

Optimal Public Insurance and Health Inequality with Biologically Founded Human Ageing*

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Abstract

This paper integrates into public economics a biologically founded, stochastic process of individual ageing. The novel approach enables us to investigate theoretically and quantitatively the interaction between health and retirement policy for welfare and for health inequality. In particular, we derive the optimal design of the public insurance system behind the veil of ignorance. Calibrating our model to Germany, we find that the health contribution rate and particularly health spending for the elderly may be considerably too low. Implementing the optimal health policy is predicted to lead to more health inequality, however. Our results from the calibrated model also suggest that the statutory retirement age should be raised by one or two years whereas the pension contribution rate should be reduced.

Key words: Ageing; Health Expenditure; Health Inequality; Social Security System; Retirement Age.

JEL classification: H50, I10, C60.

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1 Introduction

Life expectancy of adults increased by around 15 years over the 20th century and many researchers in demography and the natural sciences consider it likely to increase further (e.g. Gavrilov and Gavrilova, 1992; Oeppen and Vaupel, 2002). Fast advances in medical and pharmaceutical research led to substantial increases in the effectiveness of health spending on the ageing process and thus to a substantial increase in life expectancy at retirement age.¹ Through this channel, health spending contributes to the widely discussed problem of social security systems, namely that pension payments are likely to decline for a given contribution rate unless the statutory retirement age is changed. This implies that health and pension policy shall be examined and designed jointly. On the one hand, higher effectiveness of medical technology may make higher contribution rates to health insurance more desirable. On the other hand, one may want to offset its effect on pension payments by raising the retirement age or contributions to the pension system. There is, however, a trade-off between the two tiers of the public insurance system, as contributions to pension insurance and health insurance both reduce net income in the working period. In view of the complex linkages between the pension system and the health care system created by the endogeneity of human health and longevity, the jointly optimal design of our social insurance systems appears to be both important and a priori non-obvious.

This paper investigates the interactions between public health and pension policy to derive the optimal joint design of the public insurance system and its implications for health inequality. In line with a long tradition in public economics to justify social systems, we focus on welfare maximization behind the veil of ignorance. It is based on the idea that an ex ante identical population would agree on a public insurance system which reduces the probability of illness and insures against social hardships and long life. An interesting question in this respect is whether implementing an ex ante optimal public

¹Medical and pharmaceutical innovations became important drivers of life expectancy from the 1950s onwards. Before that, life expectancy rose predominantly because of decreases in child mortality rather than because of increases in life expectancy of adults (e.g. Milligan and Wise, 2011). In the US, the fraction of the population which is at least 65 years old is projected to be 18.8 percent in 2025, whereas it was 8.1 percent in 1950 (Poterba, 2014).

insurance system will reduce health inequality within a society compared to the status quo. In fact, reducing health inequality is a major goal of large organizations like the World Health Organization (WHO) and the European Union (EU).² It is however non-obvious whether the goal to improve health equality is in line or in conflict with the goal to maximize ex ante welfare.

Our key innovation which enables us to examine these issues is to integrate into public economics a biologically founded process of individual ageing. Ageing is understood as the stochastic and individual-specific deterioration of the functioning of body and mind (i.e., an accumulation of health deficits) that eventually culminates in death (Arking, 2006; Masoro, 2006). Our approach is based on empirical evidence from gerontology³ which suggests that (i) at any given age, the number of health deficits is approximately Poisson-distributed in the population, (ii) the average number of individual health deficits grows with age, and (iii) the probability of death strongly depends on the number of health deficits an individual accumulates over time (Mitnitski and Rockwood, 2002a, 2002b, 2005).

A salient feature of our analysis is that, insofar as health expenditures targeted to the working-aged affect the distribution of health deficits in this group, they also affect the distribution of health deficits among retirees. Improving health of the working-aged affects pension finance for two reasons. First, because it raises the productivity of workers and their contributions to the public insurance systems and, second, because it determines life expectancy for individuals at retirement age. Accordingly, we distinguish health care expenses for the working-aged from health expenditure targeted to typical illnesses of the elderly. Examples of the former would be expenses for mass examinations of the health status of pupils at schools, costs for educational health campaigns (about nutrition, usage of soft drugs, prevention of HIV infections etc.), and, in particular, expenses for treating

²See www.who.int and www.health-inequalities.eu/. According to the WHO, health inequality is defined as "differences in health status or in the distribution of health determinants between different population groups".

³Modern gerontology tries to explain human ageing by employing basic insights and mechanisms from reliability theory, which describes the human organism as a complex, redundant system (Gavrilov and Gavrilova, 1991). The notion of ageing as accelerated loss of organ reserve is in line with the mainstream view in the medical science. For example, initially, as a young adult, the functional capacity of human organs is estimated to be tenfold higher than needed for survival (Fries, 1980).

health problems and curing illnesses which typically also hit younger adults (like type 1 diabetes, virus infections, bacterial infections, orthopedic issues after accidents, psychiatric problems). Examples of the latter would be expenses to treat cardiovascular diseases, type 2 diabetes, cancer, stroke, lung disease, and arthrosis, affecting the distribution of health deficits of retirees conditional on the distribution of health deficits of the working-aged. We also measure health inequality of workers and retirees separately by the Gini coefficient of these distributions.

After reviewing the related literature in section 2, we develop in section 3 a theoretical model which is based on evidence from gerontology to highlight the fundamental interactions between public pensions and health spending targeted to working-aged individuals and retirees. In section 4, we analytically characterize the optimal mix of policy instruments in the public insurance system by deriving the interactions of public insurance policy instruments for welfare. For simplicity, we first abstract from the stochastic nature of the ageing process. Section 5 then calibrates our stochastic framework to Germany, which is characterized by a public health care system and a public pay-as-you-go pension system. As private insurance for health purposes and private old age savings quantitatively play a rather minor role,⁴ Germany is a prime candidate for examining the optimal design of a public insurance system.⁵ Section 6 then conducts numerical analysis of the optimal joint design of both health and pension policy. The last section concludes.

Our main findings may be summarized as follows. In the analytical part, after establishing the link between health spending targeted to retirees and pension payments through life expectancy, we show that the optimal allocation of health spending typically does not maximize life expectancy. The reason is that maximizing life expectancy may conflict with the goal to receive high contributions to the pension system of a productive workforce. Calibrating the model to Germany suggests that, somewhat surprisingly, the status quo health system may be severely underfinanced particularly with respect to the

⁴Retired households in Germany receive about 80 percent of income from social security (Boersch-Supan and Schnabel, 1998).

⁵The scope of our study certainly extends to other advanced countries. For instance, in the US, social security is the most important source of support of retirees for the bottom half of the income distribution (Poterba, 2014). Financial assets outside retirement accounts play a minor role for the vast majority of households.

health of retirees. Raising health spending targeted to old age diseases, however, increases health inequality (as measured by the Gini coefficient of health deficits) among retirees. The reason is that the bulk of working-aged individuals enter the retirement age relatively healthy and higher health spending targeted to the elderly shifts the distribution of health deficits to the left. Most of our results from the calibrated model also suggest that the statutory retirement age in Germany should be slightly increased, whereas the pension contribution rate should be decreased compared to the status quo.

2 Related Literature

In order to measure human functionality the medical science has proposed several indices of human capability or disability. The theory and calibration approach in the present paper is based on the so-called *frailty index*, which is particularly related to reliability theory (Gavrilov and Gavrilova, 1991).⁶ The frailty index counts for a large sample of individuals the bodily impairments which are actually present out of a long list of potential impairments, ranging from mild deficits (reduced vision, incontinence) to near lethal ones (e.g. stroke). The evidence suggests that the frailty index of an individual correlates exponentially with age, that at any given age the number of deficits in a given population is approximately Poisson-distributed, and that the probability of death strongly depends on the number of health deficits that one has accumulated over time (e.g. Mitnitski and Rockwood, 2002a, 2002b, 2005). Associating health status with a simple count measure of health deficits is thus both appealingly simple and empirically successful. 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.

Another important insight from gerontology for the present paper is that individual deficit accumulation is path-dependent. Transitions in health status can be very accurately described by a Markov-chain augmented Poisson law according to which the probability to get another health deficit next period depends positively on the number of

⁶Strulik and Vollmer (2013) used the reliability approach of Gavrilov and Gavrilova (1991) to empirically investigate its implications for long-run trends of human ageing and longevity.

already accumulated health deficits (Mitnitski et al., 2006, 2007a, 2007b). This fact makes the simultaneous investigation of health and pension policy interesting and challenging.

Notwithstanding the advances in the natural sciences to understand life cycle health, the common conceptualization of health in economics is still based on the Grossman (1972) model.⁷ The basic idea of the Grossman model is that individuals accumulate health through investment in health capital, similar to the accumulation of human capital through investment in education. Without further amendments this means that desired health expenditure drops at the point of retirement and that health depreciation is greater when the stock of health is large, that is when individuals are relatively young and healthy. Preserving health would thus require health expenditure to be high at working age and low at old age (for a critique, see also Case and Deaton, 2005). In order to counteract this problem, the literature has assumed that the health depreciation rate is increasing with age. In contrast, modern gerontology suggests that individuals, as they age, do not accumulate health capital but health deficits.

Dalgaard and Strulik (2014) integrated into life cycle economics the notion of health deficit accumulation to understand the association between income and longevity. The model has also been applied to examine the education gradient in health and life expectancy (Strulik, 2011) and the long-run evolution of retirement behavior (Dalgaard and Strulik, 2012). So far, however, the theory was confined to life cycle decisions of a single agent. In the present paper we integrate physiological ageing into a general equilibrium context with public insurance policy. We will first investigate the links between health and pension policy for public insurance budgets and their welfare effects. We then draw from these insights in order to characterize the optimal (i.e. welfare-maximizing) policy design.

There exists a relatively large literature discussing the impact of social security on labor supply and retirement and on the optimality or sustainability of public pension systems (e.g. Auerbach and Kotlikoff, 1987; Imrohorglu et al., 1995; Boersch-Supan, 2000; Jaag et al., 2010, 2011; see Liebman and Feldstein, 2002, for a survey). Particularly related to

⁷The basic Grossman framework has been extended in various directions (e.g. Ehrlich and Chuma, 1990; Hall and Jones, 2007).

our paper is the study by Conesa and Krueger (1999) who like us study welfare effects of social security reform for an economy in which heterogenous individuals face a priori uncertainty about their ability (productivity). The interaction of pension finance and health care, however, is not investigated. Sinn (1995) showed that income redistribution is desirable by increasing risk-taking of expected utility maximizing individuals behind the veil of ignorance, i.e. before idiosyncratic ability is revealed. Conesa, Kitao and Krueger (2009) have used this concept in a macro model with idiosyncratic ability of workers and a social security system. The focus of their study, however, was not on optimal social security provision but on optimal income taxation.

While most of the conventional public pension literature ignores issues of health and longevity, there exists a smaller literature investigating the impact of health on labor supply and retirement when health is exogenous (Philipson and Becker, 1998; French, 2005; Heijdra and Romp, 2009; French and Jones, 2011; Imrohroglu and Kital, 2012; Bloom, Canning and Moore, 2014) and when it is endogenously determined via the Grossman model of health capital accumulation (Wolfe, 1985; Galama et al., 2013). In Wolfe (1985), however, retirement is not determined by way of welfare maximization, and in Galama et al. (2009) longevity is not affected by health investment. Philipson and Becker (1998) investigate a life cycle model with given retirement age, longevity enhancing health expenditure and (public) annuities. They argue that retired individuals demand too much health care because they do not take into account the effect of their longevity increasing behavior on the annuity level. They thus decide to live inefficiently long rather than to live well. Heijdra and Romp (2009) analyze the impact from pension reform in a general equilibrium setting, in the presence of a realistic – but exogenously given – mortality process. Bloom, Canning and Moore (2014) develop a life cycle model and use it to gauge the impact of changes in income and life expectancy on age of retirement. Calibration to the US suggests that the optimal retirement age decreases because of an income effect when wages grow despite increases in longevity. Health and longevity, however, are exogenously given.

Pestieau, Ponthiere and Sato (2008) take up this idea and argue that private health spending should be taxed when the replacement rate is sufficiently large. Leroux, Pestieau

and Ponthiere (2011a,b) extend the model towards heterogenous agents who differ in their (genetically determined) probability of survival to retirement age. They show that optimal redistribution goes from high-productivity to low-productivity agents and from short-lived to long-lived individuals.

While the available studies point to some interesting interaction of health and public policy they ignore important other channels. Most importantly the available literature focussed on the probability to reach an exogenously given retirement age and ignored the effect of health on longevity, i.e. the years spent in retirement, a fact that was probably driving the result of optimal redistribution from the unhealthy to the healthy individuals. The available literature also did not take into account that idiosyncratic health endowments and health care during the working age of the population affects productivity and income and therewith the desired age of retirement. In particular, the path-dependency of health in working age and health in old age, emphasized in the gerontological literature, remained unexplored. In this paper, we aim to overcome these shortcomings. We also attempt to fill the gap in the existing literature to answer the question how health spending should be allocated over the life-cycle and how the health care allocation interacts with retirement age and pension benefits.

