for period 1960-2011,  $g = 0.02$  (Jones, 2016b).

We assume that individuals become economically active at age 20 and live for a at most 100 further years; 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).<sup>38</sup>

We set  $d_{\min}$  equal to the average health deficit index for a 20 years old individual in recent times. Using Canadian data, Mitnitski et al. (2002a) suggest  $d_{\min} = 0.03$ . Empirical evidence also suggests that the deficit state that leads to death for sure approximately

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<sup>38</sup>In the UK, 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).

is about two thirds (e.g. Harttgen et al., 2013); thus,  $d_{\max} = 0.67$ .

The remaining parameters are the mortality rate curvature parameters  $(\sigma, \phi)$ , the labor requirement per unit of health good  $(\chi)$ , medical R&D technology parameters  $(\xi, \delta^Q, \vartheta)$ , innovation step size  $(\gamma)$ , the strength of the intertemporal innovation spillover  $(\eta)$ , the price markup for health goods  $(\Gamma)$ , health deficit accumulation parameters  $(\varrho, \kappa)$ , the initial quality index of health goods  $(Q_0)$ , the time path of the health care wedge,  $\{\varphi_t\}_{t=0}^\infty$ , the time path of initial survival rates,  $\{S_{v,v}\}_{v=0}^\infty$ , and health deficits of all cohorts with living members in the initial period  $(\mathbf{d}_0)$ .<sup>39</sup> These parameters are chosen to simultaneously match the observables highlighted in the main text: (i) empirical UK survival rates for ages 20-100 and periods 1950, 1970, 1990, 2010, (ii) the UK ratio of health expenditure to GDP  $(\mathfrak{s}_t)$  between 1980-2010, (iii) the recent average rate of change of the health deficit index  $(d_{v,t})$  in the cross-section of Canadian cohorts, (iv) the UK employment share in the health sector  $(\ell_t^H)$ .

To match UK survival rates from year 1950 onwards (www.mortality.org), as  $T = 101$ , we need to specify initial conditions for the deficit index of all cohorts with living members in year 1850, i.e. we choose 1850 as initial period ( $t = 0$ ). According to (1), (2) and (15), given the vector of initial deficit states  $(\mathbf{d}_0)$ , the deficit state of each cohort member at age 20 ( $d_{\min}$ ) and the initial quality index of health goods,  $Q_0$ , 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,  $\{Q_t\}_{t=0}^\infty$ . We assume that  $\mathbf{d}_0$  results from a policy regime in which a health care system has never existed (i.e.  $\varphi_0 = 1$ ).<sup>40</sup> Moreover,  $Q_0$  is set to one percent of the steady state value of  $Q$  that results for  $\varphi = 0.05$  (assumed to be reached in 2080).<sup>41</sup> The evolution of the time-variant parameters is displayed in Figure I.

For the time-invariant parameters, a steady state analysis is instructive to understand the relationship between endogenous observables and helps us to calibrate the model. First, setting  $Q_{t+1} = Q_t = Q$  in (41) and using both  $\bar{\mu} = \eta Q$  and  $\tilde{\mu} = \xi(L^Q)^{1-\vartheta}$ , we

<sup>39</sup>Initial labor efficiency  $A_0$ , does not enter the dynamical system for the positive analysis (Appendix A).

<sup>40</sup>Formally, recall that  $\mathfrak{d}_{\mathfrak{a},0}$  denotes the deficit index of a surviving individual of age  $\mathfrak{a}$  in period 0. According to (15), we have  $\mathfrak{d}_{\mathfrak{a},0} = d_{\min}(1 + \varrho)^{\mathfrak{a}}$  for all  $\mathfrak{a} \in [0, \bar{\mathfrak{a}}_0]$ , where  $\bar{\mathfrak{a}}_0$  is the maximum age in period 0. Thus,  $\mathbf{d}_0 = (\mathfrak{d}_{1,0}, \mathfrak{d}_{2,0}, \mathfrak{d}_{3,0}, \dots, \mathfrak{d}_{\bar{\mathfrak{a}}_0,0})$ .

