Negligible senescence: An economic life cycle model for the future.

Negligible Senescence: An Economic Life Cycle Model for the Future*

Davide Dragone† University of Bologna

Holger Strulik‡ University of Göttingen

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Abstract

We propose a model of aging and health deficit accumulation model with an infinite time horizon and a steady state of constant health. The time of death is uncertain and endogenous to lifestyle and health behavior. This setup can be conceptualized as a strive for immortality that is never reached. We discuss adjustment dynamics and show that the new setup is particularly useful to understand aging of the oldest old, i.e. of individuals for which morbidity and mortality have reached a plateau. We then show how the existence of a steady state can be used to perform comparative dynamics exercises analytically. As an illustration we investigate the effects of more expensive health investment and of advances in medical technology on optimal short run and long run health behavior.

Keywords: Comparative dynamics, Endogenous mortality, Life-expectancy, Medical progress

JEL codes: D91, I12, J17

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†University of Bologna, Department of Economics, Piazza Scaravilli 2, 40126, Bologna, Italy; e-mail: davide.dragone@unibo.it

‡University of Göttingen, Department of Economics, Platz der Göttinger Sieben 3, 37073 Göttingen, Germany; email: holger.strulik@wiwi.uni-goettingen.de

The Universal Declaration of Human Rights does not say humans have
'the right to life until the age of ninety'.
It says that every human has a right to life, period.
That right isn't limited by any expiry date.
(Yuval Noah Harari, 2016)

1 Introduction

Human life is finite but the time of death is unknown. In this paper we build on this fact to investigate a theoretical model of aging where mortality is stochastic and endogenously affected by individual behavior and lifestyle. We show that this scenario can be conveniently formalized as an infinite time horizon problem in which human life is conceptualized as a process where a state of constant health is a meaningful long run goal. We use this setup to study the determinants of aging and longevity and to explain the observed aging of the oldest old, i.e. of individuals for which morbidity and mortality have reached a plateau. We then propose a method to investigate how exogenous shocks affect health behavior over the life cycle.

Any discussion of the determinants and limits of human aging makes sense only with the notion of aging as a biological (or physiological) phenomenon. While chronological aging is given by passing calendar time, biological aging is defined as the intrinsic, cumulative, progressive, and deleterious loss of function (Arking, 2006). In contrast to chronological aging, biological aging is modifiable. It could be slowed down and perhaps, eventually, abandoned (Jones and Vaupel, 2017). A plausible and straightforward measure of biological aging has been established in gerontology by the so called frailty index, also known as the health deficit index. The measure has been developed by Mitnitski and Rockwood (2001, 2002) and it has by now been used in hundreds of gerontological studies. The health deficit index simply computes the relative number of health conditions that an individuals has from a (long) list of potential conditions. As the index rises, the individual is viewed as increasingly frail, and in this sense physiologically older.[1] There exist a strong positive association between the health deficit index and mortality (Rockwood and Mitnitski, 2007).

While human aging, perhaps until recently, has been regarded as inevitable, the speed

[1]Originally, the methodology was established by Mitnitski, Rockwood, and coauthors as the frailty index. Newer studies use also the term health deficit index (e.g. Mitnitski and Rockwood, 2016), which seems to be a more appropriate term when the investigated population consists to a significant degree of non-frail persons. See Searle et al. (2008) for details on the construction of the health deficits index.

of this process is not immutable. The accumulation of health deficits can be influenced by health investments and health behavior. This idea has been formalized in health economics by Dalgaard and Strulik (2014). The literature building on the Dalgaard and Strulik (2014) model rules out the existence of a steady state of infinite life by imposing appropriate parameter restrictions (on, for example, the power of medical technology in repairing health deficits). Income-constrained individuals are assumed to maximize the value of life given that survival beyond a certain maximum number of health deficits is impossible. In this setup it is shown that health deficits optimally increase in a quasi-exponential way and the mortality- or hazard-rate also increases in such a quasi-exponential way, akin to the Gompertz (1825) law of mortality. This predicted pattern on health deficits and mortality is sketched by solid lines in Figure 1.

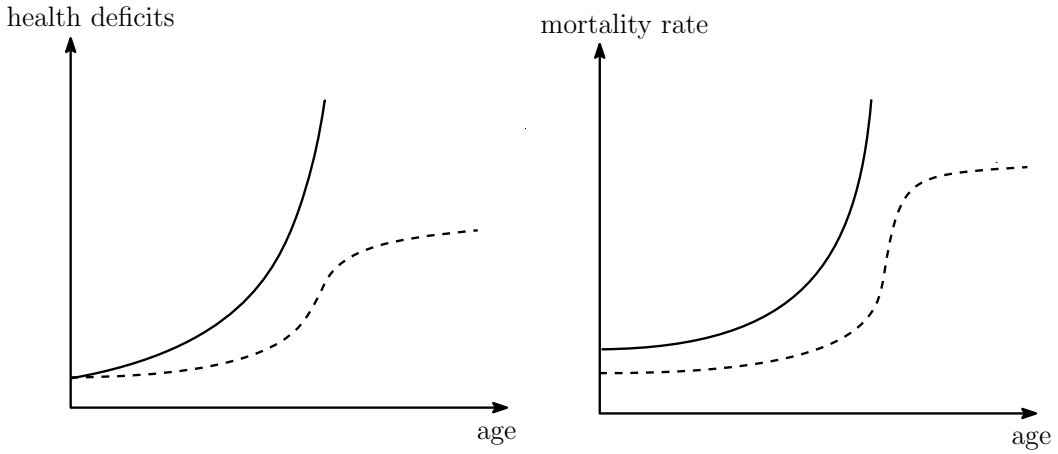


Figure 1: Stylized life cycle trajectories for health deficit accumulation (left) and mortality rate (right). Solid lines: standard model; dashed lines: existence of steady state.

In this paper we show that health deficit accumulation and mortality follow a decidedly different life cycle trajectory when a steady state of constant health exists. Instead of growing exponentially these trajectories follow an *s*-shaped (or convex-concave) pattern; they increase in middle age and level off in old age, as shown by dashed lines in Figure 1. It is a well established fact that the quasi-exponential increase of mortality is only a good approximation for ages below about 90. For the oldest old, the increase of mortality slows down and reaches a plateau for supercentenarians, i.e. individuals above age 110 (Horiuchi and Wilmoth, 1998; Maier et al., 2010; Barbi et al., 2018). When the rate of mortality and health deficits stabilize at a constant level, individuals converge towards a state where they are no longer aging in physiological terms. In the model of health deficit accumulation,

such a slowdown is impossible if there exists no steady state. In this paper we consider the existence of a steady state and show that the health deficit model is capable of producing a slowdown in aging, described by adjustment dynamics along the stable saddlepath towards the steady state. Along the adjustment path, health deficit accumulation slows down by increasing investment in health maintenance and repair.

It should be emphasized that our model does not imply immortality. In fact, people accumulate health deficits as in the conventional model (Dalgaard and Strulik, 2014) and their life expectancy is finite and, given a reasonable calibration of the survival function, in line with current observations. The innovation is that human aging does no longer *inevitably* end in death at some finite age. Instead, motivated by the advancements in medical technology, individuals rationally believe that aging-related health deficits can be repaired such that the state of "negligible senescence"(Finch, 2009) becomes a desirable goal. It is supported by recent research in gerontology and biodemography showing that the limits to life expectancy are broken (Oeppen and Vaupel, 2002) and that human life span is not immutable but in fact increasing over time (Wilmoth and Robine, 2003; Strulik and Vollmer, 2013). While few scholars agree with de Grey (2013) and Kurzweil and Grossman (2010), who envision human immortality for the near future, many have abandoned the belief that there exists necessarily a "capital T " beyond which human life extension is impossible (e.g. Vaupel, 2010; Kontis et al., 2017; Sinclair and LaPlante, 2019).