3 The Model

Consider the following continuous-time model. At each date t , a new cohort of ex ante identical individuals is born. The cohort size is time-invariant and normalized to unity. This assumption reflects our focus on the effects of ageing on the social insurance system caused by higher life expectancy rather than by (presumably temporary) changes in the birth rate.

Life consists of a working period and a retirement period. Ageing is stochastic in the sense that the deterioration process of health, and thus life-time, is stochastic. Hence, the length of the retirement period in terms of calendar time is uncertain and possibly different to the length of the working period. The statutory retirement age (i.e. the length of the working period for those who survive until then) is denoted by \bar{R} . The

government provides a health care system and a pension system to maximize ex ante welfare behind the veil of ignorance. Labor income is taxed at a constant rate $\tau \in (0, 1)$ in order to finance health expenditure and at a constant savings rate $s \in (0, 1)$ in order to finance pension payments. Both systems are pay-as-you-go (PAYG), i.e. the tax revenues are paid out contemporaneously. Public budgets are balanced each period. The pension system is such that relative contributions between individuals of the same cohort to the system during the working period are reflected by relative payments during retirement in each point of time. There are no frictions in the system and no other taxes.

We abstract from private forms of health expenditure and pension insurance. Specifically, a private annuity market is missing and individuals cannot save privately for the retirement period. This captures, albeit in extreme form, the little importance of private savings for retirement wealth for the vast majority of households, for instance, in Germany (Boersch-Supan and Schnabel, 1998) to which we calibrate our model. The public pension system (Social Security) is an important source of retirees' income in the US as well (Poterba, 2014). Allowing for private pension savings to complement public insurance would enhance analytical complexity to the point of intractability in the case where lifetime is uncertain. Assuming non-optimizing households is consistent with evidence from behavioral economics showing that most individuals stick to default pension plans offered by their employers.⁸ Such evidence widely opens the scope for public policy, as discussed by Beshears et al. (2009), who survey the literature. Inter alia they point to evidence by Cronqvist and Thaler (2004) who show that the rate of return of the default portfolio in the Swedish social security system was higher than the performance of individuals who opted out of the default and selected the portfolio of assets by themselves.

⁸In an interesting recent paper, Caliendo and Findley (2013) derive the optimal social security provision in the US by analyzing a calibrated model in which individuals save an exogenous fraction of their disposable income. Under such non-optimizing behavior, the current size of the US social security program is supported.

3.1 Production

At each date, there is a single homogenous consumption good which is produced according to a neoclassical, constant-returns-to-scale production technology. Output Y is given by

$$Y = F(K, AL) \equiv ALf(k), \quad k \equiv \frac{K}{AL}, \quad (1)$$

where K and L are the inputs of physical capital and labor, the latter being measured in efficiency units. A is an exogenous measure of productivity. $f(\cdot)$ is strictly increasing, strictly concave, and fulfills the Inada conditions.

Output is sold in a perfectly competitive environment. The output price is normalized to unity. The rate of return to capital, r , is internationally given (i.e. we consider a small open economy) and time-invariant. Thus, profit maximization of the representative firm implies that k is given by $r = f'(k)$, i.e. $k = (f')^{-1}(r) \equiv \bar{k}(r)$. Consequently, the wage rate per efficiency unit of human capital reads as $w = A\omega$, with $\omega \equiv f(\bar{k}(r)) - \bar{k}(r)f'(\bar{k}(r))$.

3.2 Individuals

Individuals are indexed by i . During the working period, individuals inelastically supply $l(i)$ units of labor in each year. $l(i)$ may adversely depend on the number of health deficits during the working period (indexed by 1), $n_1(i) \in S \subseteq \mathbb{N}$. Moreover, it is a non-decreasing function of the productivity-adjusted net wage rate, $\varpi(\tau, s) \equiv (1 - \tau - s)\omega$. We write

$$l(i) = \varpi(\tau, s)^\varepsilon \tilde{l}(n_1(i)), \quad (2)$$

where \tilde{l} is a non-increasing and convex function and $\varepsilon \geq 0$ is the labor supply elasticity. We distinguish the cases $\tilde{l}' = 0$ (no impact of health status on labor supply) and $\tilde{l}' < 0$. The case $\tilde{l}' < 0$ is consistent with evidence provided by Cai, Mavromaras and Oguzoglu (2014) showing that individuals who experience moderate health shocks respond by incremental reductions in labor supply. For simplicity, we neglect capital income of individuals (assuming that it accrues to foreigners). According to (2) and $w = A\omega$, net wage income of an individual i at each point in time during the working period, $C_1(i) \equiv (1 - \tau - s)wl(i)$,

is given by

$$C_1(i) = A\varpi(\tau, s)^{1+\varepsilon}\tilde{l}(n_1(i)) \equiv \tilde{C}_1(n_1(i), \tau, s). \quad (3)$$

Let $n_2(i) \in S$ be the health deficits of retiree i , provided the individual reaches the retirement age. This is the case if i has sufficiently few health deficits in the working period. Let $\tilde{T}(n)$, $n \in S$, be a strictly decreasing function with the following interpretation. Individual i reaches (does not reach) the retirement age if $\tilde{T}(n_1(i)) \geq (<) \bar{R}$. If i does not reach retirement age, lifetime is given by $\tilde{T}(n_1(i))$. If i reaches retirement age, lifetime is $\max(\bar{R}, \tilde{T}(n_2(i)))$. Let $\bar{S} \equiv \{n \in S : n > \tilde{T}^{-1}(\bar{R})\}$ and $\underline{S} \equiv \{n \in S : n \leq \tilde{T}^{-1}(\bar{R})\}$; $S = \underline{S} \cup \bar{S}$. In sum, individual lifetime, $T(i)$, is negatively depends on the number of individual health deficits (Mitnitski et al., 2005, 2007) and is given by⁹

$$T(i) = \begin{cases} \tilde{T}(n_1(i)) & \text{if } n_1(i) \in \bar{S}, \\ \bar{R} & \text{if } n_1(i) \in \underline{S} \text{ and } n_2(i) \in \bar{S}, \\ \tilde{T}(n_2(i)) & \text{otherwise.} \end{cases} \quad (4)$$

Lifetime is finite even without any health deficits during retirement. The healthiest retiree dies at age $T_{\max} \equiv \tilde{T}(0) < \infty$. The individual length of the working period, $R(i)$, is given by

$$R(i) = \tilde{R}(n_1(i), \bar{R}) \equiv \begin{cases} \tilde{T}(n_1(i)) & \text{if } n_1(i) \in \bar{S}, \\ \bar{R} & \text{if } n_1(i) \in \underline{S}. \end{cases} \quad (5)$$

Individuals derive utility from material consumption and disutility from labor market participation. We also allow for the case in which health status, as captured by the number of health deficits during working age, has a direct impact on utility. Life-time utility of an individual i reads as

$$U(i) = \int_0^{T(i)} e^{-\rho t} u(c(i, t)) dt - V(R(i), n_1(i)), \quad (6)$$

⁹The two-period set up in continuous time may implies that a non-zero mass of individuals dies exactly at statutory retirement age \bar{R} . According to (4), an individual surviving to retirement age may experience a health shock and immediately die after reaching the statutory retirement age. In the numerical analysis, reasonably, the mass of individuals dieing exactly at age \bar{R} will be negligible.

where $\rho \in (0, 1]$ is the discount rate, t indexes (calendar) time, $c(i, t)$ is consumption of individual i at time t , u is the instantaneous consumption utility function, and V represents the disutility from working (along the extensive margin), possibly dependent on health deficits at working age. We assume that $u(c)$ is increasing and strictly concave. $V(R, n_1)$ is increasing and convex as a function of the length of the working period, R , and non-decreasing and convex in the number of health deficits during the working period, n_1 . Finally, suppose that V has weakly increasing differences, i.e., if anything, a marginal increase in the length of the working period has a larger impact on the disutility of work when the worker is less healthy; formally, we assume that $V(R, n'_1) - V(R, n_1)$ is non-decreasing in R whenever $n'_1 > n_1$.¹⁰

Note that health deficits during retirement affect utility only through reducing life expectancy.¹¹ Moreover, we take the case in which V does not depend on health deficits as our benchmark in the numerical analysis. We deliberately focus on these cases to obtain a lower bound for the welfare-maximizing level of health spending in Germany, to which we calibrate our model. If we find that the status quo health spending is too low even in the case where health status has no non-material effect on utility, as assumed also in Becker (2007), then there is a strong argument to increase health spending.

Individuals rely on the pension system for retirement income and consume their net wage income during the working period, i.e.

$$c(i, t) = \tilde{C}_1(n_1(i), \tau, s) \text{ for all } t \in [0, R(i)]. \quad (7)$$

The government pays out an individual-specific and time-invariant pension income $C_2(i)$ during the retirement period (indexed by 2), as derived below; it thereby provides perfect consumption-smoothing during the retirement age as well. Pension income is not taxed.

¹⁰Under differentiability, the assumption of weakly increasing differences of disutility function V means that $V_{nR} \geq 0$, where subscripts on V denote partial derivatives throughout.

¹¹Relaxing this assumption would not affect our analytical results in sections 3 and 4.

3.3 Evolution of Health Deficits

Health spending is measured in terms of the numeraire good. We distinguish between health spending targeted to the working-aged population, H_1 (e.g. for prevention programmes and curative care for illnesses which typically also hit younger adults like virus infections and psychiatric problems) and health spending targeted to retirees, H_2 (e.g. for treating illnesses typically related to old age like cardiovascular diseases, cancer and arthrosis).

In line with empirical evidence, the number of health deficits is Poisson-distributed in both periods of life. Let

$$g(n_j, \lambda_j) = \exp(-\lambda_j)\lambda_j^{n_j}/n_j! \quad (8)$$

denote the probability density function (p.d.f.) of health deficits in period of life $j \in \{1, 2\}$. The Poisson parameters λ_1 and λ_2 (the average number and variance of health deficits in period 1 and 2, respectively, depend on the productivity-adjusted health spending levels $h_1 \equiv H_1/A$ and $h_2 \equiv H_2/A$ in period 1 and 2, respectively; they are given by

$$\lambda_1 = \tilde{a}_1(h_1), \quad (9)$$

$$\lambda_2 = \tilde{a}_2(h_2) + bn_1 \quad (10)$$

where \tilde{a}_j is a function with properties $\tilde{a}'_j < 0$ and $\tilde{a}''_j > 0$, $j \in \{1, 2\}$. $b > 0$ is a parameter which is independent of health spending. It captures that the number of health deficits in retirement age, n_2 , is path-dependent in a stochastic sense. That is, the distribution of n_2 is conditional on n_1 . Using (9) and (10) in (8), the joint p.d.f. of (n_1, n_2) is given by

$$G(n_1, n_2, h_1, h_2) \equiv g(n_1, \tilde{a}_1(h_1))g(n_2, \tilde{a}_2(h_2) + bn_1). \quad (11)$$

According to (4), life expectancy at birth is increasing in health spending and reads as

$$\begin{aligned} \text{LE} = & \sum_{n_1 \in \bar{S}} g(n_1, \tilde{a}_1(h_1)) \tilde{T}(n_1) + \bar{R} \sum_{n_1 \in \underline{S}} \sum_{n_2 \in \bar{S}} G(n_1, n_2, h_1, h_2) + \\ & \sum_{n_1 \in \underline{S}} \sum_{n_2 \in \underline{S}} G(n_1, n_2, h_1, h_2) \tilde{T}(n_2). \end{aligned} \quad (12)$$

Four remarks are in order. First, according to (9) and (10), health services can either be interpreted as public goods or (recalling that cohort size is normalized to unity) as private goods for which per capita health spending at the beginning of the respective period of life matters. Second, to maintain the amount of health services after an increase in total factor productivity, A , health spending has to increase proportionally with A . Because the capital-labor ratio in the final goods sector is constant and thus both final output and the wage rate are proportional to A , the underlying health technology could be interpreted as employing either the final good or labor (or both) as input. Third, the convexity assumptions $\tilde{a}_1'' > 0$, $\tilde{a}_2'' > 0$, capture the notion that the return of higher health expenditure on the reduction of health deficits is strictly decreasing. Finally, the path-dependency of health deficits is consistent with overwhelming evidence from gerontology which suggests that the probability to get another health deficit next period depends positively on the number of already accumulated health deficits, according to a Markov-chain augmented Poisson law (Mitnitski et al., 2006, 2007a, 2007b).