<sup>41</sup>Our calibrated model leads to the case where steady state quality of health goods  $\hat{Q} \equiv \lim_{t \rightarrow \infty} Q_t < \bar{Q}_t$ . We can verify that the steady state equilibrium of the calibrated model is saddle-point stable.

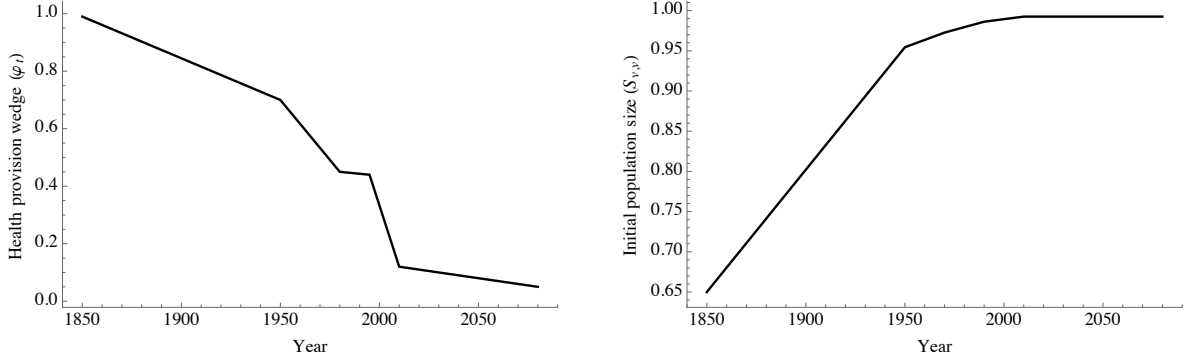


Figure I: Calibration of the time paths of the health care wedge and initial cohort sizes in the baseline scenario.

obtain

$$[\bar{\mu} + (1 - \bar{\mu})\tilde{\mu} =] \mu = \frac{\delta^Q}{\gamma\eta} \bar{\mu} = \frac{\delta^Q}{\gamma} Q. \quad (54)$$

Thus, in the long run, the total innovation probability  $\mu$  is proportional to  $\bar{\mu}$  and thus proportional to the medical knowledge stock,  $Q$ . Second, according to (8) and (40),

$$\frac{(L^Q)^\vartheta}{\xi} = \frac{(1 - \bar{\mu})L^Q}{\mu - \bar{\mu}}. \quad (55)$$

Combining (55) with (49) and using both  $L^H = \chi H$  and (54) implies that

$$\ell^Q = \frac{\frac{\delta^Q}{\gamma\eta} - 1}{\frac{1}{\bar{\mu}} - 1} \frac{(\Gamma - 1)(1 + \bar{r})\ell^H}{\frac{\bar{r} - g}{1 + g} + \frac{\delta^Q}{\gamma\eta}} \quad (56)$$

holds in the long run (recall that  $\delta^Q > \gamma\eta$ ). Third, according to (19), the health expenditure share can be written as

$$\mathfrak{s} = \frac{pH}{Y + pH} = \frac{1}{\frac{Y}{pH} + 1} = \frac{1}{\frac{L^Y}{\Gamma\omega L^H} \left(\frac{K^Y}{AL^Y}\right)^\alpha + 1} = \frac{1}{\frac{\ell^Y}{(1-\alpha)\Gamma\ell^H} + 1}, \quad (57)$$

where we used (3) and (12) 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 ( $\ell^Q$ ) nor for the health good price markup factor ( $\Gamma$ ). Fortunately, however, (56) and (57) show that given the observable employment share in health goods production,  $\ell^H$ ,

the health R&D productivity parameter  $\xi$  does neither affect (long run) levels of the employment share of medical R&D workers,  $\ell^Q$  (driving health good quality  $Q$  that in turn drives deficit accumulation and the evolution of mortality rates over time), nor the health expenditure share,  $\mathfrak{s}$ . This points to the possibility that many combinations of  $\Gamma$  and  $\xi$  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 match 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).