As discussed in Dalgaard and Strulik (2014), the health deficit model is particularly well suited for investigating aging and longevity. In the health deficit model, health deficits, if unremedied by health maintenance and repair, accumulate approximately at a constant rate. This explosive growth of deficits (at the force of aging μ) captures the gerontological notion of biological aging as the cumulative, progressive, and deleterious loss of bodily function (Arking, 2006). This process is endogenous to a person's behavior because it can be slowed down by a healthy lifestyle and by investing in health. We formalize this consideration by assuming that the probability of dying has both an endogenous and an exogenous component. The former operates through the accumulation of health deficits, while the latter depends on factors, such as the mere passing of time, that cannot be influenced by the individual.

As observed in the literature, different assumptions regarding terminal conditions have marked implications on optimal behavior (Forster, 2001). Yet, there is still no consensus on the appropriate terminal condition to be used. Since life is empirically finite, most models on aging and longevity consider a finite time horizon, either by assuming that the moment

of death is given, or endogenously chosen by the agent. In both cases this implies assuming that the agent knows with certainty the exact moment of death (Ehrlich and Chuma, 1990; Eisenring, 1999; Forster, 2001; Dalgaard and Strulik, 2014). Our approach shows that, even if life is empirically finite, since the moment of death is uncertain, a terminal condition to be fulfilled at infinity can be appropriate and theoretically-grounded.

Our results show how a rational and forward-looking individual optimally adjusts her lifestyle to exploit the intertemporal tradeoffs between health, consumption and the probability of dying. A major advantage of our model is that it allows for a convenient analytical investigation of the factors affecting human aging and longevity. In this respect, we contribute to the literature on dynamic optimization models, which typically assesses the impact of policies and shocks either through phase diagram analysis or through numerical simulations (Oniki, 1973; Ehrlich and Chuma, 1990; Eisenring, 1999; Forster, 2001; Kuhn et al., 2015). In particular, our results could be useful when the problem involves more than one state variable (in which case phase diagram analysis could be applied only under specific assumptions), or when numerical simulations are too computationally demanding. Our approach can be considered a complement to the comparative dynamics analysis proposed in Caputo (1990,1997) and in Dragone and Vanin (2015), which focus on the response of the steady state, and it can in principle be applied to any intertemporal behavior for which aiming at a stationary state is a meaningful goal. Our formulas to perform comparative dynamics analysis are obtained for a general survival function and a general utility function.

To study the optimal response of behavior and health to exogenous shocks, we focus on two different time-horizons: the response of behavior "on impact", i.e. the impulse response at the time of the shock, and the long run effect on individual choices and health. The distinction between short and long run response emphasizes that a forward-looking individual, when taking into account the effects of current behavior on future expected utility, may respond differently over different time horizons, even when preferences are stable and time-consistent.

As an illustration, we apply our method for comparative dynamics to investigate how the cost of health investment and the state of medical technology affect behavior and health, both on impact and in the long run. We show that, when the cost of repairing health deficits increases, biological aging will be faster. On impact, health investment will be lower, but it will be higher in the long run. On the contrary, with more efficient medical technology, biological aging will be slower. On impact, health investment will be higher (if

medical technology is good enough), but it will be lower in the long run. In both cases the behavioral responses in the short run and in the long run will be of opposite sign.

The paper is organized as follows. In the next section we provide an introduction to the framework of health deficit accumulation and the evolution of health deficits across age and time. In Section 3 we set up the health deficit model with endogenous mortality. In Section 4 we provide the general formulas to perform comparative dynamics analysis in the short and in the long run. In Section 5 we consider two examples: a rise in the cost of health and an improvement in medical technology. In the latter case, we consider both a sudden one-off technological improvement, as well as an extended version of the basic model which accounts for constant medical progress. Section 6 concludes.

2 Health Deficits Across Age and Time

We measure biological aging by the accumulation of health deficits with age. A straightforward metric for health deficits is the health deficit index, also known as frailty index, developed by Mitnitski and Rockwood (2001, 2002). The health deficit index as a metric of health, aging, and morbidity has been used by hundreds of studies in gerontology and medical science and it slowly gains importance in economics as well (Abeliansky and Strulik, 2018a,b; Hosseini et al., 2019). The index measures the relative number of health deficits that a person has out of a sufficiently long list of potential aging-related health deficits (see Searle et al., 2008, for methodological background). The health deficit index is an excellent predictor of mortality such that chronological age adds insignificant explanatory power when added to the regression (Rockwood and Mitnitski, 2007). It is an encompassing measure of the state of health that has a foundation in the reliability theory of human aging (Gavrilov and Gavrilova, 1991). A great advantage of the measure is that it can be compared across populations and over time (Mitnitski et al., 2005).

As humans get older they develop more health deficits such that there exists a strong quasi-exponential association between age and the health deficit index, akin to the Gompertz-Makeham law of mortality (Mitnitski and Rockwood, 2002, Abeliansky and Strulik, 2018a). This means that health deficits D accumulate according to $\dot{D} = \mu(D - E)$, in which μ is the natural force of aging and E is a stand-in for factors that slow down health deficit accumulation (as, for example, health investments). For $E = 0$ health deficits grow exponentially. On average, health deficits increase by 3 to 4 percent per year but the individual-specific accumulation of health deficits depends on healthy behavior and access to medical technol-

ogy. Later born generations develop health deficits more slowly because they benefit more from improvements in medical technology (broadly defined, i.e. including also the diffusion of knowledge about certain health behavior). Abeliansky and Strulik (2019) showed a steady decline of health deficits such that for every year of later birth, younger generations experience 1.4-1.5 percent less health deficits than earlier born generations. This trend has been found to be remarkably stable across countries and over time (i.e. over the range of the study's sample from birth year 1918 to birth year 1965).

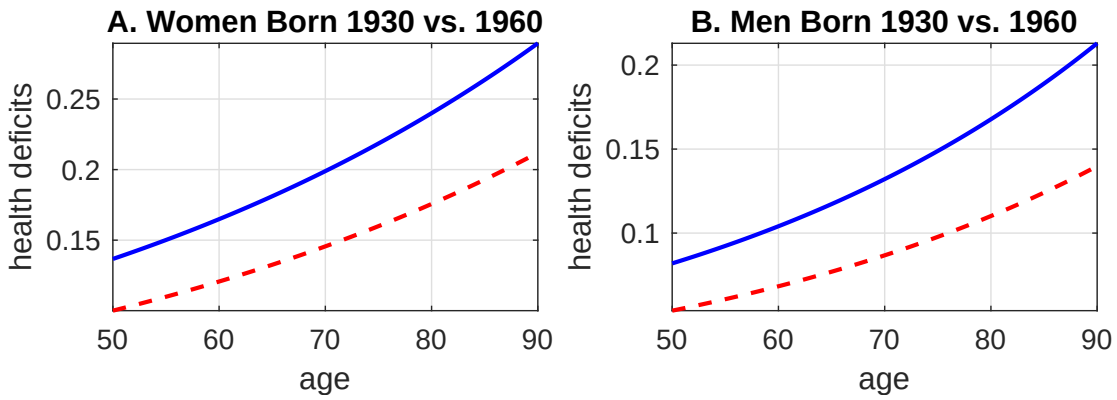


Figure 2: Average health deficits by age. Panel A: solid (blue) line: Women born 1930, dashed (red) line: women born 1960. Panel B: solid (blue) line: Men born 1930, dashed (red) line: men born 1960. Source: Abeliansky and Strulik (2019).