3.4 Government Budget Constraints

3.4.1 Health Expenditure

Using (3), the government budget constraint for health spending (financed by wage income at rate τ) is given by $H_1 + H_2 = \tau w \sum_{n_1 \in S} g(n_1, \lambda_1) \tilde{R}(n_1, \bar{R}) \varpi(\tau, s)^\varepsilon \tilde{l}(n_1)$. Thus, using $\lambda_1 = \tilde{a}_1(h_1)$ and $w = A\omega$, we write

$$h_2 = \tau \omega \varpi(\tau, s)^\varepsilon \sum_{n_1 \in S} g(n_1, \tilde{a}_1(h_1)) \tilde{R}(n_1, \bar{R}) \tilde{l}(n_1) - h_1 \equiv \tilde{h}_2(h_1, \tau, s, \bar{R}). \quad (13)$$

Lemma 1. *Function \tilde{h}_2 is increasing in the statutory retirement age, \bar{R} . If $\varepsilon > (=)0$, an increase in pension savings rate, s , reduces (does not affect) \tilde{h}_2 . If $\tilde{l}' < 0$, the impact of an increase in productivity-adjusted health spending for the working-aged, h_1 , on \tilde{h}_2 is generally ambiguous (negative if $\tilde{l}' = 0$ and $\bar{S} = \emptyset$).*

Proof. Follows from (13), assumption $\tilde{a}'_1 < 0$, and the definition of \tilde{R} in (5). ■

The impact of a higher statutory retirement age, \bar{R} , on health resources is positive since the number of contributors (i.e. the size of the working-aged population), rises with \bar{R} . A higher health contribution rate, τ , raises revenue for health spending under (A1), i.e. provided that labor taxes are sufficiently low not to run into a Laffer effect. If $\varepsilon > 0$, individuals reduce labor supply in response to a higher pension savings rate, s , in turn reducing revenue in the health system. An increase in productivity-adjusted health spending for the working-aged, h_1 , may decrease or increase the productivity-adjusted spending available for the retirees, h_2 , all other things being equal. On the one hand, for a given tax revenue, there is a trade-off between health spending targeted to differently aged populations since both kinds of spending are financed by the same source. On the other hand, however, if h_1 rises, the distribution of health deficits in the working-aged population improves. This may have two positive effects on the health budget available for retirees. First, if h_1 increases, more individuals survive to the retirement period (whenever $\bar{S} \neq \emptyset$). Second, if $\tilde{l}' < 0$, labor supply is rising in h_1 , in turn boosting revenue available for improving the health distribution of retirees. Finally, note that a reasonable policy mix would avoid Laffer effects, such that the health budget shall be enlarged by an increase in the health contribution rate τ .

3.4.2 Pensions

We next discuss the pension system. Denote by N_1 and N_2 the number of contributing workers and beneficiaries, respectively. In order to relate to the available pension literature, it is useful to consider the "dependency-ratio", defined as the number of beneficiaries

per worker, $D \equiv N_2/N_1$. The expected number of workers reads as

$$N_1 = \sum_{n_1 \in \mathcal{S}} g(n_1, \tilde{a}_1(h_1)) \tilde{R}(n_1, \bar{R}) \equiv \tilde{N}_1(h_1, \bar{R}), \quad (14)$$

where \tilde{N}_1 is non-decreasing in h_1 and increasing \bar{R} . If all individuals reach the retirement age ($\bar{S} = \emptyset$), then $N_1 = \bar{R}$. Using (11), the expected number of retirees, N_2 , can be written as

$$N_2 = \sum_{n_1 \in \underline{\mathcal{S}}} \sum_{n_2 \in \underline{\mathcal{S}}} G(n_1, n_2, h_1, h_2) \left[\tilde{T}(n_2) - \bar{R} \right] \equiv \tilde{N}_2(h_1, h_2, \bar{R}). \quad (15)$$

Because a higher number of health deficits reduces lifetime and health deficits are path-dependent, \tilde{N}_2 is increasing in both h_1 and h_2 . Moreover, holding health spending constant, the number of retirees is decreasing in the statutory retirement age \bar{R} . The "dependency-ratio",

$$D = \frac{\tilde{N}_2(h_1, h_2, \bar{R})}{\tilde{N}_1(h_1, \bar{R})} \equiv \tilde{D}(h_1, h_2, \bar{R}), \quad (16)$$

has the following properties.

Lemma 2. *The dependency ratio function, \tilde{D} , is increasing in (productivity-adjusted) health spending targeted to the elderly, h_2 , and decreasing in the statutory retirement age, \bar{R} . The impact of an increase in h_1 on \tilde{D} is generally ambiguous (positive if $\bar{S} = \emptyset$).*

Proof. Follows from (16) in view of the properties $\partial \tilde{N}_1 / \partial h_1 \geq 0$ (with equality if $\bar{S} = \emptyset$), $\partial \tilde{N}_1 / \partial \bar{R} > 0$, $\partial \tilde{N}_2 / \partial \bar{R} < 0$, $\partial \tilde{N}_2 / \partial h_1 > 0$, and $\partial \tilde{N}_2 / \partial h_2 > 0$. ■

Lemma 2 suggests that health spending typically has a dismal effect on pension finance, by raising life expectancy and increasing the dependency ratio. Only in the empirically implausible case that a substantial mass of individuals would not reach the retirement age, an increase in h_1 may reduce the dependency ratio by helping more people to survive.

As pension payments are proportional to wage income, according to (3), the ratio of the pension payments of two individuals i and i' who reach the retirement period is given

by

$$\frac{C_2(i)}{C_2(i')} = \frac{\tilde{l}(n_1(i))}{\tilde{l}(n_1(i'))}. \quad (17)$$

Denote by C_2^{\max} the pension payment for an individual with no health deficits during the working period and denote by $\ell_0 \equiv \tilde{l}(0)$ the labor supply of such an individual. According to property (17),

$$C_2(i) = \tilde{l}(n_1(i)) \frac{C_2^{\max}}{\ell_0}. \quad (18)$$

In a PAYG pension system, the total revenue from the pension contributions (financed by wage income at rate s) must equal the aggregate expenses. Thus, using expression (2) for individual labor supply and (18) for the expenditure side,

$$sw \sum_{n'_1 \in \mathcal{S}} g(n'_1, \lambda_1) \tilde{R}(n'_1, \bar{R}) \varpi(\tau, s)^\varepsilon \tilde{l}(n'_1) = \sum_{n'_1 \in \underline{\mathcal{S}}} \sum_{n'_2 \in \underline{\mathcal{S}}} G(n'_1, n'_2, h_1, h_2) \left[\tilde{T}(n'_2) - \bar{R} \right] \tilde{l}(n'_1) \frac{C_2^{\max}}{\ell_0}. \quad (19)$$

Solving (19) for C_2^{\max}/ℓ_0 , inserting into (18) and using $\lambda_1 = \tilde{a}_1(h_1)$ implies that pension payments of beneficiary i are given by

$$\begin{aligned} C_2(i) &= \tilde{l}(n_1(i)) \frac{s \varpi(\tau, s)^\varepsilon w \sum_{n'_1 \in \mathcal{S}_1} g(n'_1, \tilde{a}_1(h_1)) \tilde{R}(n'_1, \bar{R}) \tilde{l}(n'_1)}{\sum_{n'_1 \in \underline{\mathcal{S}}} \sum_{n'_2 \in \underline{\mathcal{S}}} G(n'_1, n'_2, h_1, h_2) \left[\tilde{T}(n'_2) - \bar{R} \right] \tilde{l}(n'_1)} \\ &\equiv \tilde{C}_2(n_1(i), h_1, h_2, \tau, s, \bar{R}). \end{aligned} \quad (20)$$

Proposition 1. *The PAYG pension payment function \tilde{C}_2 is increasing in the statutory retirement age, \bar{R} . If $\varepsilon > (=) 0$, \tilde{C}_2 is decreasing in (independent of) the health contribution rate τ . \tilde{C}_2 is decreasing in health spending targeted to retirees, h_2 . The effect of an increase in health spending targeted to the working-aged, h_1 , on \tilde{C}_2 is generally ambiguous (negative if $\tilde{l}' = 0$ and $\bar{\mathcal{S}} = \emptyset$).*

Proof. Follows from (20) in view of $\tilde{a}'_1 < 0$, $\tilde{a}'_2 < 0$, and (5). ■

An increase in the statutory retirement age, \bar{R} , raises pension payments by decreasing the dependency ratio, all other things being equal. Pension payments are decreasing in the health contribution rate, τ , if the wage elasticity of labor supply is positive ($\varepsilon > 0$).

In this case, an increase in τ lowers contributions to the pension system. Proposition 1 particularly highlights the interaction between health spending and pension finance. An increase in old-age health care spending, h_2 , raises life-expectancy and lowers pension payments per retiree. By contrast, an increase in health care spending for workers, h_1 , may as well boost pension payments. It raises labor supply (if $\tilde{l}' < 0$) and helps that fewer individuals die before they reach retirement age (if $\bar{S} \neq \emptyset$). Both effects increase the contributions to the pension system. However, these positive effects need to dominate the effect originating from the path-dependency of health deficits: if the average number of health deficits prior to retirement is reduced by raising h_1 , life-expectancy at retirement age increases, which in turn raises the dependency ratio. Finally, because a reasonable policy mix would avoid Laffer effects, pension payments are increasing in the pension savings rate s .

Using (7) in (6) and observing (3) and (20), utility of individual i can be written as

$$U(i) = \begin{cases} \hat{U}(n_1(i), \tau, s) & \text{if } n_1(i) \in \bar{S} \\ \check{U}(n_1(i), \tau, s, \bar{R}) & \text{if } n_1(i) \in \underline{S} \text{ and } n_2(i) \in \bar{S} \\ \tilde{U}(n_1(i), n_2(i), h_1, h_2, \tau, s, \bar{R}) & \text{otherwise,} \end{cases} \quad (21)$$

where

$$\hat{U}(n_1, \tau, s) \equiv \left(1 - e^{-\rho \tilde{T}(n_1)}\right) \frac{u(\tilde{C}_1(n_1, \tau, s))}{\rho} - V(\tilde{T}(n_1), n_1), \quad (22)$$

$$\check{U}(n_1, \tau, s, \bar{R}) \equiv \left(1 - e^{-\rho \bar{R}}\right) \frac{u(\tilde{C}_1(n_1, \tau, s))}{\rho} - V(\bar{R}, n_1), \quad (23)$$

$$\begin{aligned} \tilde{U}(n_1, n_2, h_1, h_2, \tau, s, \bar{R}) \equiv & \frac{1 - e^{-\rho \bar{R}}}{\rho} u(\tilde{C}_1(n_1, \tau, s)) - V(\bar{R}, n_1) + \\ & \frac{e^{-\rho \bar{R}} - e^{-\rho \tilde{T}(n_2)}}{\rho} u(\tilde{C}_2(n_1, h_1, h_2, \tau, s, \bar{R})). \end{aligned} \quad (24)$$

Welfare behind the veil of ignorance then reads as

$$\begin{aligned}
W(h_1, h_2, \tau, s, \bar{R}) \equiv & \sum_{n_1 \in \bar{S}} g(n_1, \tilde{a}_1(h_1)) \hat{U}(n_1, \tau, s) + \sum_{n_1 \in \underline{S}} \sum_{n_2 \in \bar{S}} G(n_1, n_2, h_1, h_2) \check{U}(n_1, \tau, s, \bar{R}) + \\
& \sum_{n_1 \in \underline{S}} \sum_{n_2 \in \underline{S}} G(n_1, n_2, h_1, h_2) \tilde{U}(n_1, n_2, h_1, h_2, \tau, s, \bar{R}). \tag{25}
\end{aligned}$$

The optimal policy mix is given by maximizing (25) subject to (13), $h_2 = \tilde{h}_2(h_1, \tau, \bar{R})$.

4 Optimal Policy and Welfare Under Certainty

We next attempt to characterize the interaction between policy instruments for welfare analytically. For this part, we simplify by abstracting from uncertainty, i.e. all individuals are identical also ex post and reach the retirement age. The results derived in this section guide us to interpret the quantitative results of section 5, in which health will be again stochastically determined.