As stated in the main text, in addition to matching (i)-(iv), our calibrated model implies an unintentional probability of a successful innovation of  $\bar{\mu}_{t+1} = 0.034$  and a total probability of  $\mu_{t+1} = 0.08$  in the year 2010. 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.<sup>42</sup>

### C. Consumption Paths (Normative Analysis)

- **Without policy reform:** We assumed that 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 in Figure I (Appendix) as long as there is no policy switch. In this case, using  $S_{v,t} = S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u})$  in (21), the Lagrangian  $\mathcal{L}_v$  associated with maximizing  $U_v$  subject to (24) and  $a_{v,v+T} \geq 0$  is

$$\begin{aligned} \mathcal{L}_v = & \dots + \beta^{t-v} S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u}) \frac{\log c_{v,t}}{(1 + d_{v,t})^\zeta} + \\ & \beta^{t+1-v} S_{v,v} \prod_{u=v}^t (1 - m_{v,u}) \frac{\log c_{v,t+1}}{(1 + d_{v,t+1})^\zeta} + \dots + \\ & \lambda_{v,t} [(1 - \tau_t)w_t + (1 + r_{v,t})a_{v,t} - c_{v,t} - a_{v,t+1}] + \\ & \lambda_{v,t+1} [(1 - \tau_{t+1})w_{t+1} + (1 + r_{v,t+1})a_{v,t+1} - c_{v,t+1} - a_{v,t+2}] + \dots \quad (58) \end{aligned}$$

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<sup>42</sup>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 recent immigration into the UK labor market that was primarily enabled by the free movement of labor within the European Union. Rather than focussing on the level, we are thus rather interested on changes of  $DPR_t$  over time.

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

$$\frac{\beta^{t-v} S_{v,v} \prod_{u=v}^{t-1} (1 - m_{v,u})}{(1 + d_{v,t})^\zeta c_{v,t}} = \lambda_{v,t}, \quad (59)$$

$$\frac{\beta^{t+1-v} S_{v,v} \prod_{u=v}^t (1 - m_{v,u})}{(1 + d_{v,t+1})^\zeta c_{v,t+1}} = \lambda_{v,t+1}, \quad (60)$$

$$\lambda_{v,t} = \lambda_{v,t+1} (1 + r_{v,t+1}). \quad (61)$$

Combining (59)-(61) leads to

$$\frac{(1 + d_{v,t+1})^\zeta c_{v,t+1}}{(1 + d_{v,t})^\zeta c_{v,t}} = \beta (1 - m_{v,t}) (1 + r_{v,t+1}). \quad (62)$$

Using (25) in (62) implies

$$c_{v,t+1} = \left( \frac{1 + d_{v,t}}{1 + d_{v,t+1}} \right)^\zeta \beta (1 + \bar{r}) c_{v,t}. \quad (63)$$

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

$$c_{v,t} = \left( \frac{1 + d_{\min}}{1 + d_{v,t}} \right)^\zeta \beta^{t-v} (1 + \bar{r})^{t-v} c_{v,v}. \quad (64)$$

From (24), (25),  $a_{v,v} = 0$  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

$$c_{v,v} + \sum_{t=v+1}^{v+T-1} \left( \frac{c_{v,t}}{\prod_{u=v+1}^t (1 + r_{v,u})} \right) = (1 - \tau_v) w_v + \sum_{t=v+1}^{v+R-1} \left( \frac{(1 - \tau_t) w_t}{\prod_{u=v+1}^t (1 + r_{v,u})} \right). \quad (65)$$

Using (25) and (64), we obtain for the left-hand side of (65) that

$$c_{v,v} + \sum_{t=v+1}^{v+T-1} \left( \frac{c_{v,t}}{\prod_{u=v+1}^t (1 + r_{v,u})} \right) = c_{v,v} \left( 1 + \sum_{t=v+1}^{v+T-1} \beta^{t-v} \left( \frac{1 + d_{\min}}{1 + d_{v,t}} \right)^\zeta \prod_{u=v}^{t-1} (1 - m_{v,u}) \right). \quad (66)$$