Figure 2 illustrates the benchmark results for men and women from Abeliansky and Strulik (2019). Solid (blue) lines show the health deficits predicted for the specific age for average Europeans born 1930 and dashed (red) lines show predictions for the cohort born 1960.[2] At any age, the later born cohort exhibits substantially fewer health deficits. Moreover, the difference between early and late born individuals increases with age, which means that the slope of health deficit accumulation becomes flatter. The results show that human aging has been substantially delayed in the period considered. For example, the level of health deficits experienced at age 65 by individuals born 1930 is predicted to be

[2]The estimates are based on a panel of 14 European countries (Austria, Belgium, Czech Republic, Denmark, France, Germany, Greece, Ireland, Italy, Netherlands, Poland, Spain, Sweden, and Switzerland) and five waves of the SHARE data set, an index based on 38 health deficits, and individuals aged 50 to 85. Similar results were obtained without the upper age restriction but health deficit accumulation for the oldest old is less well approximated by the quasi-exponential law of health deficit accumulation, a phenomenon to which we return later in this paper.

experienced at about age 80 by individuals born 1960. The continuous slowdown of health deficit accumulation over time is reminiscent of Oeppen and Vaupel's (2002) finding of a continuous increase of best-performance life expectancy by about a quarter of a year per year of birth from 1840 to 2000.

If the slowdown of health deficit accumulation continues due to future medical progress, it becomes eventually possibly to break the exponential accumulation of deficits with age and the possibility of a steady state of constant health emerges. At the steady state, the inevitably arising physiological damage due to human metabolism is continuously repaired by health care investments such that the life cycle trajectory of deficit accumulation becomes essentially flat. As discussed in the Introduction, if and in particular when such a scenario becomes reality is highly debated among gerontologists and other natural scientists. In any case, however, it is an interesting research question whether and how the possibility of a steady state will affect life cycle behavior. Here, we take a first step in this direction by investigating an environment in which such a steady state of negligible senescence exists but is attained only asymptotically. Compared to optimistic scenarios that envision future immortality for everyone (as in Kurzweil and Grossman, 2010) this is a relatively mild modification of the status quo. It means that negligible senescence emerges as a reasonable goal, which is, however, never reached.

Aging as the accumulation of health deficits has been integrated into economic life cycle theory by Dalgaard and Strulik (2014) and refined towards a stochastic conceptualization of death by Strulik (2015b). So far, however, the analysis of the health deficit model focussed on parameter constellations such that a non-negative steady state is either non-existent or globally unstable. Optimal lifetime trajectories thus end when a feasible maximum of health deficits has been developed. In the stochastic version (Strulik, 2015b) this means that there exists an upper bound of health deficits at which individuals die with certainty, $S(D) = 0$. Here, we show that an interior saddlepoint stable steady state exists if the parameters governing medical technology are favorable enough. This feature fundamentally changes adjustment dynamics. Formally, individuals no longer solve a free terminal time problem (when to die) but converge along the stable manifold towards the steady state. The unique trajectory towards the steady state is qualitatively different to all other life cycle paths since all other paths eventually diverge at increasing speed from the steady state. In contrast to the so far available theory, which focused on increasing age profiles for health deficits and health expenditure, the presence of a steady state generates convergence of health deficits and health expenditure towards constants. This paper explores the comparative dynamics

when such a plateau exists. While we have emphasized that this proposes a life cycle model for the future, it is interesting to see that such a plateau is already discernable today for health behavior and outcomes of the oldest old in a population. De Nardi et al. (2016) observe that, for elderly Americans, health expenditure net of nursery care does no longer rise with age for ages above 85. Manton et al. (2008) and Barbi et al. (2018) argue that mortality rates stabilize at a high level for ages above 100. These phenomena cannot be explained by the so far available health deficit models. They require the existence of an approachable steady state.

3 A model of endogenous aging with uncertain lifetime

3.1 The model

Consider an agent whose health condition is represented by the number of health deficits accumulated over lifetime. The process of health deficits accumulation depends on the stock of health deficits D and on medical care h at time t,

$$\dot{D} = f\left(D\left(t\right), h\left(t\right)\right). \tag{1}$$

The accumulation of health deficits is faster when health deficits are large ($f_D\left(D, h\right) > 0$), and it is slower when the agent buys medical care ($f_h\left(D, h\right) < 0$). As motivated in Section 2 and discussed in detail by Dalgaard and Strulik (2014), the accumulation of health deficits is well represented by the quasi-exponential function,

$$f\left(D\left(t\right), h\left(t\right)\right) = \mu\left[D\left(t\right) - a - A\left(h\left(t\right)\right)^{\gamma}\right], \tag{2}$$

Parameter $\mu > 0$ represents the natural force of aging, $a \geq 0$ is a measure of the repairing rate of the body (absent any medical care) and A and γ reflect the state of medical technology. The parameter $A > 0$ captures the general efficiency of medical care in the repair of health deficits, while $\gamma \in (0, 1)$ captures the degree of decreasing returns of medical care. Note that, if the level of deficits were low enough, i.e. $D\left(t\right) < a$, then the body would be able to repair its health deficits even in absence of health investment and there would be no "natural aging". To avoid this trivial situation, we assume that the initial condition $D\left(0\right)$ is larger than a.

Besides spending income on final goods and health care, individuals may save or borrow at a net interest rate r. The individual takes all prices as exogenously given. The law of motion for individual wealth k is thus given by

$$\dot{k}(t) = rk(t) + Y - c(t) - ph(t), \tag{3}$$

where k is capital, r is the interest rate, Y is income and c is a composite good whose price is normalized to one. The price of medical care is p, and it includes the cost of medicines, as well as the opportunity cost of health investment.

At time t_0, the agent's problem is to choose consumption and medical care over her lifetime. In a deterministic environment this amounts to consider the following intertemporal utility function:

$$\int_{t_0}^{T} e^{-\rho t} U(c(t))\, dt, \tag{4}$$

where $U(c)$ is the instantaneous utility function, ρ is the discount rate due to individual impatience, and T is the age at death. As in Hall and Jones (2007), we normalize utility after death to zero. This implies that individuals with positive utility from consumption experience a positive value of life. They thus desire a long life and have no incentive to minimize the length of their life.[3] The age at death T could be determined ex-ante, as usually in macroeconomic life cycle models of generational accounting (e.g. Erosa and Gervais, 2002), or it could be endogenously determined by individual choices, as in most life cycle models in health economics (e.g. Grossman, 1972; Ehrlich and Chuma, 1990; Kuhn et al., 2015).

Here we consider a third alternative, where the age at death T is unknown, but can still be influenced through individual behavior. The stochastic nature of death has been investigated within the health deficit framework by Strulik (2015b). The main difference to that study, Dalgaard and Strulik (2014), and all other previous studies of human aging in a life cycle model is that we allow for and focus on the existence of a steady state of health deficits D^{ss} that is biologically feasible, i.e. $D^{ss} < D^{\max}$. Accordingly, death is conceptualized as a stochastic event, which occurs when either the individual is hit by a stochastic adverse shock, or when health deficits reach a biological maximum level D^{max}.[4]

[3] As usual, the utility function is strictly increasing and concave in consumption. Our results qualitatively hold also if the health condition has a utility and a productivity value (Grossman, 1972). Here we neglect these channels and focus on the role of health deficits in affecting the probability of dying.

[4] There exists a logically upper bound $D = 1$ (100% of health deficits have been accumulated) for the health deficit index. However, the empirical literature has found that individuals typically do not survive beyond $D = 0.7$ (see Rockwood and Mitnitski, 2006; Hubbard et al., 2013).