As $R(i) = \bar{R}$ for all i and cohort size is normalized to unity, the mass of working-aged individuals is $N_1 = \bar{R}$. Under certainty, with a degenerated distribution function g , the number of health deficits of each individual in period 1 and 2 of life equals

$$n_1 = a_1 = \tilde{a}_1(h_1), \tag{26}$$

$$n_2 = a_2 + bn_1 = \tilde{a}_2(h_2) + b\tilde{a}_1(h_1), \tag{27}$$

respectively. The relationship between health spending for the working-aged and for retirees reads as

$$h_2 = \tilde{h}_2(h_1, \tau, s, \bar{R}) = \bar{R}\omega\tau\varpi(\tau, s)^\varepsilon \tilde{l}(\tilde{a}_1(h_1)) - h_1, \tag{28}$$

according to (13) and (26). Substituting (28) into (27) leads to

$$n_2 = \tilde{a}_2(\tilde{h}_2(h_1, \tau, s, \bar{R})) + b\tilde{a}_1(h_1) \equiv \tilde{n}_2(h_1, \tau, s, \bar{R}). \tag{29}$$

According to (3) and (26), net income and thus consumption of working-aged individuals at each date is given by

$$C_1 = A\varpi(\tau, s)^{1+\varepsilon}\tilde{l}(\tilde{a}_1(h_1)). \quad (30)$$

Moreover, under certainty, $l(i) = \varpi(\tau, s)^\varepsilon\tilde{l}(n_1)$ for all i , according to (2). Thus, the budget constraint for pension payments in the PAYG system (aggregate expenses equal aggregate contributions) can be written as $N_2C_2 = N_1sw\varpi(\tau, s)^\varepsilon\tilde{l}(n_1)$, where the number of worker and retirees are given by $N_1 = \bar{R}$ and $N_2 = \tilde{T}(n_2) - \bar{R}$, respectively. Using (26) and (29), we thus find that the pension payment (and consumption) per retiree at each date is given by

$$C_2 = \frac{\bar{R}ws\varpi(\tau, s)^\varepsilon\tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(\tilde{n}_2(h_1, \tau, s, \bar{R})) - \bar{R}}. \quad (31)$$

According to (6) and $T = \tilde{T}(n_2)$, individual welfare is given by $U = (1 - e^{-\rho\bar{R}})u(C_1)/\rho + (e^{-\rho\bar{R}} - e^{-\rho\tilde{T}(n_2)})u(C_2)/\rho - V(\bar{R}, n_1)$. Using (30), (31), (26) and (29) we thus obtain

$$U = \frac{1 - e^{-\rho\bar{R}}}{\rho}u\left(A\varpi(\tau, s)^{1+\varepsilon}\tilde{l}(\tilde{a}_1(h_1))\right) - V(\bar{R}, \tilde{a}_1(h_1)) + \frac{e^{-\rho\bar{R}} - e^{-\rho\tilde{T}(\tilde{n}_2(h_1, \tau, s, \bar{R}))}}{\rho}u\left(\frac{\bar{R}ws\varpi(\tau, s)^\varepsilon\tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(\tilde{n}_2(h_1, \tau, s, \bar{R})) - \bar{R}}\right) \equiv \hat{U}(h_1, \tau, s, \bar{R}). \quad (32)$$

Subscripts on \hat{U} and V will denote partial derivatives. A social planner sets policy parameters to solve

$$\max_{h_1 \geq 0, \tau \in [0, 1], s \in [0, 1], \bar{R} \in [0, T_{\max}]} \hat{U}(h_1, \tau, s, \bar{R}). \quad (33)$$

Let $\mathbf{z}^* = (h_1^*, \tau^*, s^*, \bar{R}^*)$ denote the maximizers of optimization problem (33) and define the resulting health spending targeted to the retirees by $h_2^* \equiv \tilde{h}_2(\mathbf{z}^*)$. To avoid only mildly interesting discussions about potential corner solutions, we focus our analysis on interior solutions of (33). First, we deal with the question whether the optimal allocation of health spending towards working-aged and retired individuals, (h_1^*, h_2^*) , maximizes lifetime.¹²

Proposition 2. *Suppose that $\mathbf{z}^* = (h_1^*, \tau^*, s^*, \bar{R}^*)$ is an interior maximizer of (33).*

Then the following holds:

¹²For analytical simplicity, we treat health deficits n_1 and n_2 as (non-negative) real numbers rather than as integers in the remainder of this section.

(i) If $\tilde{l}' = V_n = 0$, the optimal allocation of health spending across periods of life maximizes life expectancy and is characterized by $b\tilde{a}'_1(h_1^*) = \tilde{a}'_2(h_2^*)$.

(ii) If $\tilde{l}' < 0$ or $V_n > 0$, health spending targeted to working-aged individuals is higher than under the expenditure structure which maximizes life expectancy.

Proof. See Appendix A. ■

If an increase in health spending targeted to the working-aged has no effect on labor supply ($\tilde{l}' = 0$) and working-aged individuals do not care about health status per se ($V_n = 0$), then the social planner wants to maximize the span of life in which individuals earn retirement income. This is achieved by minimizing health deficits of the elderly, $n_2 = \tilde{n}_2(h_1, \tau, s, \bar{R})$. If $\tilde{l}' < 0$, however, an increase in labor supply that results from an increase in h_1 raises labor income and thus increases revenue from the pension contributions. Hence, it is optimal to sacrifice lifetime to improve consumption in each point of time for both working-aged individuals and retirees. Also if workers have disutility from illness independently of consumption ($V_n > 0$), the social planner biases the health spending structure towards workers. Although this indirectly affects lifetime through the path-dependency of health deficits, the budgetary trade-off in the health system between h_1 and h_2 implies that the structure of health spending does not maximize life expectancy. Proposition 1 would also hold under a constraint optimization problem where pension policy (s, \bar{R}) is treated as given.

We now characterize the optimal policy mix by looking at the interaction between policy instruments on ex ante welfare. To reduce algebraic complexity, we specify labor supply as an iso-elastic function of health deficits in working age,

$$\tilde{l}(n_1) = \ell_0 e^{-\delta n_1}, \quad (34)$$

$\ell_0 > 0$, $\delta \geq 0$, and instantaneous consumption utility by

$$u(c) = \log(c). \quad (35)$$

Log-utility implies that the degree of relative risk aversion equals unity. Evidence by

Chetty (2006), among others, largely supports this value, which we also assume for our baseline calibration below.

Proposition 3. *Suppose that $\mathbf{z}^* = (h_1^*, \tau^*, s^*, \bar{R}^*)$ is an interior maximizer of (33), and (34), (35), $\tilde{T}''(n) \leq \rho \tilde{T}'(n)^2$ hold. Then we have:*

(i) $\hat{U}_{hh}(\mathbf{z}^*) < 0$, $\hat{U}_{\tau\tau}(\mathbf{z}^*) < 0$, and, if $\varepsilon \leq 1$, then $\hat{U}_{ss}(\mathbf{z}^*) < 0$;

(ii) $\hat{U}_{h\tau}(\mathbf{z}^*) > 0$, $\hat{U}_{h\bar{R}}(\mathbf{z}^*) > 0$; $\hat{U}_{hs}(\mathbf{z}^*) = 0$ if $\tilde{l}' = V_n = \varepsilon = 0$ and $\hat{U}_{hs}(\mathbf{z}^*) < 0$ otherwise.

Proof. See Appendix B. ■

The concavity properties in part (i) of Proposition 3 suggest that there exists an interior optimal solution for the joint design of health spending and pension policy. The desirability of retirement presumes that there is disutility of work ($V_{\bar{R}} > 0$). Taken the other policy instruments as given, an interior optimum for the retirement age, \bar{R} , presumes that disutility function V is "sufficiently convex" as a function of \bar{R} .

Part (ii) of Proposition 3 deals with the effects of changes in the health contribution rate (τ) and retirement policy (s, \bar{R}) on the marginal utility of raising health spending targeted to working-aged individuals (h_1). First, $\hat{U}_{h\tau} > 0$ means that sacrificing a higher fraction of labor income for health purposes shall partly be used to raise health spending for workers. The result follows from the decreasing marginal productivity of the health technology (for retirees) and the possible dependency of labor supply on the health status of workers. If an increase in h_1 raises labor supply ($\delta > 0$), it raises contributions to the health system which in turn prolongs lifetime. A higher retirement age, \bar{R} , also improves the government budget for financing the health system. It means that individuals contribute for a longer time. Reducing health deficits of the working-aged thus becomes more valuable. Moreover, suppose that $V(\bar{R}, n_1)$ has increasing differences. In this case, when h_1 is raised, the disutility of late retirement decreases, since healthier individuals suffer less from working. Both considerations explain why $\hat{U}_{h\bar{R}} > 0$. Next consider the interaction between health spending for workers and the pension contribution rate for welfare. The result $\hat{U}_{hs} \leq 0$ means that, typically, an increase in the pension contribution rate s lowers the effect of an increase in h_1 on welfare; it has no impact ($\hat{U}_{hs} = 0$) if

and only if the health expenditure structure maximizes lifetime (Proposition 2) and labor income taxation does not lead to a loss of revenue because of labor supply responses.

We shall note that other cross derivatives of the welfare function are typically ambiguous. For instance, consider the interaction of the pension contribution rate, s , with the health contribution rate, τ , for welfare. On the one hand, raising τ may make an increase in s less worthwhile and vice versa because contributions to the health system and the pension system come from the same source (labor earnings) and the marginal utility of consumption is declining. On the other hand, an increase in τ implies that individuals live longer. All other things equal, particularly holding fixed the retirement age, \bar{R} , this raises the benefit to contribute more to the pension system. In total, the sign of $\hat{U}_{s\tau}$ can be negative or positive. Moreover, a higher retirement age may increase or decrease the effect of higher contribution rates to the health and pension budget. If \bar{R} increases and contribution rates are held fixed, the number of contributors to both tiers of the social insurance system, N_1 , rises. Higher health spending would increase lifetime, such that it is not unambiguously welfare-enhancing if the pension contribution rate is reduced. Higher pension payments make it worthwhile to live longer, such that it is not unambiguously welfare-enhancing if the pension contribution rate is reduced either.

We next investigate a numerically calibrated version of the model in order to disambiguate the theoretical findings and to assess optimal health and pension policies and the welfare implications quantitatively.

5 Calibration for Germany

We calibrate our model for Germany, which has a public PAYG pension system and a public health system with a common health budget for workers and retirees.¹³

We assume that technology (1) has the Cobb-Douglas form $Y = K^\eta(AL)^{1-\eta}$, $\eta \in (0, 1)$. For an exogenous interest rate, r , the wage rate is given by $w = \omega A$, where $\omega = (1 - \eta)(\eta/r)^{\eta/(1-\eta)}$. For later reference, GDP is inferred as $Y = wL/(1 - \eta)$. We

¹³Our approach could also be used in the US context, which explicitly has a health budget for retirees (medicaid expenditure), by assuming that revenue is collected from taxing labor income.

set typical values $\eta = 1/3$ for the output elasticity of capital and $r = 0.06$ for the annual return on capital, consistent with the long run average (e.g. Mehra and Prescott, 1985). Moreover, the productivity parameter is set to $A = 1000$ in order to ensure high enough income for non-negative utility from consumption in old age in any meaningful numerical exercise. As explained below, this ensures that individuals, *ceteris paribus*, always prefer a longer life.

We interpret a unit of calendar time in the model as 45 years. Assuming that people start on average working at age 20, the working period lasts 45 years, which is regarded as the normal earnings history in the German system (*Eck-Rentner*). In terms of our model, the statutory retirement age in Germany is thus captured by $\bar{R} = 1$. For the benchmark run, we set $s = 0.2$ according to the share of gross wages deducted for social security (*Gesetzliche Rentenversicherung*). The pension system is assumed to be self-sufficient. The model assumes that there are no private savings for old age. In the case of Germany this seems to be an acceptable approximation since retired households receive about 80 percent of income from social security (see Boersch-Supan and Schnabel, 1998). Moreover, we set $\tau = 0.155$ such that in line with the data 15.5 percent of gross labor income is paid for public health care insurance (*Gesetzliche Krankenversicherung*).

Let \bar{n} be the maximum number of human health deficits, i.e. $S = \{0, 1, \dots, \bar{n}\}$. We set $\bar{n} = 20$. Our results are independent from the metric of health deficits as long as \bar{n} is high enough.¹⁴ According to (4), life span is a function of the accumulated health deficits; we specify $\tilde{T}(n) = T_{max} \cdot \exp(-\chi \cdot n)$, $\chi > 0$. We set maximum life span T_{max} to $T_{max} = 1.78$, i.e. $20 + 1.78 \cdot 45 = 100$ years.

According to (2), health deficits may affect labor supply, according to (34). Although empirical evidence shows that individuals with poorer health status retire earlier (Gustman and Steinmeier, 2014), Cai et al. (2014) strongly argue that individuals typically respond to health shocks by gradually reducing labor supply rather than opting out fully. They present evidence on the effect of health shocks and health status on the intensive

¹⁴Our results are virtually identical when alternatively setting $\bar{n} = 30$ or $\bar{n} = 40$ (not shown). Interestingly, important statistical relations based on the frailty index are also independent of the number of potential bodily impairments as long as \bar{n} is high enough; see Rockwood and Mitnitski (2007) and Searle et al. (2008).

and the extensive margin. Quantitatively, the bulk of the response to health shocks is at the intensive margin, in line with our model (which ignores the extensive margin for simplicity, unless workers die before reaching the statutory retirement age). According to their Table 1, both men and women with "fair" health (the fourth out of five categories for health status) supply, on average, about 25 percent less working hours than those with "excellent" health (the highest category). Associating "excellent" health with zero health deficits and "fair" health with three health deficits suggests to set δ in (34) to 0.1.¹⁵

Naturally, the labor supply elasticity varies with the concept of the household. According to Bargain, Orsini and Peichl (2014) the labor supply elasticity in Germany (in 2001) is estimated to be 0.14 for men in couples and 0.31 for women in couples. In the benchmark run we set ε to 0.2, which corresponds with the estimated labor supply elasticity for single man in Bargain et al. (2014). It turns out that optimal taxes depend strongly on the elasticity of labor supply. Below we thus provide a sensitivity analysis with respect to ε . We normalize ℓ_0 such that $\tilde{l}(0) = 1$. In the robustness checks we adjust ℓ_0 in order to keep the initial distribution of labor supply from the benchmark case.