Equating the right-hand sides of (65) and (66), and using (25),  $w_t = \omega A_t$  with  $\omega$  given by (4), (2) with  $S_{v,v} = 1$  and  $A_t = A_v (1 + g)^{t-v}$ , implies that the initial

consumption level,  $c_{v,v}$ , is given by

$$c_{v,v} = \omega A_v \frac{1 - \tau_v + \sum_{t=v+1}^{v+R-1} (1 - \tau_t) \left( \frac{1+g}{1+\bar{r}} \right)^{t-v} \frac{S_{v,t}}{S_{v,v}}}{1 + \sum_{t=v+1}^{v+T-1} \beta^{t-v} \left( \frac{1+d_{\min}}{1+d_{v,t}} \right)^\zeta \frac{S_{v,t}}{S_{v,v}}}. \quad (67)$$

For the welfare analysis, for each cohort we feed in the consumption path (64) with initial level (67).

- **With policy reform:** We now turn to the case where currently living individuals experience policy shocks in period  $t_0$ . That is, for  $t < t_0$  they follow the same consumption path as computed in the previous case and then they re-optimize in  $t_0$ . According to (63), knowing  $c_{v,t_0}$ , the path of consumption of any living member of generation  $v$  for future dates  $t \geq t_0$  evolves as

$$c_{v,t} = \left( \frac{1 + d_{v,t_0}}{1 + d_{v,t}} \right)^\zeta \beta^{t-t_0} (1 + \bar{r})^{t-t_0} c_{v,t_0}. \quad (68)$$

Using (24) and  $a_{v,v} = 0$ , for  $t_0 < v + R$  we have

$$\frac{a_{v,t_0}}{\prod_{u=v+1}^{t_0-1} (1 + r_{v,u})} = (1 - \tau_v) w_v - c_{v,v} + \sum_{t=v+1}^{t_0-1} \frac{(1 - \tau_t) w_t - c_{v,t}}{\prod_{u=v+1}^t (1 + r_{v,u})} \quad (69)$$

Using (25), (64) and (2), we obtain

$$c_{v,v} + \sum_{t=v+1}^{t_0-1} \left( \frac{c_{v,t}}{\prod_{u=v+1}^t (1 + r_{v,u})} \right) = c_{v,v} \left( 1 + \sum_{t=v+1}^{t_0-1} \beta^{t-v} \left( \frac{1 + d_{\min}}{1 + d_{v,t}} \right)^\zeta \frac{S_{v,t}}{S_{v,v}} \right). \quad (70)$$

Using (25) and (2), we also get

$$\frac{1}{\prod_{u=v+1}^{t_0-1} (1 + r_{v,u})} = \frac{S_{v,t_0-1}}{S_{v,v} (1 + \bar{r})^{t_0-1-v}}. \quad (71)$$

Substituting (70), (71),  $w_t = \omega A_t$  and  $A_t = A_v (1 + g)^{t-v}$  into (69), the wealth

holding of a member of generation  $v$  in  $t_0 < v + R$  is given by

$$a_{v,t_0} = (1 + \bar{r})^{t_0-v-1} \frac{S_{v,v}}{S_{v,t_0-1}} \left[ A_v \omega \left( 1 - \tau_v + \sum_{t=v+1}^{t_0-1} (1 - \tau_t) \left( \frac{1+g}{1+\bar{r}} \right)^{t-v} \frac{S_{v,t}}{S_{v,v}} \right) - c_{v,v} \sum_{t=v+1}^{t_0-1} \left( \frac{1+d_{\min}}{1+d_{v,t}} \right)^\zeta \beta^{t-v} \frac{S_{v,t}}{S_{v,v}} - c_{v,v} \right]. \quad (72)$$