To account for the stochastic nature of death, in the proceeding we consider the following survival function

$$\mathcal{S}\left(D\left(t\right),t\right)=s\left(t\right)S\left(D\left(t\right)\right)=e^{-qt}S\left(D\left(t\right)\right). \tag{5}$$

Function $\mathcal{S}\left(D\left(t\right),t\right)$ provides the conditional probability (as of time zero) to die at age t when D health deficits are accumulated. The health-dependent component $S\left(D\right)$ is equal to one in absence of health deficits ($S\left(0\right)=1$) and equal to zero when the biological maximum level is reached ($S\left(D\right)=0$ for $D\geq D^{\max}$). It is non negative, continuous, and strictly decreasing in the relevant range (i.e. $S(D)>0$, $S_D(D)<0$ for $D\in(0,D^{\max})$.

As more health deficits are accumulated the probability decreases. The multiplicative specification allows to disentangle the hazard function $\Gamma\equiv-\partial\log\mathcal{S}\left(\cdot\right)/\partial t$ in two components,

$$\Gamma=q+\mathcal{Z}\left(D\right)\dot{D}. \tag{6}$$

where $q=-\mathcal{S}_t\left(\cdot\right)/\mathcal{S}\left(\cdot\right)>0$ and $\mathcal{Z}\left(D\right)\equiv-\mathcal{S}_D\left(\cdot\right)/\mathcal{S}\left(\cdot\right)>0$. The term q sums up the role of environmental factors and individual characteristics that are out of the control of the agent. It does not depend on health deficits and it can be considered as the *exogenous* component of the hazard rate. In contrast, the second component $\mathcal{Z}\left(D\right)\dot{D}$ does not explicitly depend on time and can be considered as *endogenous*.

3.2 Solving the model

Under the hypothesis of uncertain time of death, the agent chooses the path of consumption and medical care that solves the following problem

$$\max_{c,h}\mathbb{E}_g\left[\int_0^T e^{-\rho t}U\left(c\left(t\right)\right)dt\right] \tag{7}$$

$$\dot{k}\left(t\right)=rk\left(t\right)+Y-c\left(t\right)-ph\left(t\right) \tag{8}$$

$$\dot{D}\left(t\right)=\mu\left[D\left(t\right)-a-A\left(h\left(t\right)\right)^{\gamma}\right] \tag{9}$$

$$k\left(0\right)=k_0,\qquad D\left(0\right)=D_0>a. \tag{10}$$

As suggested by Yaari (1965), the expected intertemporal utility function 7 can be conveniently transformed into a more treatable intertemporal expected utility function which weighs the instantaneous utility function by the individual survival probability.[5] Accordingly, the objective function of the agent from time t_0 onwards can be written as (see the

[5]Age 0 should be conceptualized as real age 20 since, by assumption, individuals are "born" as young adults. Consistent with the literature, the time arguments used to denote the state variables $D\left(t\right)$ and $k\left(t\right)$ and the control variables $h\left(t\right)$ and $c\left(t\right)$ should be interpreted as time labels. In the proceeding these

Appendix for details)

$$V(t_0) = \mathbb{E}_g \left[\int_{t_0}^{T} e^{-\rho t} U(c(t))\, \mathrm{d}t \right] = \int_{t_0}^{\infty} e^{-(\rho+q)t} S(D)\, U(c(t))\, \mathrm{d}t. \tag{11}$$

Equation 11 represents the expected value of life of the agent. The goal of the agent is to maximize it under 8 to 10.

The corresponding current-value Hamiltonian function is:

$$H = S(D)\, U(c) + \lambda \dot{D} + \eta \dot{k}, \tag{12}$$

where $\lambda = \lambda(t)$ and $\eta = \eta(t)$ are the costate variables associated with the dynamics of health deficits and capital, respectively.

The associated first order conditions are (omitting the arguments henceforth):

$$h^* \quad : \quad H_h = 0 \quad \Leftrightarrow \quad \lambda \gamma \mu A h^{\gamma - 1} = -p\eta \tag{13}$$

$$c^* \quad : \quad H_c = 0 \quad \Leftrightarrow \quad S U_c = \eta. \tag{14}$$

Note that $\eta > 0$ and $\lambda \leq 0$. The above conditions are also sufficient if the (Mangasarian) concavity condition is satisfied:

$$\Phi \equiv S_D^2 U_c^2 - (S U_{cc})\, U S_{DD} < 0. \tag{15}$$

Since $S > 0$ and $U_{cc} < 0$, concavity requires $U S_{DD} < 0$. This is an interesting necessary condition, as it allows for two possible cases. If the utility function has positive values, the Mangasarian conditions require survival functions that are concave. This occurs, for example, when $U = \frac{c^{1-\sigma}}{1-\sigma} + b$ and $\sigma < 1$, or $\sigma > 1$ and b large enough, and $S = 1 - D^2$. If, instead, the utility function would exhibit negative values (e.g. $U = \frac{c^{1-\sigma}}{1-\sigma} + b$ when $\sigma > 1$ and $b = 0$), then survival function were required to be convex (say, $S = e^{-D}$). A negative utility function, however, means that individuals prefer being dead (associated with zero utility) to living (which yields negative utility). For this reason, we focus on the case where the utility function has positive values and the deficit-dependent component of the survival function is concave, so that the condition $U S_{DD} < 0$ is satisfied in the relevant range. A concave survival function appears also to be more plausible than a convex one. The concave

time labels will be omitted to simplify the notation. We assume that the accumulated wealth becomes an unintended bequest when the individual dies. Yaari (1965)'s transformation implicitly relies on the assumption that the individual is risk neutral with respect to the length of life. See Bommier (2006) for an analysis with preferences over the length of life.

function reflects that additional health deficits have a small effect on survival when the level of health deficits is low and a large effect when many deficits are present. This means it captures the feature of "compression of morbidity" (Fries, 1980) since for most individuals the greatest decline in health is experienced chronologically close to death.[6]

Note that condition 13 does not hold if medical care is completely ineffective in repairing health deficits ($A = 0$). In fact, in this case investment in medical care would be nil and health deficits would inevitably accumulate (see eq. 9). This process of *progressive senescence* ends when the maximum biological level $D = D^{\max}$ is reached (or the agent is hit by an adverse shock, which occurs with probability e^{-qt}).

When instead medical care is effective ($A > 0$), the agent can invest in health repair to slow down health deficit accumulation, eventually reaching a state of *negligible senescence* where health deficits no further accumulate. For this scenario, the evolution of optimal behavior, health deficits, and capital is described by the following system of differential equations:

$$\dot{h} = \frac{h}{1-\gamma}\left[r - \mu - \frac{\gamma\mu A}{p}\frac{U(c)}{U_c(c)}\mathcal{Z}(D)\,h^{\gamma-1}\right] \tag{16}$$

$$\dot{c} = -\frac{U_c(c)}{U_{cc}(c)}\left[r - \rho - q - \mathcal{Z}(D)\,\dot{D}\right] \tag{17}$$

$$\dot{D} = \mu\left(D - a - Ah^{\gamma}\right) \tag{18}$$

$$\dot{k} = rk + Y - ph - c \tag{19}$$

Equations 16 and 17 represent the Euler equations of medical care and consumption, respectively, and they describe how the optimal choices of the agent change as function of the primitives of the model. With respect to the literature where the time of death is known, note that the endogenous hazard rate $\mathcal{Z} > 0$ affects both the dynamics of medical care and consumption.