Mitnitski et al. (2007) have shown that the intergenerational distribution of deficits can be precisely described by a Poisson process, as captured by (8). The Poisson parameters λ_1 and λ_2 which determine the arrival of new deficits in the two periods of life, are given by (9) and (10), respectively. We specify

$$\tilde{\alpha}_j(h_j) = \alpha_j \cdot \exp(-\beta_j \cdot h_j), \quad \alpha_j > 0, \quad \beta_j \geq 0, \quad (36)$$

$j \in \{1, 2\}$, to capture in a parsimonious way that the arrival rates for new deficits depend on the general health environment, health technology, and – with decreasing returns – on health expenditure. We calibrate the health- and survival-parameters in (36) such that the model approximates actual survival probabilities for each age group. For that purpose we assume that health care expenditure before the 20th century was ineffective in prolonging life of adults (20 years and older), i.e. $\beta_1 = \beta_2 = 0$ for the year 1900 (and

¹⁵For $\delta = 0.1$, $\tilde{l}(3)/\ell_0 = \exp(-0.3) \approx 0.74$. For being consistent with the working hours of those with "poor" health, which are about 75% lower than of those with "excellent" health, when $\delta = 0.1$, $\tilde{l}(n_1)/\ell_0 \approx 0.25$ is reached for $n_1 = 14$.

earlier). This assumption is approximately true. Before the 20th century life-expectancy rose predominantly because of fewer deaths in infancy and childhood. Improving adult life-expectancy is a phenomenon of the 20th century. According to Milligan and Wise (2011), mortality at age 65 did not decline substantially until the 1970s. We use the fact that for ages above 20 the force of mortality, that is the conditional probability $\mu(x)$ to die at age x , is precisely measured by *Gompertz law*, $\mu(x) = B \exp(\phi x)$. Taking the data from the Human Mortality Database (www.mortality.org) we estimate $\phi = 0.11$, $B = 0.00001$ for the year 2000 and $\phi = 0.0092$, $B = 0.00078$ for the year 1950. Unfortunately we do not have mortality data for Germany earlier than 1950. From Strulik and Vollmer (2013) we know that the average Western European values were $\phi = 0.08$, $B = 0.00018$ in 1900. For England and Sweden exists data with a longer range. The average European values in the year 1900 are approximately also observed for England in 1850-1900 and for Sweden in 1750-1900, see Strulik and Vollmer (2013). The constancy of these numbers is consistent with the general observation that adult mortality was very similar in Western Europe and did not change much before the 20th century. We thus set $\phi = 0.08$ and $B = 0.00018$ for 1900 and earlier and $\phi = 0.11$ and $B = 0.00001$ for the year 2000. From these values we compute the unconditional survival probability $S(x)$ by solving $\dot{S}(x)/S(x) = \mu(x)$ for $S(x)$. The result is shown in Figure 1. The solid blue line shows survival rates in 1900, the red dashed line shows survival rates in 2000.

We begin with estimating χ , α_1 , α_2 , and b such that the predicted age-dependent survival probabilities provide the best fit of the actual survival probabilities in the year 1900 (given $\beta_1 = \beta_2 = 0$). This leads to the estimates $\chi = 0.062$, $\alpha_1 = 1.9$, $\alpha_2 = 3.8$ and $b = 2.5$. The estimated survival probabilities are shown by blue dots in Figure 1. How much of the upward shift of the survival curve during the 20th century has been caused by improved health care is a debated issue, which is not yet completely resolved. Much of the improved survival at working age was likely to be due to improved nutrition and public health measures like sanitation and the implied reduction in the spread of diseases (McKeown, 1976; Fogel; 1994). Old age diseases like cardiovascular diseases and cancer, however, were largely unaffected by these trends and they were actually increasing during the first half of the 20th century. Moreover, the reductions in mortality at old age

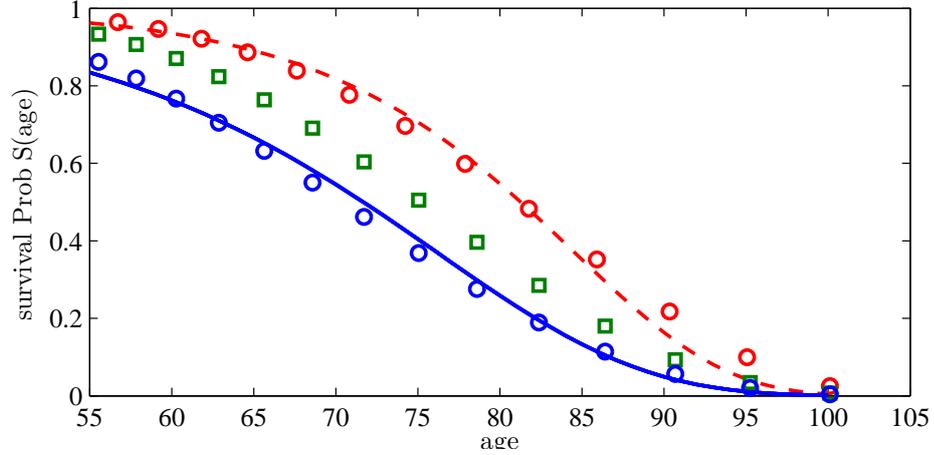
achieved since the 1950s can be largely attributed to medical innovations and improved medical care (Cutler and Meara, 2001). We take these stylized facts into account and assume for the benchmark run of the model that about 80 percent of improved survival of the working aged is caused by “improved health environment”, as shown by the green squares in Figure 1. It is reached by an exogenous reduction of α_1 from 1.9 to 1.3, while leaving α_2 unchanged. Notice that survival in retirement improves as well because of the intergenerational transmission of better health as driven by $b > 0$.

We assume that the remainder of the shift of the survival curve has been caused by health technology and health expenditure. To jointly calibrate technology parameters β_1 , β_2 , and the structure of health expenditure, H_1 , H_2 , we assume that (i) 47 percent of health care expenditure is spent for the ages of 65 and above (i.e. $H_2/(H_1 + H_2) = 0.47$), consistent with evidence for the year 2006,¹⁶ and (ii) H_1 , H_2 fulfill health budget constraint $H_1 + H_2 = \tau wL$, where total labor supply is the aggregate over all frailty groups, $L = \varpi(\tau, s)^\varepsilon \sum_{n_1=0}^{\bar{n}} g(n_1, \lambda_1) \tilde{l}(n_1) \tilde{R}(n_1, \bar{R})$. Under these assumptions, the best fit of the survival curve for the year 2000 is reached for $H_1 = 17.9$, $H_2 = 15.8$ (i.e. $h_1 = 0.0179$, $h_2 = 0.0158$), $\beta_1 = 15$ and $\beta_2 = 20$. Predicted survival is shown by red circles in Figure 1. The calibrated model correctly predicts that actual life-expectancy at birth, LE as given by (12), is 79.8 years in the year 2000. Moreover, it predicts a GDP share of health care expenditure of 10.3 percent while actually it was 10.7 percent in the year 2008.

We expect results to respond sensitively to the curvature of the utility function. In order to establish robustness of our main results we thus assume generally iso-elastic period utility $u(c) = c^{1-\sigma}/(1-\sigma)$ for $\sigma \neq 1$ and $u(c) = \log(c)$ for $\sigma = 1$. For the benchmark case we consider log-utility, consistent with evidence by Chetty (2006), Engelhardt and Kumar (2009) and Hartley, Lanot and Walker (2013). We discuss the role of the degree of relative risk aversion, σ , for our results and the empirical evidence employed to calibrate σ below. Disutility of work at the extensive margin (retirement) is driven by a preference for leisure, specifying $V(R, n_1) = \nu \cdot (1 + \xi \cdot n_1) \cdot R^{1+1/\gamma}$, $\nu > 0$, $\gamma > 0$, $\xi \geq 0$. We primarily focus on the case $\xi = 0$, where health does not enter utility independently (in line with Becker, 2007), relegating the case $\xi > 0$ to the sensitivity analysis. When $\xi = 0$, all influence

¹⁶See Robert Koch Institut, Gesundheitsberichterstattung des Bundes, Themenheft 48, 2009.

Figure 1: Actual and Predicted Survival Probability

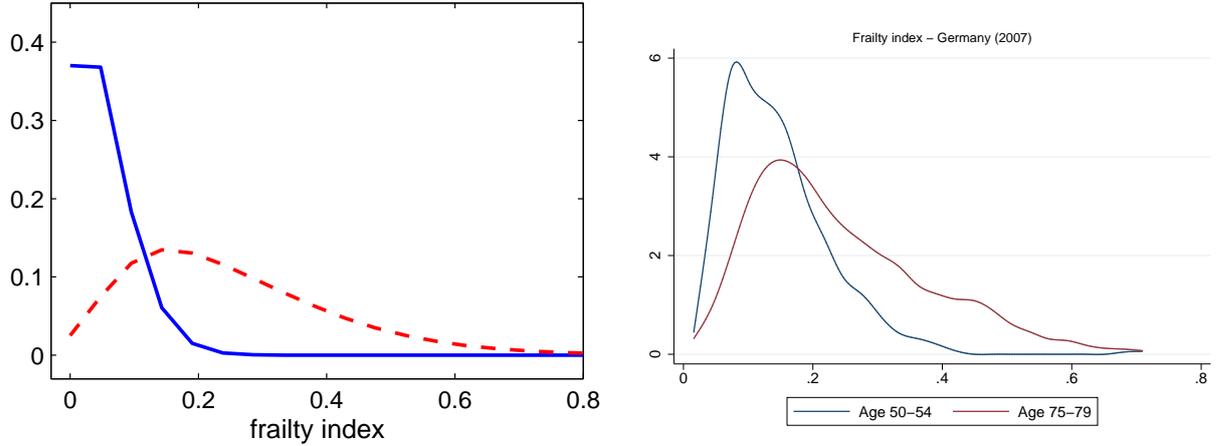


Survival probabilities in Germany according to Gompertz law in 1900 (solid line) and 2000 (dashed line) and predictions by the model (dots). Squares indicate improvement of survival not originating from improved health care. See text for details.

of health on individual welfare originates from its impact on life-expectancy and labor productivity. This provides a lower bound on individual preferences for health. When $\xi > 0$, not only does health affect non-material welfare but also the marginal disutility from later retirement is influenced by individual health status. We set $\gamma = 0.25$ according to recent evidence on the Frisch elasticity of labor supply at the extensive margin (Chetty et al., 2011) and the time preference rate to 1 percent, $\rho = 0.01$. This leaves us with one degree of freedom, the value of ν . We pin down ν by assuming that it is optimal under the present social security system to retire at the age of 65 (i.e. $\bar{R} = 1$). This provides $\nu = 0.36$.

For further comparison with the actual data we computed the implied distribution of the frailty index, i.e. of the relative number of health deficits out of a long list of potential bodily impairments. Harttgen et al. (2013) have calculated the frailty index from the SHARE data for several European countries including Germany. Estimates and predictions are shown in Figure 2. As a reading example for the left panel of Figure 2, since the maximum number of health deficits in the calibrated model is $\bar{n} = 20$, a frailty index of 0.2 means four health deficits. The model approximates the overall distribution reasonably well. The working-aged individuals in our model are a bit too healthy when

Figure 2: The Frailty Index: Calibration vs. Estimation from SHARE Data for Germany



Left: predicted probability distribution of the frailty index for working age population (solid line) and retired population (dashed line). Right: Estimated density of the frailty index for the age group 50-54 and 75 to 79.

compared with 50-54 year old persons from the SHARE sample. Unfortunately, SHARE does not provide any data for persons younger than 50, which is already quite close to the retirement age compared to the average German worker. The frailty distribution of the retired population approximates the actual frailty distribution of the 75-79 year olds very well.

6 Optimal Health and Pension Policy

Following the methodology introduced above, optimal social insurance policy maximizes expected individual welfare (25) subject to (13) behind the veil of ignorance, i.e. before the idiosyncratic health outcome is revealed. It is instructive to begin the numerical experiments with a constrained optimization problem. Taking the health system as given we determine the optimal pension system, i.e. the statutory retirement age \bar{R} and the savings rate s that simultaneously maximize welfare. The constrained optimal policy is found to be $s = 0.144$ and $\bar{R} = 1.045$, i.e. at age 67 instead of 65.0. Constrained by the current health system, individuals prefer to reduce the savings rate for retirement by 28 percent and to retire two years later. These results depend, of course, heavily on the chosen labor supply elasticity. For $\varepsilon = 0.1$ we obtain a pension savings rate of 16.7

percent and a statutory retirement age of 66.5 years as preferred policy.