Analogously, for  $t_0 \geq v + R$ , we have

$$a_{v,t_0} = (1 + \bar{r})^{t_0-v-1} \frac{S_{v,v}}{S_{v,t_0-1}} \left[ A_v \omega \left( 1 - \tau_v + \sum_{t=v+1}^{v+R-1} (1 - \tau_t) \left( \frac{1+g}{1+\bar{r}} \right)^{t-v} \frac{S_{v,t}}{S_{v,v}} \right) - c_{v,v} \sum_{t=v+1}^{t_0-1} \left( \frac{1+d_{\min}}{1+d_{v,t}} \right)^\zeta \beta^{t-v} \frac{S_{v,t}}{S_{v,v}} - c_{v,v} \right]. \quad (73)$$

Recall that  $c_{v,v}$  is the initial consumption level chosen before the unanticipated shock occurs. Next, use (24) and  $a_{v,v+T} = 0$  to obtain

$$c_{v,t_0} + \sum_{t=t_0+1}^{v+T-1} \frac{c_{v,t}}{\prod_{u=t_0+1}^t (1 + r_{v,u})} = (1 + r_{v,t_0}) a_{v,t_0} + (1 - \tau_{t_0}) w_{t_0} + \sum_{t=t_0+1}^{v+R-1} \frac{(1 - \tau_t) w_t}{\prod_{u=t_0+1}^t (1 + r_{v,u})}. \quad (74)$$

Using (68) implies

$$c_{v,t_0} + \sum_{t=t_0+1}^{v+T-1} \frac{c_{v,t}}{\prod_{u=t_0+1}^t (1 + r_{v,u})} = c_{v,t_0} \left( 1 + \sum_{t=t_0+1}^{v+T-1} \left( \frac{1+d_{v,t_0}}{1+d_{v,t}} \right)^\zeta \beta^{t-t_0} \prod_{u=t_0}^{t-1} (1 - m_{v,u}) \right). \quad (75)$$

Equating the right-hand sides of (74) and (75) and using (25),  $w_t = \omega A_t$  and  $A_t = A_{t_0} (1 + g)^{t-t_0}$  implies, for  $t_0 < v + R$ , the consumption level:

$$c_{v,t_0} = \frac{\frac{1+\bar{r}}{1-m_{v,t_0-1}} a_{v,t_0} + \omega A_{t_0} \left( 1 - \tau_{t_0} + \sum_{t=t_0+1}^{v+R-1} (1 - \tau_t) \left( \frac{1+g}{1+\bar{r}} \right)^{t-t_0} \prod_{u=t_0}^{t-1} (1 - m_{v,u}) \right)}{1 + \sum_{t=t_0+1}^{v+T-1} \left( \frac{1+d_{v,t_0}}{1+d_{v,t}} \right)^\zeta \beta^{t-t_0} \prod_{u=t_0}^{t-1} (1 - m_{v,u})} \quad (76)$$

with  $a_{v,t_0}$  given by (72) and  $A_{t_0} = A_v (1 + g)^{t_0-v}$ . Analogously, for  $t_0 \geq v + R$  (i.e.



the individual is retired when the shock hits), we have

$$c_{v,t_0} = \frac{\frac{1+\bar{r}}{1-m_{v,t_0}-1} a_{v,t_0}}{1 + \sum_{t=t_0+1}^{v+T-1} \left( \frac{1+d_{v,t_0}}{1+d_{v,t}} \right)^\zeta \beta^{t-t_0} \prod_{u=t_0}^{t-1} (1 - m_{v,u})} \quad (77)$$

with  $a_{v,t_0}$  given by (73). For the welfare analysis, we again we feed in for each cohort the consumption path (64) with initial level (67) for  $t < t_0$  (before the policy switch) and the consumption path (68) with initial level (77) for  $t \geq t_0$  (after the policy switch).