To characterize the optimal path of consumption and health care over the agent's lifetime, it is necessary to determine the steady states where consumption, health care, health deficits, and capital are constant. Although the steady state will only be reached for $t \to \infty$, it is a meaningful goal if the survival function $\mathcal{S}$ is defined over an infinite time horizon. Such an assumption, however, is not restrictive: in the survival literature, which typically employs functions such as the exponential, the Weibull, and the Gompertz-Makeham distributions, the standard is to assume that surviving at very old ages is possible, but very

[6]For completeness, the conditions for existence and stability of a steady state in the case where $U < 0$ are reported in Appendix 7.3.

unlikely. In other words, we take into account the (almost trivial) insight from gerontology that "however old we are, our probability to die within the next hour is never equal to one" (Jacquard, 1982).[7] Accordingly, the realistic scenario of a finite but uncertain lifetime is equivalent to assume that people face no predetermined time of death. Hence the following Remark applies:

Remark 1 *If the time of death is uncertain at all t, making plans for the future is always optimal.*

The above Remark implies that it is optimal to make plans for the future *as if* there is a possibility that the time of death is infinitely far away (although death will surely occur in finite time). This fundamentally changes the nature of the optimization problem, which is no longer a free terminal time problem (of when to die) but the solution of adjustment dynamics towards a state of constant health. We show below that it leads to fundamentally different predictions. In particular, adjustment dynamics towards a steady state are characterized by survival probability, health deficits, and health expenditure reaching a plateau whereas if a steady state does not exist, survival probability, health deficits, and health expenditure, increase exponentially with age for all ages until death. The existence of a steady state allows to apply a new analytical method to obtain individual responses to parameter changes (like prices of medical care or medical technology) at the steady state and off the steady state. The feature that we can investigate adjustment dynamics off the steady state is particularly useful for the problem at hand because, we argue, (most) individuals are far off the steady state of constant health. Moreover, as we show below, the model predicts a qualitative difference between short- and long-run responses to shocks. For example, the model predicts that, as a response to a price increase of medical care, medical expenditure declines in the short run and increases in the long run. In order to obtain a conclusive picture on the impact of price changes it is thus essential to investigate both short-run and long-run adjustment dynamics.

Using an approach often adopted in life cycle models, in the proceeding we assume $q =$

[7] All standard survival functions imply that, in principle, infinite life is allowed for, although it is likely that such an event will occur with negligible probability. In addition to this mathematical and rather obvious argument, from a more philosophical viewpoint one could claim that the fact that we have never observed a human being living forever does not mean, *per se*, that human beings cannot reach immortality. In fact, it is possible that we have not observed any human being living forever just because it is a very unlikely event.

$r-\rho$.[8] The main consequence of this choice is that the Euler equation 17 for consumption depends only on the elasticity of intertemporal substitution $-U_c/(cU_{cc})$, on the endogenous hazard rate $\mathcal{Z}(D)$ and on the dynamics of health deficits:

$$\frac{\dot{c}}{c} = \frac{U_c}{cU_{cc}} \mathcal{Z}(D) \dot{D}. \tag{20}$$

Note that, since $U_c/(cU_{cc}) < 0$ and $\mathcal{Z}(D) > 0$, consumption decreases (increases) when health deficits increase (decrease).

Since equations 16 to 19 potentially allow for multiple steady states, we must establish conditions under which they are appropriate end-points of the optimal consumption and medical care paths.

We first consider existence of internal steady states, i.e. steady states in which the level of health repair and health deficits is positive, which we denote as states of negligible senescence:

Proposition 1 *Consider the endogenous aging problem 7 to 10 and $r = q + \rho$. A steady state of negligible senescence $(h^{ss}, D^{ss}, c^{ss}, k^{ss})$ satisfies*

$$h^{ss} = \left[\frac{\gamma\mu A\mathcal{Z}(D^{ss})}{pU_c(c^{ss})} \frac{U(c^{ss})}{(r-\mu)}\right]^{\frac{1}{1-\gamma}} \tag{21}$$

$$r = q + \rho \tag{22}$$

$$D^{ss} = a + A(h^{ss})^{\gamma} \tag{23}$$

$$k^{ss} = \frac{1}{r}(ph^{ss} + c^{ss} - Y) \tag{24}$$

where $h^{ss}, c^{ss} > 0$ and $D^{ss} \in (a, D^{\max})$.

The restriction $D^{ss} < D^{max} \leq 1$ distinguishes our paper from the literature (e.g. Strulik, 2015b). It highlights the existence of a state of negligible senescence that the individual will eventually reach by optimally investing in health and slowing down the process of health deficit accumulation.[9]

[8] In the literature on partial equilibrium life cyle models of intertemporal behavior it is common to focus on an analogue condition ($r = \rho$). This implies focusing on Frisch demand functions where the marginal utility of wealth is constant (see, e.g. Grossman, 1972, Heckman, 1974, 1976, Becker and Murphy, 1988, Ried, 1998, and eq. 38 in the Appendix). This allows to abstract from the dynamics originated by changes in individual wealth. Here we follow a similar approach.

[9] A corner steady state where health investment is nil and the individual remains in the best state of health ($D = a$) always exists. If the corner solution has saddle point stability, and if the financial resources available to the individual are large enough, it is admissible and reachable. If instead the corner steady state is unstable (as in the parametric configurations used in the examples of this paper), it is never reached unless $D_0 = a$.

For later reference, observe that the requirement of a positive level of health deficits repair, $h^{ss} > 0$, implies the following:

Corollary 1 *$r > \mu$ is a necessary condition for a steady state of negligible senescence to exist.*

Note that $r > \mu$ is a necessary but not sufficient condition for the existence of a state of negligible senescence. The sufficient condition additionally requires that $D^{ss} < D^{max}$.[10] If the steady state exists, it is a meaningful long run goal if there exists a path of investment (and consumption) trajectories that allows reaching it. Formally, this requires assessing the asymptotic stability of the steady state and computing the determinant $|\mathcal{J}^{ss}|$ of the Jacobian matrix associated to the dynamic system 16, 18 and 19. This amounts to checking the sign of:

$$|\mathcal{J}^{ss}| = \mu r \left(r - \mu\right)\left(1 - K\right) \qquad \text{where } K = -\frac{A\gamma h^{\gamma}\Phi}{\left(1-\gamma\right) SUS_D U_{cc}} > 0. \tag{25}$$

When $|\mathcal{J}^{ss}|$ is negative, the steady state is a saddle point, which means that there exists an optimal path of consumption and medical care that allows reaching a state of negligible senescence. If, instead, $|\mathcal{J}|$ is positive, the steady state is unstable and therefore not reachable.

Proposition 2 *Consider $r > \mu$ and a steady state of negligible senescence. The steady state is a saddle point if and only if $K > 1$.*

We thus arrive at two qualitatively different cases of human aging: progressive senescence and negligible senescence. These cases are illustrated in Figure 3. The difference between the two panels is the level of medical technology A, which is lower in panel (a). All parameter values of the numerical example are listed below the Figure. The interest rate r is set to 6 percent, close to the estimated average of the real interest rate over the last century (Jorda et al., 2019) and the natural rate of aging is set to 3 percent, close to empirical estimates of the rate of aging (Mitnitski et al., 2002; Abeliansky and Strulik, 2018). The value of the elasticity of intertemporal substitution is close to unity as suggested by Chetty (2006), implying that the utility function is close to logarithmic, as it is set in many calibration studies of dynamic models. The value of the exogenous probability of death has

[10] Existence and saddle point stability of a state of negligible senescence is possible if one allows for utility functions to be negative. In such a case, as shown in the Appendix, the necessary condition for existence is the opposite, i.e. $r < \mu$.

been proposed in calibrations of the perpetual youth model (Farmer, 2018). The implied value of the time preference, of four percent, is in an empirical plausible range and supports constant lifetime consumption, in line with empirical results on the age-consumption profile of childless households (Browning and Ejrnaes, 2009). Other parameter values, however, have no empirical justification. In particular, the medical parameter values γ and A are much higher than those calibrated by Dalgaard and Strulik for an application of the health deficit model for an average U.S. American in the year 2000 (where the steady state of negligible senescence is unstable). The required high values for the medical parameters are the main reason why we consider our approach as a "life cycle model for the future". Our ignorance about medical technology (or income) in the future prevents a full calibration of the model for, for example, a representative American. We thus modestly address these numerical representations of the model as examples or illustrations.