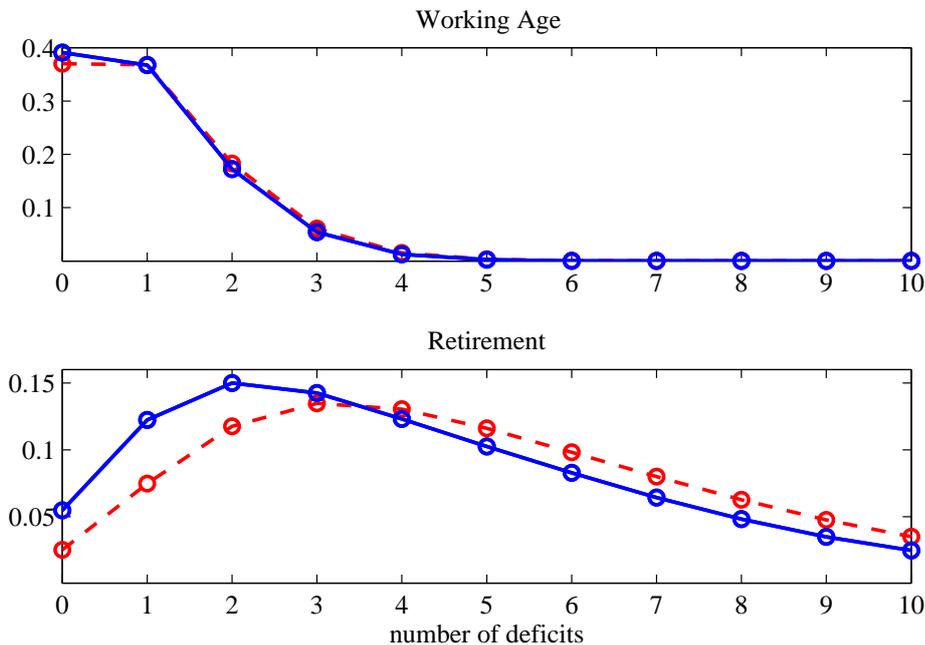
6.1 Benchmark Scenario

We next determine the optimal pension and health system simultaneously, i.e. we maximize expected welfare over (h_1, h_2, s, \bar{R}) . Results are shown in the second row of Table 1. The first row displays the status quo before optimization. The best policy is characterized by a mild increase of health expenditure for the working-aged and doubling of health expenditure for the elderly (from about 15.8 to 31). Accordingly, the optimal health contribution rate, τ , increases from 15.5 to 24.5 percent. These health care improvements lead to an increase of life expectancy at birth from about 79.8 to 82.7 years. Furthermore, individuals prefer to retire about 1.4 years later and to reduce their pension contribution rate, s from 20 to 15.2 percent. Compared to the constrained optimal solution, however, individuals prefer to save more for retirement (15.2 instead of 14.4 percent) because they expect to spend a longer time in retirement due to increasing life expectancy.

Figure 3 shows the impact of the optimal policy on the health deficit distribution. For better visibility the figure focusses on the range from zero to ten deficits. Among the working population, shown in the upper panel, improved health care has the predominant effect of (mildly) raising the share of completely healthy persons in the population. Among the retired the largest effect is observed for those suffering from one or three health deficits. As shown in Table 1 the improvement of average health deficits for the working-aged is relatively small compared to the gain of the retired persons who suffer on average from about one health deficit less.

Interestingly, better health care is predicted to worsen health inequality, as measured by the Gini coefficient of health deficits, in particular for the elderly ($Gini_2$) for whom the Gini coefficient rises from from 35 to 39 percent. Increased health spending implies that the distribution of health deficits shifts to the left and the bulk of working-aged individuals enter the retirement period relatively healthy. Although rising health spending h_1 and h_2 reduces the Poisson parameters λ_1 and λ_2 (i.e. the variance of the distribution of health

Figure 3: Deficit Distribution: Actual vs. Optimal Policy



Dashed lines: actual policy quadruple (s, R, H_1, H_2) . Solid lines: optimal policy quadruple.

deficits in the respective period of life) it increases health inequality.¹⁷ As shown in the last column of Table 1, going from the status quo to the preferred policy promises a welfare gain of one percent.

6.2 Sensitivity Analysis

We continue with sensitivity analysis. First we investigate the role of the elasticity of labor supply. Assuming $\varepsilon = 0.1$ instead of 0.2, the optimal τ increases towards 29 percent and the optimal s falls by less, toward 18 percent, a natural consequence of the reduced harm of labor taxation on labor supply (row 3 in Table 1). Moreover, individuals do not want to modify the status quo retirement age. When $\varepsilon = 0.3$ and labor supply responds heavily to net wages, the estimated taxes are much lower. The optimal τ is 21 percent and the optimal s is 13.3 percent (row 4 in Table 1). The pattern which emerges is thus that current pension contributions are identified as being too high and current health

¹⁷Suppose, hypothetically, that all individuals entered the retirement age with the same number of health deficits. In this case, an increase in h_2 would reduce health inequality among the elderly.

expenditure is identified as being too low.¹⁸ Individuals want a social system that allows them to live a longer and healthier life and are willing to substitute consumption now and in old age in order to achieve this goal. Another systematic pattern is that the optimal policy increases health inequality, particularly among the elderly, by improving largely the health of individuals with only a few deficits.

Our next numerical experiments consider the power of medical technology. To begin with we assume that all improvement in health during the 20th century can be attributed to medical technology. This hypothetical scenario provides an upper bound for the welfare gain that could be expected from health and pension reform (provided that health status does not directly affect utility, $\xi = 0$). For that purpose we keep α_1 at its level from the year 1900, which provides the estimates $\beta_1 = 25$ and $\beta_2 = 30$. Results for this "high tech" scenario are shown in row 5 of Table 1. Not surprisingly it is now optimal to increase health expenditure during working age quite drastically. This comes at the expense of slightly less health expenditure for retirees than in the benchmark case, which is, however, still considerably higher than the status quo expenditure in Germany. Overall, the optimal health expenditure contribution rate rises towards 33.5 percent of total labor income ($\tau = 0.335$). As a result, life expectancy is predicted to be 8.6 years higher than at the status quo and welfare rises by 8.9 percent. The policy has also strong implications for health inequality. The Gini coefficient of health deficits rises markedly, among both workers and the elderly. The reason is that thanks to the powerful health technology there are large gains in life expectancy for those who developed only a small number of health deficits. For the unlucky at the right end of the health deficit distribution, however, health technology is still too weak in order to improve life expectancy substantially.

In row 6 of Table 1 we show results for a "low tech"-scenario. For that we assumed that nothing of the improvement of health of the working age population during the 20th century can be attributed to health technology and health expenditure. Matching the survival curve for the working-aged for the year 2000 for the case $\beta_1 = 0$ requires to set $\alpha_1 = 1$ (instead of 1.3); the other parameters are like in the benchmark scenario. When

¹⁸This pattern disappears, of course, if the labor supply elasticity is assumed to be implausibly high. For $\epsilon = 0.5$ the optimal τ is obtained as 15.0, i.e. mildly lower than at the status quo.

$\beta_1 = 0$, it is obviously optimal to spend nothing on health care of the working population. More interestingly, the model predicts that even more resources than in the benchmark scenario (row 2) should be devoted to health improvements of the elderly (whose health production function did not change). Overall the optimal health contribution rate, τ , becomes 17.8 percent, whereas it is 15.5 at the status quo. According to the low-tech scenario the status quo health expenditure for the working-aged is wasted and consequently, when all resources are devoted to the elderly, it predicts larger increases of life expectancy and welfare than the benchmark scenario. Both in the low tech scenario and the high tech scenario the optimal retirement age is somewhat higher than currently in Germany and a bit lower than in the benchmark scenario. The optimal savings rate rises more in the low tech scenario, compared to the benchmark optimal policy. This is because lower health spending in the low tech scenario makes it more attractive to save for retirement, leaving the retirement age unaffected.

We expect results to be largely affected by the curvature of the utility function. Intuitively, living a year longer expands utility from consumption linearly while consuming more at any given year suffers from decreasing marginal utility. A more concave utility function reduces the gain from consuming more at any given year and increases the propensity to forego consumption in favor of health expenditure in order to live a healthier and longer life. Put differently, a more concave utility function is associated with a higher degree of relative risk aversion and motivates individuals to defer consumption and demand more health care in order to reduce health risk (or repair health deficits) and increase life expectancy.

In principle, instantaneous utility $u(c) = (c^{1-\sigma} - 1)/(1 - \sigma)$ could assume negative values when $\sigma > 1$. In most settings the scale of utility is harmless. In the present context, however, negative utility would imply that individuals prefer a life as short as possible. In order to avoid this degenerate outcome we set the value of the scale factor A at a high enough value such that the positive constant $-1/(1 - \sigma)$ is sufficient to ensure positive utility for all types of individuals. As in Hall and Jones (2009) adding a positive constant avoids negative utility. In contrast to Hall and Jones, the constant is not chosen arbitrarily but pinned down by the requirement that $u(c)$ converges to $\log(c)$ for $\sigma \rightarrow 1$. Another

Table 1: Optimal Health and Pension Policy

Case	H_1	H_2	τ	s	\bar{R}	LE	$E(n_1)$	$E(n_2)$	$Gini_1$	$Gini_2$	$\frac{\Delta V}{V}$
1) status quo	17.9	15.8	15.5	20	65.0	79.8	1.00	5.24	0.52	0.35	0.0
2) benchm.	21.6	31.0	24.6	15.2	66.4	82.7	0.94	4.38	0.54	0.39	1.0
3) low ε	28.0	35.0	29.2	18.1	65.0	83.8	0.85	4.02	0.56	0.41	1.3
4) high ε	17.9	27.7	20.9	13.3	67.2	82.0	1.00	4.65	0.52	0.38	0.9
5) high tech	43.8	27.9	33.5	15.7	65.6	86.6	0.64	3.23	0.62	0.44	4.1
6) low tech	0.00	38.6	17.8	18.0	65.8	83.2	1.00	4.24	0.52	0.41	3.9
7) high σ	50.1	56.7	53.7	11.5	65.2	88.4	0.61	2.76	0.63	0.49	8.9
8) low σ	0.00	3.70	1.70	11.7	67.8	76.5	1.30	6.70	0.47	0.31	1.6
9) high ρ	17.9	31.4	22.9	15.0	66.6	82.4	1.00	4.50	0.52	0.39	0.9
10) health util	34.9	27.1	28.6	13.8	67.5	83.5	0.77	4.13	0.58	0.39	1.5
11) $\delta=0$	17.9	37.8	23.6	15.8	66.4	83.2	1.00	4.26	0.52	0.41	1.0

Policy parameters are jointly set to optimal values. \bar{R} is the retirement age converted to years, LE is life expectancy at birth, $E(n_j)$ is expected health deficits in period $j \in \{1, 2\}$, $Gini_j$ is the Gini coefficient for health deficits in period j , and $\Delta V/V$ is the welfare gain compared to status quo in percent. τ and s are expressed in percent.

consequence of a higher σ is that individuals prefer leisure relatively more compared to the benchmark case. We thus recalibrate ν such that retirement at age 65 remains the constrained optimal solution for $s = 0.2$ and the status quo health system.

Results for $\sigma = 1.5$ are shown in row 7 of Table 1. We reduce ν to 0.062 in order to ensure retirement at age 65 at the status quo. Given the higher degree of risk aversion individuals prefer to spend much more on health and the average number of health deficits $E(n_1)$, $E(n_2)$, reduces to almost half of the status quo number in both periods. In row 8 we show results for $\sigma = 0.75$ (and ν raised to 1.05). Given the lower degree of risk aversion, individuals experience relatively high additional utility when they extend contemporaneous consumption and thus relatively little utility from a long life. Consequently they prefer to spend nothing on health during working age and to reduce expenditure in retirement to a mere 3.6. They also, counter-intuitively, prefer to die about 3 years earlier than at the status quo.

Summarizing, we find that deviations from log-utility induce quite drastic responses of optimal health expenditure. One interpretation of these results could be that the status quo health system is hugely inefficient, providing much too little or much too much health care, depending on the degree of relative risk aversion. An alternative interpretation is

inspired by a recent literature that estimates the empirical size of elasticities from careful calibration of economic models. For instance, Chetty (2006) proposes a method in which the degree of relative risk aversion can be inferred from empirical estimates of the elasticity of labor supply. His preferred estimate suggests that the degree of relative risk aversion is plausibly close to unity. A completely different approach is proposed by Hartley et al. (2013). They exploit a large data set from a famous television game show to infer the degree of relative risk aversion via non-parametric estimation. They find a constant degree of risk aversion equal to unity approximates utility well over a large range of the values of potential winnings.

Compared to the curvature parameter, results are relatively little affected by the discount rate. This conclusion can be inferred from the experiment of row 9 in Table 1, which shows results when ρ equals 0.03 (instead of 0.01). Individuals want to spend less on health during the working period such that the status quo spending turns out to be optimal for relatively impatient individuals. However, individuals still want to spend more on health in retirement. The preferred τ remains considerably larger than at the status quo.

Row 10 in Table 1 shows results when health deficits affect utility directly (and not just through labor productivity and life expectancy). For that purpose we set $\xi = 1$ and re-calibrate ν to 0.18 in order to match a status quo retirement age of 65 years. We see that the qualitative conclusions regarding health policy remain intact. Individuals prefer to spend substantially more on health at working age and a little less on health in retirement compared to the benchmark optimal policy mix. The average number of health deficits is lower also during retirement, reflecting the path-dependency of health deficits. The preferred health contribution rate τ increases to 28.6 percent. Because health affects utility of workers, individuals prefer to spend more on health during working age and because of that they are generally more healthy and thus more productive during working age. As this reduces the costs of later retirement, the preferred statutory retirement age increases. Moreover, the optimal pension contribution rate declines to compensate for the increased health costs. These results highlight again the importance of taking health endogenously.

Finally, we investigate the role of the dependency of labor supply on health status in working age, parameterized by δ . In row 11 of Table 1, rather than $\delta = 0.1$, we set $\delta = 0$ (i.e. $\tilde{l}' = 0$) – a case which was discussed in the analytical part (albeit not being supported by evidence). Maintaining an optimal statutory retirement age of 65 for the status quo policy requires to re-calibrate $\nu = 0.36$. As health status during working age does not contribute to the social insurance systems when $\delta = 0$, individuals do not prefer higher health spending H_1 than at the status quo. Optimal health spending for retirees, H_2 , however, rises even beyond that of the benchmark scenario. The optimal pension policy is quite robust against changes in the dependency of labor supply on health status.