## Online-Appendix: Additional Figures and Tables

List of 40 Variables included in the frailty index	Cut Point	
Help Bathing	Yes = 1, No = 0	
Help Dressing	Yes = 1, No = 0	
Help getting in/out of Chair	Yes = 1, No = 0	
Help Walking around house	Yes = 1, No = 0	
Help Eating	Yes = 1, No = 0	
Help Grooming	Yes = 1, No = 0	
Help Using Toilet	Yes = 1, No = 0	
Help up/down Stairs	Yes = 1, No = 0	
Help lifting 10 lbs	Yes = 1, No = 0	
Help Shopping	Yes = 1, No = 0	
Help with Housework	Yes = 1, No = 0	
Help with meal Preparations	Yes = 1, No = 0	
Help taking Medication	Yes = 1, No = 0	
Help with Finances	Yes = 1, No = 0	
Lost more than 10 lbs in last year	Yes = 1, No = 0	
Self Rating of Health	Poor = 1, Fair = 0.75, Good = 0.5, V. Good = 0.25, Excellent = 0	
How Health has changed in last year	Worse = 1, Better/Same = 0	
Stayed in Bed at least half the day due to health (in last month)	Yes = 1, No = 0	
Cut down on Usual Activity (in last month)	Yes = 1, No = 0	
Walk outside	<3 days = 1, ≤ 3 days = 0	
Feel Everything is an Effort	Most of time = 1, Some time = 0.5, Rarely = 0	
Feel Depressed	Most of time = 1, Some time = 0.5, Rarely = 0	
Feel Happy	Most of time = 0, Some time = 0.5, Rarely = 1	
Feel Lonely	Most of time = 1, Some time = 0.5, Rarely = 0	
Have Trouble getting going	Most of time = 1, Some time = 0.5, Rarely = 0	
High blood pressure	Yes = 1, Suspect = 0.5, No = 0	
Heart attack	Yes = 1, Suspect = 0.5, No = 0	
CHF	Yes = 1, Suspect = 0.5, No = 0	
Stroke	Yes = 1, Suspect = 0.5, No = 0	
Cancer	Yes = 1, Suspect = 0.5, No = 0	
Diabetes	Yes = 1, Suspect = 0.5, No = 0	
Arthritis	Yes = 1, Suspect = 0.5, No = 0	
Chronic Lung Disease	Yes = 1, Suspect = 0.5, No = 0	
MMSE	<10 = 1, 11–17 = 0.75, 18–20 = 0.5, 20–24 = 0.25, >24 = 0	
Peak Flow	See Table 2	
Shoulder Strength	See Table 2	
BMI	See Table 2	
Grip Strength	See Table 2	
Usual Pace	See Table 2	
Rapid Pace	See Table 2	
Variable	Deficit for Men	Deficit for Women
Peak Flow (liters/min)	≤ 340	≤ 310
Body Mass Index (BMI)	<18.5, ≥ 30 as a deficit. 25-<30 as a 'half deficit'	<18.5, ≥ 30 as a deficit. 25-<30 as a 'half deficit'
Shoulder Strength (kg)	≤ 12	≤ 9
Grip Strength (GS in kg)	For BMI ≤ 24, GS ≤ 29 For BMI 24.1–28, GS ≤ 30 For BMI >28, GS ≤ 32	For BMI ≤ 23, GS ≤ 17 For BMI 23.1–26, GS ≤ 17.3 For BMI 26.1–29, GS ≤ 18 For BMI >29, GS ≤ 21
Rapid pace Walk (sec)	>10	>10
Usual pace Walk (sec)	>16	>16

Table A.1: List of deficits in Searle et al. (2008, Tab. 1 and 2)

Note: The individual health deficit index in Searle et al. (2008) is computed by summing up the cut points for an individual and dividing by 40.