Panel (a) in Figure 3 shows a scenario of progressive senescence, which results when a steady state of negligible senescence does not exist or is unstable and thus unreachable. As shown by the thick line, the path of health deficits increases over time until it eventually hits the upper bound $D = D^{\max}$.

Panel (b) shows the case in which a state of negligible senescence exists and is a saddlepoint and thus reachable. Notice from 25 that this case comes into existence when the level of medical technology A becomes sufficiently large. In principle, the steady state can be approached from below or from above, depending on the initial level of deficits. The case that better describes aging and health expenditure along the human life cycle begins with initially low health deficits (of a young agent). Facing low deficits, the agent spends most income on consumption goods, and little on medical care. This slows down the process of deficit accumulation, although it does not reverse it. Deficits accumulate over time progressively affecting the trade-off between consumption and medical care. As a consequence, as the individual ages, medical care progressively increases, first at a slow rate, and subsequently at a faster rate. When the level of health deficits further increases and approaches the steady state, medical care reaches a plateau level. At the steady state of negligible senescence, health deficits, the hazard rate and medical care level out. This pattern is also shown, as a function of calendar time, by the solid lines of Figures 5 and 6. The predicted pattern of medical care expenditure and biological aging is remarkably consistent with the observed patterns of increasing budget shares for medical care over the lifetime (Banks et al, 2016), which reach a plateau at high ages above 85 (de Nardi et al., 2016), and with the evidence on the dynamics of hazard rates observed in supercentenar-

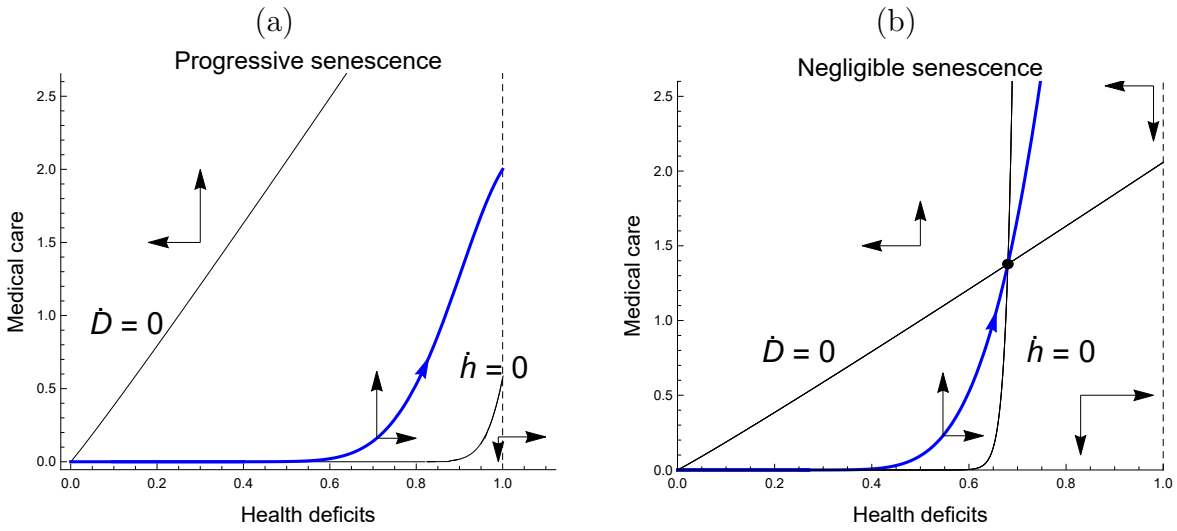


Figure 3: Progressive and negligible senescence: phase diagrams. Panel (a): Health investment increases over time until health deficits eventually hit the maximum sustainable level D^{max}. Panel (b): Health investment increases over time until reaching a stationary level of health deficits. Thick blue lines: optimal medical care; Thin black lines: nullclines $\dot{h} = \dot{D} = 0$. For this and the subsequent figures $U(c) = \frac{c(t)^{1-\sigma}}{1-\sigma} + b$ for the utility of consumption and $S(D) = \frac{1+\alpha}{1+\alpha e^{\phi D}}$ for biological aging, with $\mu = \frac{3}{100}$, $r = \frac{3}{50}$, $q = \frac{1}{50}$, $\rho = \frac{1}{25}$, $p = 1$, $Y = 1$, $\sigma = \frac{9}{10}$, $\gamma = \frac{24}{25}$, $\alpha = \frac{1}{100}$, $\phi = 4$, $\eta = 2$, $a = 0$, $b = 0$, $D^{\max} = 1$. For panel (a): $A = \frac{1}{4}$; for panel (b): $A = \frac{1}{2}$

ians (Barbi et al., 2018). In particular, note that our model of deficit accumulation with uncertain lifetime does not change the main predictions from the standard health deficit model that medical care increases with age, a prediction in line with observable life time pattern of medical care (e.g. Dalgaard and Strulik, 2014; Schünemann et al., 2017). The main difference is that the realistic uncertain lifetime assumption allows to better capture some end-of-life patterns that are empirically observed, in which medical care, consumption and hazard rates reach a plateau in the long run.

The second approaching path depicted in panel (b) of Figure 3 is characterized by an agent beginning her life being very unhealthy. In such a case, the level of health deficits is larger than the steady state level, and it is necessary to reverse the process of biological aging by spending most income on medical care and very little on consumption. In terms of predictions, this would produce an odd pattern: medical care levels are so high (and effective) that the process of biological aging is reversed and, despite the agent becoming chronologically older, her body becomes biologically 'younger'. This paradoxical result of decreasing health deficits and decreasing medical care is typically not observed,

but it is a theoretical possibility that can emerge under the non trivial assumptions that the medical technology is advanced enough and that there are no economic constraints, including liquidity constraints.

The two simulated scenarios depicted in Figure 3 differ by the choice of the efficiency parameter A. Progressive senescence results when A is low, while negligible senescence is possible when A is high enough. This allows to highlight a direct path to life extension, which is heavily discussed in medical science and gerontology: improvements in the repair of health damages, achieved, for example, through elimination of damaged cells, telomerase reactivation, or mitophagy (the removal of damaged mitochondria). While these therapeutic strategies are not yet fully developed, it is likely that they will be available at some point in the future. In section 5.3 we explore this possibility considering a scenario in which, starting from a low level of medical efficiency, medical technology improves over time until negligible senescence becomes feasible. This approach is consistent with the finding that medical research on aging advanced greatly over the last 20 years. The biological mechanisms of health deficit accumulation are now well understood and for most of the gateways of bodily decay solutions have been suggested and explored in animal studies (See Lopez-Otin et al., 2013, for a detailed discussion). The observation that natural scientists started to envision the postponement of aging by health interventions, has motivated us to explore the economic theory of health deficit accumulation in this direction.

4 Comparative dynamics: Formulas to compute the response on impact and in the long run

In the previous section we have shown the conditions under which planning over a long (eventually infinite) time horizon is meaningful. In this Section we show how to perform comparative dynamics using our model of endogenous aging with uncertain lifetime. We consider an unexpected permanent shock on a generic parameter ω and we investigate how medical care and consumption are affected by changes in the economic and technological environment.