We conclude that the health contribution rate and particularly health spending for the elderly are likely to be suboptimally low in Germany. Improving health of the elderly, however, significantly raises health inequality. Generally, individuals prefer to retire somewhat later and to save significantly less for retirement. In sum, compared to the status quo, individuals prefer to sacrifice more consumption in working age and in retirement in favor of a healthier and longer life.

7 Conclusion

We integrated into public economics a biologically founded, stochastic process of individual ageing. The novel approach makes it possible to investigate theoretically and quantitatively the interaction between health and retirement policy for welfare and for health inequality. In particular, we derive the optimal design of the public insurance system behind the veil of ignorance. We show that the optimal allocation of health spending between working-aged individuals and retirees is typically biased towards workers in the sense that it does not maximize life expectancy at retirement age except for a special case which is not supported by empirical evidence on the response of labor supply to health shocks. Most importantly, results from the calibration of the model for Germany suggest that the health contribution rate and particularly health spending for the elderly may, nevertheless, be considerably too low. Improving health of the elderly, however, significantly raises health inequality. This result is interesting in view of the intensive debate

on the health distribution. For instance, the WHO explicitly aims at reducing health inequity, defined as being "avoidable" in the sense that it is "attributable to the external environment and conditions mainly outside the control of the individuals concerned". Abstracting from behavioral decisions which may affect individual health, our analysis suggests that this goal may be in conflict with welfare maximization of ex ante identical individuals behind the veil of ignorance. Compensating for the higher health spending, it is optimal ex ante to significantly reduce the savings rate for old-age pensions. Finally, our results suggest that the statutory retirement age should be slightly raised, in line the public debate about higher retirement age as a consequence of increased longevity.

In future research we plan to endogenize the health technology within the developed framework.¹⁹ For instance, it is interesting to investigate how the incentive to innovate in the pharmaceutical sector interacts with the public insurance system. It is also important to examine to which degree health innovations should be promoted vis-à-vis non-health innovations.

Appendix

Appendix A. Proof of Proposition 2

Define $\mathbf{z} \equiv (h_1, \tau, s, \bar{R})$. First, we establish the following properties.

Lemma A.1. *At an interior solution to (33), $\mathbf{z}^* = (h_1^*, \tau^*, s^*, \bar{R}^*)$, the following holds:*

- (i) *If $\tilde{l}' = V_n = 0$, then $\partial \tilde{n}_2(\mathbf{z}^*) / \partial h_1 = 0$;*
- (ii) *If $\tilde{l}' < 0$ or $V_n > 0$, then $\partial \tilde{n}_2(\mathbf{z}^*) / \partial h_1 > 0$.*

Proof. Let us define

$$P(s, \tau) \equiv \tau \varpi(\tau, s)^\varepsilon, \text{ i.e. } P(\tau, s) = s \varpi(\tau, s)^\varepsilon, \quad (37)$$

¹⁹In a recent paper, Grossmann (2013) studies the role of institutional regulations in the pharmaceutical sector and co-insurance schemes in the health system on pharmaceutical innovations. However, he does not capture the interactions with the social security system and does not endogenize life expectancy.

where the latter follows from $\varpi(\tau, s) = \varpi(s, \tau)$. Thus, according to (28), we can write

$$\tilde{h}_2(\mathbf{z}) = P(s, \tau) \bar{R} \omega \tilde{l}(\tilde{a}_1(h_1)) - h_1. \quad (38)$$

Moreover, define

$$\begin{aligned} \Phi(n_2, \mathbf{z}) \equiv & \left(e^{-\rho \tilde{T}(n_2)} u \left(\frac{\bar{R} w P(\tau, s) \tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(n_2) - \bar{R}} \right) - \frac{e^{-\rho \bar{R}} - e^{-\rho \tilde{T}(n_2)}}{\rho} \times \right. \\ & \left. u' \left(\frac{\bar{R} w P(\tau, s) \tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(n_2) - \bar{R}} \right) \frac{\bar{R} w P(\tau, s) \tilde{l}(\tilde{a}_1(h_1))}{(\tilde{T}(n_2) - \bar{R})^2} \right) \tilde{T}'(n_2), \end{aligned} \quad (39)$$

$$\hat{\Phi}(\mathbf{z}) \equiv \Phi(\tilde{n}_2(\mathbf{z}), \mathbf{z}). \quad (40)$$

Partially differentiating (32) with respect to h_1 and τ , we obtain

$$\begin{aligned} \hat{U}_h(\mathbf{z}) = & \left(\frac{(1 - e^{-\rho \bar{R}}) u' \left(A \varpi(\tau, s)^{1+\varepsilon} \tilde{l}(\tilde{a}_1(h_1)) \right) A \varpi(\tau, s)^{1+\varepsilon} \tilde{l}'(\tilde{a}_1(h_1))}{\rho} + \frac{e^{-\rho \bar{R}} - e^{-\rho \tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} \times \right. \\ & \left. u' \left(\frac{\bar{R} w P(\tau, s) \tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(\tilde{n}_2(\mathbf{z})) - \bar{R}} \right) \frac{\bar{R} w P(\tau, s) \tilde{l}'(\tilde{a}_1(h_1))}{\tilde{T}(\tilde{n}_2(\mathbf{z})) - \bar{R}} - V_n(\bar{R}, \tilde{a}_1(h_1)) \right) \tilde{a}'_1(h_1) + \hat{\Phi}(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1}, \end{aligned} \quad (41)$$

$$\begin{aligned} \hat{U}_\tau(\mathbf{z}) = & \hat{\Phi}(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau} - \frac{1 - e^{-\rho \bar{R}}}{\rho} u' \left(A \varpi(\tau, s)^{1+\varepsilon} \tilde{l}(\tilde{a}_1(h_1)) \right) A \omega (1 + \varepsilon) \varpi(\tau, s)^\varepsilon \tilde{l}(\tilde{a}_1(h_1)) - \\ & \frac{e^{-\rho \bar{R}} - e^{-\rho \tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} u' \left(\frac{\bar{R} w s \varpi(\tau, s)^\varepsilon \tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(\tilde{n}_2(\mathbf{z})) - \bar{R}} \right) \frac{\bar{R} w s \omega \varepsilon \varpi(\tau, s)^{\varepsilon-1} \tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(\tilde{n}_2(\mathbf{z})) - \bar{R}}, \end{aligned} \quad (42)$$

where, according to (29) and (38),

$$\frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} = \tilde{a}'_2(\tilde{h}_2) \left[P(s, \tau) \bar{R} \omega \tilde{l}'(\tilde{a}_1) \tilde{a}'_1(h_1) - 1 \right] + b \tilde{a}'_1(h_1), \quad (43)$$

$$\frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau} = \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) P_\tau(s, \tau) \bar{R} \omega \tilde{l}(\tilde{a}_1(h_1)). \quad (44)$$

At the optimum, there cannot be Laffer effects, i.e. the partial derivative of function P , as defined in (37), with respect to τ fulfills

$$P_\tau(s^*, \tau^*) = \omega^\varepsilon (1 - \tau^* - s^*)^{\varepsilon-1} [1 - s^* - (\varepsilon + 1)\tau^*] > 0. \quad (45)$$

Thus, according to (44) and (45), $\partial \tilde{n}_2(\mathbf{z}^*) / \partial \tau < 0$. Hence, at an interior solution \mathbf{z}^* to (33), where $\hat{U}_\tau(\mathbf{z}^*) = 0$, we have

$$\hat{\Phi}(\mathbf{z}^*) < 0, \quad (46)$$

according to (42). By definition of \mathbf{z}^* , $\hat{U}_h(\mathbf{z}^*) = 0$. The results thus follow from (41) and (46). ■

Moreover, using assumptions $\tilde{a}_1'' > 0$, $\tilde{a}_2'' > 0$ and $\tilde{l}'' \geq 0$, according to (43), we find that

$$\begin{aligned} \frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial (h_1)^2} &= \tilde{a}_2''(\tilde{h}_2(\mathbf{z})) \left(P(s, \tau) \bar{R} \omega \tilde{l}'(\tilde{a}_1(h_1)) \tilde{a}_1'(h_1) - 1 \right)^2 + b \tilde{a}_1''(h_1) + \\ &\tilde{a}_2'(\tilde{h}_2(\mathbf{z})) P(s, \tau) \bar{R} \omega \left(\tilde{l}''(\tilde{a}_1(h_1)) \tilde{a}_1'(h_1) + \tilde{l}'(\tilde{a}_1) \tilde{a}_1''(h_1) \right) > 0. \end{aligned} \quad (47)$$

Observing $\tilde{T}' < 0$ concludes the proof. ■

Appendix B. Proof of Proposition 3

First, we establish the following properties (subscripts on Φ and $\hat{\Phi}$ denote partial derivatives).

Lemma A.2. *Under the presumptions of Proposition 3, we have (i) $\Phi_n(n, \mathbf{z}^*) \leq 0$, (ii) $\hat{\Phi}_h(\mathbf{z}^*) < 0$, (iii) $\hat{\Phi}_\tau(\mathbf{z}^*) \geq 0$, (iv) $\hat{\Phi}_{\bar{R}}(\mathbf{z}^*) \geq 0$, and (v) if $\varepsilon > (=) 0$, then $\hat{\Phi}_s(z^*) < (=) 0$.*

Proof. Using $u(c) = \log c$ in (39), we have

$$\Phi(n_2, \mathbf{z}) \equiv \left[e^{-\rho \tilde{T}(n_2)} \log \left(\frac{\bar{R} \omega P(\tau, s) \tilde{l}(\tilde{a}_1(h_1))}{\tilde{T}(n_2) - \bar{R}} \right) - \frac{1}{\rho} \frac{e^{-\rho \bar{R}} - e^{-\rho \tilde{T}(n_2)}}{\tilde{T}(n_2) - \bar{R}} \right] \tilde{T}'(n_2), \quad (48)$$

It is straightforward to show that

$$\begin{aligned} \Phi_n(n, \mathbf{z}) &= \frac{\Phi(n, \mathbf{z}) \left[\tilde{T}''(n) - \rho \tilde{T}'(n)^2 \right]}{\tilde{T}'(n)} + \\ &\quad \frac{\tilde{T}'(n)^2 e^{-\rho \tilde{T}(n)} \left[e^{q(n, \bar{R})} (1 - q(n, \bar{R})) - (1 + q(n, \bar{R})) \right]}{\rho \left(\tilde{T}(n) - \bar{R} \right)^2}, \end{aligned} \quad (49)$$

where $q(n, \bar{R}) \equiv \rho \left(\tilde{T}(n) - \bar{R} \right)$. As $\hat{\Phi}(\mathbf{z}^*) < 0$ when (h_1^*, τ^*) is an interior solution of (33) and $\tilde{T}' < 0$, the first summand on the right-hand side of (49) is non-positive. The second summand is negative if $q \geq 1$ or if $q < 1$ and $e^q \leq \frac{1+q}{1-q}$. The latter holds in view of properties $q(n, \bar{R}) \geq 0$ and $e^q \leq \frac{1}{1-q}$ for $q < 1$. This confirms part (i). To prove part (ii), use the definition in (40) to derive from (48) that

$$\hat{\Phi}_h(\mathbf{z}) = \Phi_n(\tilde{n}_2(\mathbf{z}), \mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} + \Phi_h(\tilde{n}_2(\mathbf{z}), \mathbf{z}), \text{ where} \quad (50)$$

$$\Phi_h(n_2, \mathbf{z}) = e^{-\rho \tilde{T}(n_2)} \tilde{T}'(n_2) \frac{\tilde{l}'(\tilde{a}_1(h_1)) \tilde{a}'_1(h_1)}{\tilde{l}(\tilde{a}_1(h_1))} < 0. \quad (51)$$

Using in (50) that $\Phi_n(n, \mathbf{z}^*) \leq 0$ (part (i) of Lemma A.2), $\Phi_h(n, \mathbf{z}^*) < 0$ from (51), and $\partial \tilde{n}_2(\mathbf{z}^*) / \partial h_1 \geq 0$ (Lemma A.1) holds, we confirm part (ii). To prove part (iii), note that

$$\hat{\Phi}_\tau(\mathbf{z}) = \Phi_n(\tilde{n}_2(\mathbf{z}), \mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau}. \quad (52)$$

Recalling from part (i) of Lemma A.2 that $\Phi_n(n, \mathbf{z}^*) \leq 0$ and using $\partial \tilde{n}_2(\mathbf{z}^*) / \partial \tau < 0$ confirms part (iii) from (52). To prove part (iv), note that

$$\hat{\Phi}_{\bar{R}}(\mathbf{z}) = \Phi_n(\tilde{n}_2(\mathbf{z}), \mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \bar{R}}. \quad (53)$$