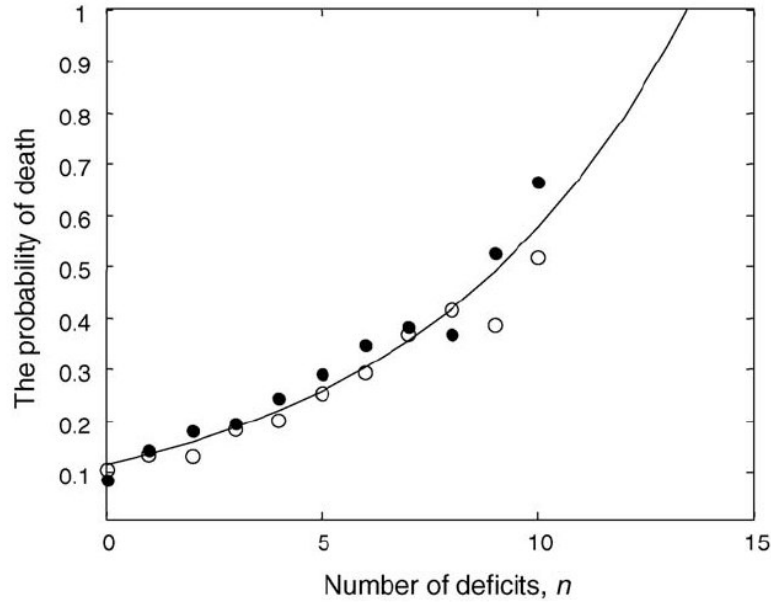


Figure A.1: Mortality rates and the number of health deficits (out of 31 potential deficits) for a cross section of Canadian cohorts aged 65+ from three waves.

Source: Mitnitski, Bao and Rockwood (2006, Fig. 2).

Notes: (1) Data from the Canadian Study of Health and Aging (CSHA), “a representative cohort study designed to study dementia and other age-related problems [...]”. Briefly, in 1990-1991, during the first wave of the study (CSHA-1) 9008 community-dwelling people age 65 and over were assessed using a self-report questionnaire, of whom complete data are available for 5586 survivors for the second wave (CSHA-2, conducted in 1995-1996) and 3211 for the third wave (CSHA-3, conducted in 2000-2001).” Mitnitski et al. (2006, p. 492). (2) Original note: “Probability estimates come from the combined model of CSHA-1 to CSHA-2 (filled circles), and CSHA-2 to CSHA-3 (empty circles). Circles represent observational data and lines show the fit.”

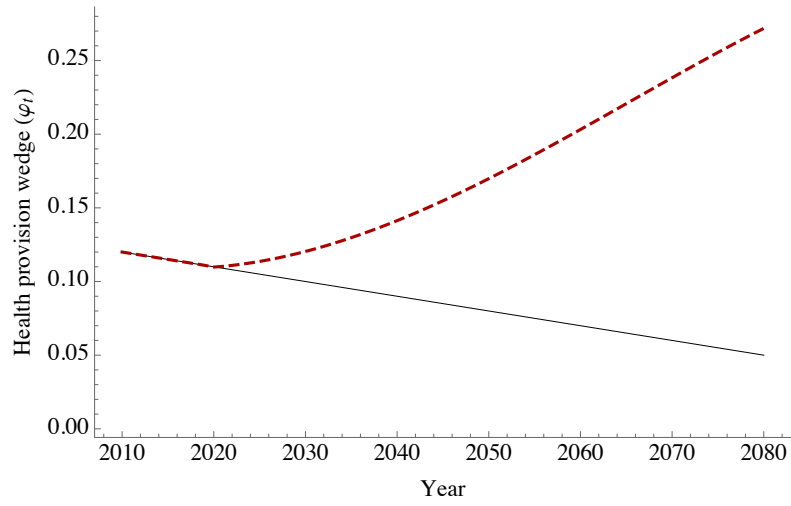


Figure A.2: Calibration of the time paths of the health care wedge from year 2010 onwards in the rationing scenario (dashed line) and baseline scenario (solid line).