A considerable advantage of our model is that it allows to study the determinants of longevity *analytically*, without resorting to numerical simulations. Hence we can provide general formulas for (i) the *impulse response*, i.e. the short run response of medical care at the time of the shock, for given health condition $D = D_0$, and (ii) the *long run response*,

i.e. the change in the steady state medical care and health deficit accumulation.

To derive the change in medical care and the level of deficits at the steady state, we implement the comparative dynamics procedure described in Dragone and Vanin (2015). Essentially, it requires applying the implicit function theorem to the system of equations 16 to 18. Recall that $\mathcal{J}$ is the Jacobian matrix associated to 16 to 18 and define

$$\mathcal{J}_{h,\omega} \equiv \begin{bmatrix} \frac{\partial \dot{h}}{\partial \omega} & \frac{\partial \dot{h}}{\partial k} & \frac{\partial \dot{h}}{\partial D} \\ \frac{\partial \dot{k}}{\partial \omega} & \frac{\partial \dot{k}}{\partial k} & \frac{\partial \dot{k}}{\partial D} \\ \frac{\partial \dot{D}}{\partial \omega} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial D} \end{bmatrix}, \qquad \mathcal{J}_{D,\omega} \equiv \begin{bmatrix} \frac{\partial \dot{h}}{\partial h} & \frac{\partial \dot{h}}{\partial k} & \frac{\partial \dot{h}}{\partial \omega} \\ \frac{\partial \dot{k}}{\partial h} & \frac{\partial \dot{k}}{\partial k} & \frac{\partial \dot{k}}{\partial \omega} \\ \frac{\partial \dot{D}}{\partial h} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial \omega} \end{bmatrix}. \tag{26}$$

After a permanent shock on a general parameter ω, the steady state level of medical care and health deficits change as follows (see Dragone and Vanin, 2015):

Lemma 2 (Long run response) *After an unexpected permanent change in parameter ω, the long run medical care and level of deficits change as follows:*

$$h^{ss}_{\omega} = -\frac{|\mathcal{J}_{h,\omega}|}{|\mathcal{J}|}, \qquad D^{ss}_{\omega} = -\frac{|\mathcal{J}_{D,\omega}|}{|\mathcal{J}|}, \tag{27}$$

where the determinants $|\mathcal{J}|$, $|\mathcal{J}_{h,\omega}|$ and $|\mathcal{J}_{D,\omega}|$ are computed at the steady state before the shock takes place.

Given that $|\mathcal{J}|$ is negative because of saddlepoint stability, the sign of the response of the steady state to a change in ω depends on the sign of the numerator of the two equations in 27. As shown in the following sections, this task can be carried out easily. Assessing the impulse response to a shock is more complicated, as in principle it requires knowing the explicit expression of the policy function directed toward the steady state. Unless under special circumstances, this expression is generally not available, which may explain why impulse response analysis to shocks is often conducted through numerical simulations.

In the following Proposition we show that a numerical approach is not necessary to study impulse response functions, as analytical sufficient conditions can be provided. The advantage of our approach is that it does not require explicit knowledge of the saddle path in closed form, nor it needs numerical simulations. In the realistic case where the initial level of deficits is lower than the long-run level, the following holds:

Proposition 3 (Impulse response) *After an unexpected permanent change in parameter ω, the impulse response of medical care h^0_{ω} is:*

$$h^0_{\omega} = h^{ss}_{\omega} - xD^{ss}_{\omega} - \int_{D_0}^{D^{ss}} \frac{\partial}{\partial \omega}\left(\frac{d\hat{h}}{dD}\right) dD. \tag{28}$$

where x is the linearized slope of the policy function at the steady state and $\frac{d\hat{h}}{dD}$ is the slope of the policy function along the path to the steady state.

The following Corollary describes sufficient conditions under which a non-ambiguous prediction on the impulse response can be made.

Corollary 2 (Sufficient conditions) *On impact, after an exogenous permanent shock:*

- *Medical care increases if $h^{ss}_{\omega} - xD^{ss}_{\omega} > 0$ and $\frac{\partial}{\partial \omega}\left(\frac{d\hat{h}}{dD}\right) < 0$ for all $D \in (D_0, D^{ss})$;*
- *Medical care decreases if $h^{ss}_{\omega} - xD^{ss}_{\omega} < 0$ and $\frac{\partial}{\partial \omega}\left(\frac{d\hat{h}}{dD}\right) > 0$ for all $D \in (D_0, D^{ss})$.*

Proposition 3 shows that assessing the impulse response of medical care to a parameter change essentially requires knowing three bits of information: how steady state medical care and health deficits respond to the shock, and how the slope of the policy function changes. Information on the response of the steady state is obtained using Proposition 2; while information on the policy function is obtained by exploiting the time-elimination method presented in Barro and Sala-i-Martin (1995). Essentially, it requires taking the ratio $\dot{h}/\dot{D}$ using equations 16 and 18, and studying how the ratio changes when ω increases (see the Appendix for details).

To understand equation 28, consider the simple case in which the steady state does not change when perturbing ω. In such a case, the first two terms are zero. Hence, if medical care becomes, say, more sensitive to changes in health deficits after the shock (i.e. the policy function becomes more steep), on impact medical care is predicted to decrease.

Figure 4 shows the general case in which also the steady state shifts. The overall response on impact is described by the vertical jump from the original saddle path (in blue) to the new one (in red), as represented by the vertical distance between points A and B. The impulse response depends on three terms. The first term h^{ss}_{ω} is the change in medical care at the steady state (vertical distance between S' and G). The second term xD^{ss}_{ω} represents how steady state medical care changes as a consequence of a change in the steady state deficits (vertical distance between point G and H).[11] The net effect of the first and second term, represented by the vertical distance HS', describes the effect of the shock on the extensive margin. The third term $\int_{D_0}^{D^{ss}} \frac{\partial}{\partial \omega}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right)$represents the impact of the shock

[11] Point G is obtained by multiplying the change in the steady state level of deficits and the slope x of the original policy function at the original steady state.

on the intensive margin. Formally, it is described as the cumulative change in the slope of the policy function over the interval $[D^0, D^{ss}]$, and it can be computed as the difference between two medical care "gaps". The first gap is FC, which measures the medical care gap over the interval $[D^0, D^{ss}]$ *after* the shock has occurred. The second gap is SE, which measures the medical care gap over the interval $[D^0, D^{ss}]$ *before* the shock has occurred.

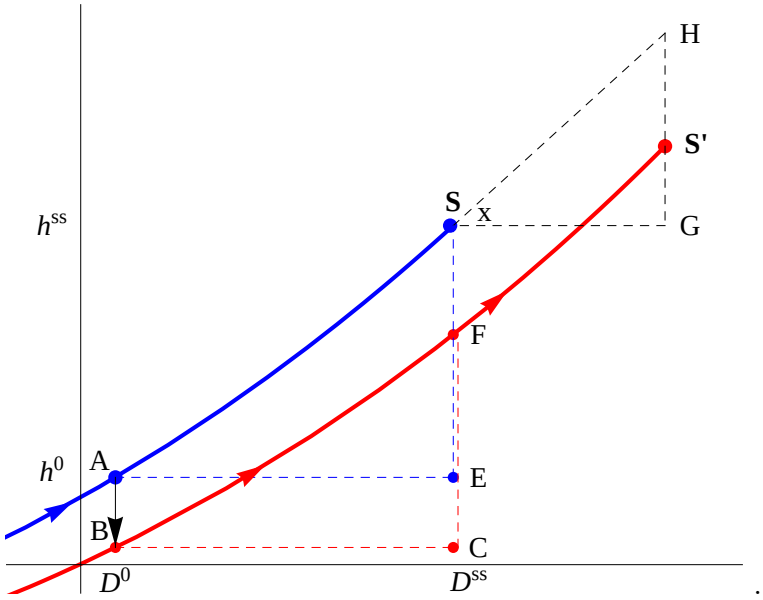


Figure 4: Response of medical care to an exogenous shock. A: Initial point A; Original steady state S; New steady state S'. Response on impact: Vertical distance AB; Response of steady state medical care: vertical distance GS'; Response of steady state health deficits: horizontal distance SG.