To confirm part (iv), again, recall $\Phi_n(n, \mathbf{z}^*) \leq 0$ and note that

$$\frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \bar{R}} = \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) P(s, \tau) \omega \tilde{l}(\tilde{a}_1(h_1)) < 0, \quad (54)$$

according to (29) and (38). Finally, to prove part (v), note that

$$\hat{\Phi}_s(\mathbf{z}^*) = \Phi_n(\tilde{n}_2(\mathbf{z}^*), \mathbf{z}^*) \frac{\partial \tilde{n}_2(\mathbf{z}^*)}{\partial s}, \quad (55)$$

where, according to (29),

$$\frac{\partial \tilde{n}_2(\mathbf{z})}{\partial s} = \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) \frac{\partial \tilde{h}_2(\mathbf{z})}{\partial s} > (=)0 \text{ if } \varepsilon > (=)0 \quad (56)$$

since, according to (28), $\partial \tilde{h}_2(\mathbf{z})/\partial s < (=)0$ if $\varepsilon > (=)0$. This concludes the proof. ■

Lemma A.3. *In an interior solution to (33),*

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial h_1 \partial \tau} < 0, \quad (57)$$

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial h_1 \partial \bar{R}} < 0, \quad (58)$$

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial \tau^2} > 0, \quad (59)$$

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial s^2} > 0 \text{ if } \varepsilon \in (0, 1) \text{ and } \frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial s^2} > 0 \text{ if } \varepsilon = 0 \text{ or } \varepsilon = 1 \quad (60)$$

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial s \partial h_1} > (=)0 \text{ if } \varepsilon > (=)0. \quad (61)$$

Proof. According to (43), we have

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial h_1 \partial \tau} = \left[\tilde{a}''_2(\tilde{h}_2(\mathbf{z})) \frac{\partial \tilde{h}_2(\mathbf{z})}{\partial h_1} \tilde{l}(\tilde{a}_1(h_1)) + \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) \tilde{l}'(\tilde{a}_1(h_1)) \tilde{a}'_1(h_1) \right] P_\tau(s, \tau) \bar{R} \omega, \quad (62)$$

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial h_1 \partial \bar{R}} = \left[\tilde{a}''_2(\tilde{h}_2(\mathbf{z})) \frac{\partial \tilde{h}_2(\mathbf{z})}{\partial h_1} \tilde{l}(\tilde{a}_1(h_1)) + \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) \tilde{l}'(\tilde{a}_1(h_1)) \tilde{a}'_1(h_1) \right] P(s, \tau) \omega. \quad (63)$$

According to Lemma A.1,

$$\frac{\partial \tilde{n}_2(\mathbf{z}^*)}{\partial h_1} \geq 0, \quad (64)$$

which, according to (43), implies that

$$\frac{\partial \tilde{h}_2(\mathbf{z}^*)}{\partial h_1} = P(s^*, \tau^*) \bar{R}^* \omega \tilde{l}'(\tilde{a}_1(h_1^*)) \tilde{a}'_1(h_1^*) - 1 < 0. \quad (65)$$

Recalling that $P_\tau(s^*, \tau^*) > 0$, $\tilde{a}'_1 < 0$, $\tilde{l}' \leq 0$, $\tilde{a}'_2 < 0$ and $\tilde{a}''_2 > 0$, as well as using (65) in (62) and (63) confirms (57) and (58), respectively.

To prove (59), use (28) and (44) to find

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z}^*)}{\partial \tau^2} = \left[\tilde{a}''_2(\tilde{h}_2(\mathbf{z}^*)) P_\tau(s^*, \tau^*)^2 \bar{R} \omega \tilde{l}(\tilde{a}_1(h_1^*)) + \tilde{a}'_2(\tilde{h}_2(\mathbf{z}^*)) P_{\tau\tau}(s^*, \tau^*) \right] \bar{R} \omega \tilde{l}(\tilde{a}_1(h_1^*)), \quad (66)$$

According to (45), we have

$$P_{\tau\tau}(s^*, \tau^*) = -\varepsilon \omega^\varepsilon (1 - \tau^* - s^*)^{\varepsilon-2} [2(1 - s^*) - (\varepsilon + 1)\tau^*] < 0, \quad (67)$$

confirming (59).

Finally, according to (56),

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial s^2} = \tilde{a}''_2(\tilde{h}_2(\mathbf{z})) \left(\frac{\partial \tilde{h}_2(\mathbf{z})}{\partial s} \right)^2 + \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) \frac{\partial^2 \tilde{h}_2(\mathbf{z})}{\partial s^2}, \quad (68)$$

$$\frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial s \partial h_1} = \tilde{a}''_2(\tilde{h}_2(\mathbf{z})) \frac{\partial \tilde{h}_2(\mathbf{z})}{\partial h_1} \frac{\partial \tilde{h}_2(\mathbf{z})}{\partial s} + \tilde{a}'_2(\tilde{h}_2(\mathbf{z})) \frac{\partial^2 \tilde{h}_2(\mathbf{z})}{\partial s \partial h_1}. \quad (69)$$

According to (28), $\partial \tilde{h}_2(\mathbf{z})/\partial s \leq 0$, $\partial^2 \tilde{h}_2(\mathbf{z})/\partial s \partial h_1 \leq 0$ (both holding with equality if $\varepsilon = 0$) and, if $\varepsilon < 1$, $\partial^2 \tilde{h}_2(\mathbf{z})/\partial s^2 < 0$ ($\partial^2 \tilde{h}_2(\mathbf{z})/\partial s^2 = 0$ if $\varepsilon = 0$ or $\varepsilon = 1$). Also recall from (65) that $\partial \tilde{h}_2(\mathbf{z}^*)/\partial h_1 < 0$. Using these properties in (68) and (69) confirm (60) and (61), respectively. This concludes the proof. ■

Using $u(c) = \log c$ and $\tilde{l}'/\tilde{l} = -\delta$ from (34) in (41) and (42), we obtain

$$\hat{U}_h(\mathbf{z}) = \hat{\Phi}(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} - \left(\frac{1 - e^{-\rho \tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} \delta + V_n(\bar{R}, \tilde{a}_1(h_1)) \right) \tilde{a}'_1(h_1), \quad (70)$$

$$\hat{U}_\tau(\mathbf{z}) = -\frac{1 - e^{-\rho \bar{R}}}{\rho} \frac{1 + \varepsilon}{1 - \tau - s} + \hat{\Phi}(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau} - \frac{e^{-\rho \bar{R}} - e^{-\rho \tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} \frac{\varepsilon}{1 - \tau - s}, \quad (71)$$

respectively. Using (70), we find

$$\begin{aligned}\hat{U}_{hh}(\mathbf{z}) = & - \left(\frac{1 - e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} \delta + V_n(\bar{R}, \tilde{a}_1(h_1)) \right) \tilde{a}_1''(h_1) + \hat{\Phi}_h(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} - \\ & \left(e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))} \tilde{T}'(\tilde{n}_2(\mathbf{z})) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} \delta + V_{nn}(\bar{R}, \tilde{a}_1(h_1)) \tilde{a}_1'(h_1) \right) \tilde{a}_1'(h_1) + \hat{\Phi}(\mathbf{z}) \frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial (h_1)^2},\end{aligned}\quad (72)$$

$$\hat{U}_{h\tau}(\mathbf{z}) = -e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))} \tilde{T}'(\tilde{n}_2(\mathbf{z})) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau} \delta \tilde{a}_1'(h_1) + \hat{\Phi}_\tau(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} + \hat{\Phi}(\mathbf{z}) \frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial h_1 \partial \tau}, \quad (73)$$

$$\begin{aligned}\hat{U}_{h\bar{R}}(\mathbf{z}) = & - \left(e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))} \tilde{T}'(\tilde{n}_2(\mathbf{z})) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \bar{R}} \delta + V_{n\bar{R}}(\bar{R}, \tilde{a}_1(h_1)) \right) \tilde{a}_1'(h_1) + \\ & \hat{\Phi}_{\bar{R}}(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial h_1} + \hat{\Phi}(\mathbf{z}) \frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial h_1 \partial \bar{R}}.\end{aligned}\quad (74)$$

In view of (72), $\hat{U}_{hh}(\mathbf{z}^*) < 0$ follows from (47), (64), $\hat{\Phi}(\mathbf{z}^*) < 0$ (recall (46)), $\hat{\Phi}_h(\mathbf{z}^*) < 0$ (part (ii) of Lemma A.2), $V_{nn} \geq 0$, $\tilde{T}' < 0$, $\tilde{a}_1' < 0$, $\tilde{a}_1'' > 0$. In view of (73), $\hat{U}_{h\tau}(\mathbf{z}^*) > 0$ follows from (44), (57) as given in Lemma A.3, (64), $\hat{\Phi}(\mathbf{z}^*) < 0$ (recall (46)), $\hat{\Phi}_\tau(\mathbf{z}^*) \geq 0$ (part (iii) of Lemma A.2), $\tilde{T}' < 0$, $\tilde{a}_1' < 0$. In view of (74), $\hat{U}_{h\bar{R}}(\mathbf{z}^*) > 0$ follows from (54), (64), (58), $\hat{\Phi}(\mathbf{z}^*) < 0$ (recall (46)), $\hat{\Phi}_{\bar{R}}(\mathbf{z}^*) \geq 0$ (part (iv) of Lemma A.2), $V_{n\bar{R}} \geq 0$, $\tilde{T}' < 0$, $\tilde{a}_1' < 0$.

Moreover, (71) implies

$$\begin{aligned}\hat{U}_{\tau\tau}(\mathbf{z}) = & - \frac{1 - e^{-\rho\bar{R}}}{\rho} \frac{1 + \varepsilon}{(1 - \tau - s)^2} + \hat{\Phi}_\tau(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau} + \hat{\Phi}(\mathbf{z}) \frac{\partial^2 \tilde{n}_2(\mathbf{z})}{\partial \tau^2} - \\ & \frac{e^{-\rho\bar{R}} - e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} \frac{\varepsilon}{(1 - \tau - s)^2} - \frac{\varepsilon}{1 - \tau - s} e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))} \tilde{T}'(\tilde{n}_2(\mathbf{z})) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial \tau},\end{aligned}\quad (75)$$

In view of (75), $\hat{U}_{\tau\tau}(\mathbf{z}^*) < 0$ follows from (44), $\hat{\Phi}(\mathbf{z}^*) < 0$ (recall (46)), $\hat{\Phi}_\tau(\mathbf{z}^*) \geq 0$ (part (iii) of Lemma A.2) and (59).

Next, use (32) and $u(c) = \log c$ and definition $P(\tau, s) = s\varpi(\tau, s)^\varepsilon$ from (37) to obtain

$$\hat{U}_s(\mathbf{z}) = \frac{e^{-\rho\bar{R}} - e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho} \Gamma(\tau, s) - \frac{1 - e^{-\rho\bar{R}}}{1 - \tau - s} \frac{1 + \varepsilon}{\rho} + \hat{\Phi}(\mathbf{z}) \frac{\partial \tilde{n}_2(\mathbf{z})}{\partial s}, \quad (76)$$

$$\begin{aligned} \hat{U}_{ss}(\mathbf{z}) = & e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))}\tilde{T}'(\tilde{n}_2(\mathbf{z}))\frac{\partial\tilde{n}_2(\mathbf{z})}{\partial s}\Gamma(\tau, s) + \frac{e^{-\rho\bar{R}} - e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))}}{\rho}\Gamma_s(\tau, s) - \\ & \frac{1 - e^{-\rho\bar{R}}}{(1 - \tau - s)^2}\frac{1 + \varepsilon}{\rho} + \hat{\Phi}_s(\mathbf{z})\frac{\partial\tilde{n}_2(\mathbf{z})}{\partial s} + \hat{\Phi}(\mathbf{z})\frac{\partial^2\tilde{n}_2(\mathbf{z})}{\partial s^2}, \end{aligned} \quad (77)$$

$$\hat{U}_{sh}(\mathbf{z}) = e^{-\rho\tilde{T}(\tilde{n}_2(\mathbf{z}))}\tilde{T}'(\tilde{n}_2(\mathbf{z}))\frac{\partial\tilde{n}_2(\mathbf{z})}{\partial h_1}\Gamma(\tau, s) + \hat{\Phi}_h(\mathbf{z})\frac{\partial\tilde{n}_2(\mathbf{z})}{\partial s} + \hat{\Phi}(\mathbf{z})\frac{\partial^2\tilde{n}_2(\mathbf{z})}{\partial s\partial h_1}, \quad (78)$$

where we defined

$$\Gamma(\tau, s) \equiv \frac{P_s(\tau, s)}{P(\tau, s)} = \frac{1}{s} - \frac{\varepsilon}{1 - \tau - s}, \text{ i.e. } \Gamma_s(\tau, s) < 0 \quad (79)$$

for the partial derivative of Γ with respect to s . Using properties (46), (56), (60) (79), (55) and part (v) of Lemma A.2 in (77) confirms $\hat{U}_{ss}(\mathbf{z}^*) < 0$. Finally, $\hat{U}_{sh}(\mathbf{z}^*) < 0$ follows from part (ii) of Lemma A.2, (46), (61) together with $\partial\tilde{n}_2(\mathbf{z}^*)/\partial h_1 \geq 0$ (according to (64), with equality iff $\tilde{l}' = V_n = 0$) and $\partial\tilde{n}_2(\mathbf{z})/\partial s \geq 0$ (according to (56), with equality iff $\varepsilon = 0$). This concludes the proof. ■

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