Age	1950		1970		1990		2010	
	Data	Model	Data	Model	Data	Model	Data	Model
20	51.8	53.0	53.8	54.4	56.7	56.8	61.0	60.6
25	47.0	48.1	49.0	49.4	51.9	51.9	56.1	55.7
30	42.4	43.2	44.2	44.6	47.1	47.0	51.2	50.8
35	37.8	38.4	39.4	39.8	42.3	42.2	46.4	46.0
40	33.2	33.7	34.7	35.0	37.5	37.5	41.6	41.2
45	28.7	29.1	30.1	30.4	32.8	32.8	36.9	36.5
50	24.4	24.7	25.7	26.0	28.2	28.3	32.3	31.9
55	20.3	20.5	21.6	21.7	23.9	24.0	27.8	27.5
60	16.6	16.6	17.7	17.7	19.8	19.9	23.5	23.3
65	13.2	13.0	14.3	14.1	16.1	16.1	19.4	19.3
70	10.2	9.9	11.3	10.8	12.9	12.6	15.5	15.6
75	7.6	7.2	8.7	8.0	10.0	9.6	12.0	12.2
80	5.6	5.0	6.5	5.7	7.5	7.0	8.9	9.3
85	4.1	3.4	4.8	3.9	5.5	4.9	6.4	6.8
90	3.0	2.1	3.5	2.5	4.0	3.3	4.4	4.8
95	2.2	1.2	2.6	1.5	2.9	2.1	3.1	3.2
100	1.8	0.6	2.0	0.8	2.1	1.2	2.2	2.0

Table A.2: Comparison of remaining period life expectancy according to age: UK data vs calibrated model, for years 1950, 1970, 1990, 2010.

Age	2020	2050		2080	
	Baseline	Baseline	Reform	Baseline	Reform
20 .....	63.6	73.5	72.7	83.7	79.1
25 .....	58.7	68.6	67.9	78.8	74.3
30 .....	53.8	63.8	63.0	74.0	69.4
35 .....	49.0	59.0	58.2	69.2	64.6
40 .....	44.2	54.2	53.4	64.4	59.9
45 .....	39.5	49.4	48.7	59.6	55.2
50 .....	34.8	44.8	44.0	54.9	50.5
55 .....	30.3	40.1	39.4	50.3	46.0
60 .....	26.0	35.6	34.9	45.7	41.5
65 .....	21.9	31.2	30.5	41.2	37.2
70 .....	18.0	26.9	26.3	36.8	33.0
75 .....	14.4	22.9	22.3	32.5	28.9
80 .....	11.2	19.0	18.5	28.4	25.0
85 .....	8.5	15.5	15.0	24.4	21.3
90 .....	6.1	12.3	11.9	20.5	17.8
95 .....	4.3	9.5	9.1	16.9	14.5
100 ....	2.8	7.1	6.8	13.4	11.4

Table A.3: Implied remaining period life expectancies according to age: baseline vs. rationing scenario for years 2020, 2050, 2080.

Age	2020		2050		2080	
	Baseline	Reform	Baseline	Reform	Baseline	Reform
20 .....	86.2	80.3	91.0	80.6	92.8	80.6
25 .....	80.2	75.2	85.5	75.8	87.5	75.7
30 .....	74.2	69.9	80.0	71.0	82.3	70.8
35 .....	68.0	64.4	74.5	66.2	77.0	66.1
40 .....	61.4	58.5	68.9	61.4	71.7	61.4
45 .....	54.6	52.3	63.4	56.6	66.4	56.7
50 .....	47.8	46.0	57.8	51.8	61.1	52.1
55 .....	41.1	39.8	52.2	47.0	55.8	47.6
60 .....	34.7	33.9	46.7	42.3	50.5	43.1
65 .....	28.7	28.2	41.1	37.4	45.3	38.7
70 .....	23.1	22.8	35.5	32.3	40.2	34.4
75 .....	18.1	17.9	29.8	27.3	35.2	30.2
80 .....	13.7	13.6	24.4	22.4	30.3	26.1
85 .....	10.0	10.0	19.5	18.0	25.5	22.0
90 .....	7.0	7.0	15.2	14.0	20.9	18.0
95 .....	4.7	4.7	11.4	10.5	16.5	14.2
100 ....	3.0	3.0	8.2	7.6	12.6	10.8

Table A.4: Implied remaining cohort life expectancies according to age: baseline vs. rationing scenario for years 2020, 2050, 2080.