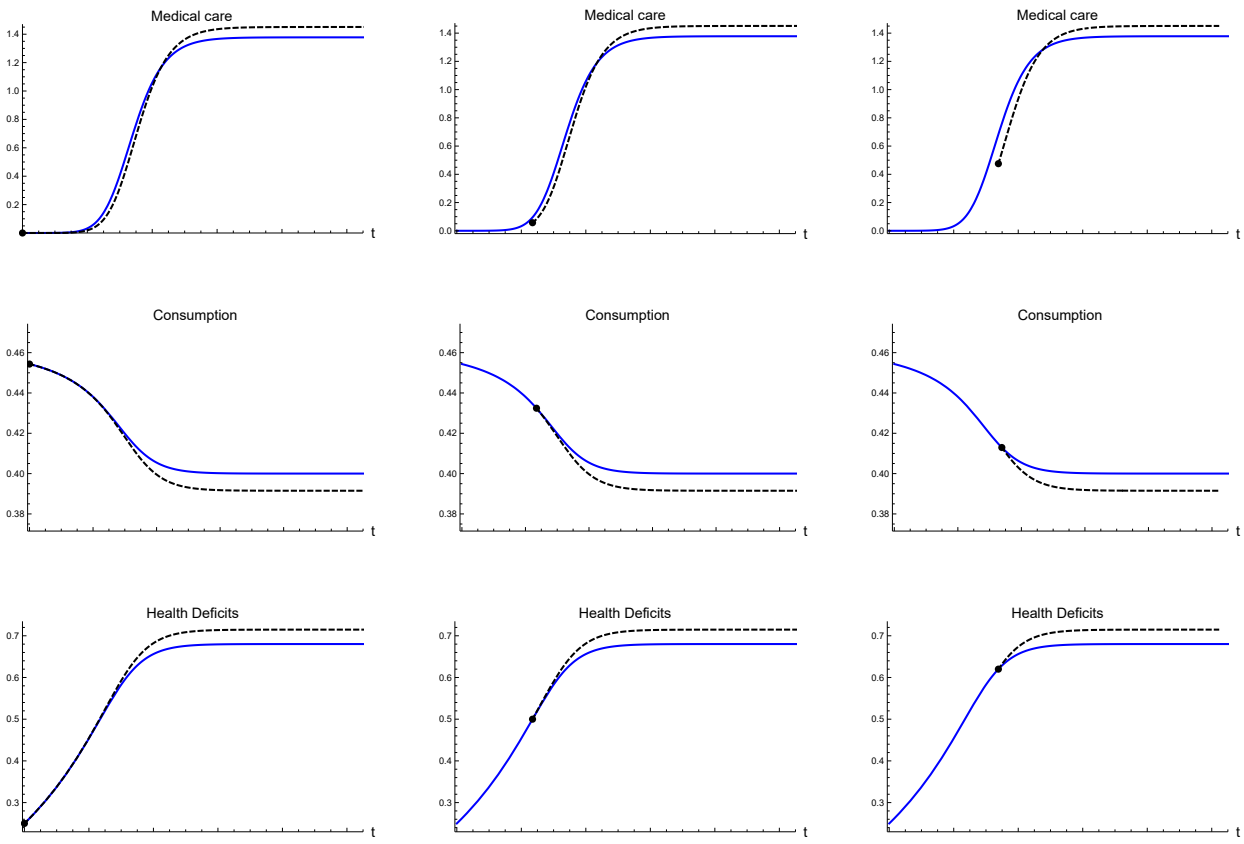
5 Studying the determinants of longevity

In the proceeding we study how two key determinants of longevity affect medical care choices and the accumulation of health deficits. The first key determinant is the price (p) of health care, which allows highlighting the role of a change in the relative price of medical care with respect to consumption. The second one is the productivity (A) of medical care in slowing down the process deficit accumulation. We will first consider the impact of a one-off improvement in medical technology, and then consider an augmented model in which medical technology is expected to improve over time and make negligible senescence becomes feasible.

5.1 Increasing medical care costs

In the following we consider the case in which medical care becomes more expensive. All statements will be reversed in sign in case medical care becomes cheaper. Using the formulas presented in the previous Section, the following holds for any utility function and survival probability:[12]

Proposition 4 *If medical care becomes more costly, medical care will be lower on impact, but higher in the long run. Over the lifetime biological aging will be faster.*



(a) shock when young (b) shock when middle-aged (c) shock when old

Figure 5: More expensive medical care. Solid lines: initial time trajectories; Dashed lines: new time trajectories after the price shock (from $p = 1$ to $p = 1.1$). Left panels: shock when young, central panels: shock when middle-aged, right panels: shock when old. Parameters and functional forms as for Figure 3.

Adjustments after a price change can be decomposed into an immediate response as jump from the old to the new saddlepath and a movement along the new saddlepath towards

[12] We consider the case in which the initial level of deficits is low and an internal steady state of negligible senescence exists.

the new steady state (see Figure 5). It is intuitive that individuals respond immediately to increasing health care prices by demanding less health care. However, when individuals demand less health care, they accumulate more health deficits and converge to a steady state where health deficits are larger such that they demand more health care in the long-run. Proposition 4 states that this is generally the case. The intuition is that health deficits are self-productive (Dragone and Vanin, 2015), which means that an increase in deficits speeds up the accumulation of further deficits. The immediate response to increasing health care prices is to demand less health care. However, when individuals demand less health care, they accumulate more health deficits and converge to a steady state where health deficits are larger, such that in the long-run they demand more health care.

As an illustration of the results of Proposition 4, Figure 5 depicts the effect of 10% increase in the cost of medical care (from $p = 1$ to $p = 1.1$) in three different scenarios: when the agent is young, middle-aged or old (or, more precisely, when she has few, some, or many deficits). With respect to Figure 3, now all graphs are represented as functions of chronological age. This allows to emphasize that, although calendar and biological aging (i.e. the level of health deficits accumulated at a certain age) are positively correlated, they do not coincide, as shown in the panels in the last row .

In all scenarios, on impact medical care drops as a response to the higher price of medical care, while consumption is not affected (since it depends only on the current level of health deficits). As a consequence of the initial period of reduced health care, health deficits accumulate at a faster rate. Over time, this will also drive medical care to increase and consumption to decrease. In fact, the effects of more expensive medical care are persistent, and the initially lower level of medical care is not compensated as the agent ages. Over the long run the agent will still aim at a steady state of constant health-deficits and health care, but such steady state features a higher level of deficits, it requires more health care, and it is associated with a higher endogenous hazard rate and lower expected utility $S(D)U(c)$.

Although the short and long run qualitative response is similar in all three scenarios, the magnitude of the short run response of medical care differs: it is smaller when the agent is young, and larger when she is old. This is not due to wealth or income considerations, which are ruled out by the Frisch compensation, but due to the fact that optimal medical care is more sensitive to the accumulation of deficits at higher level of deficits (see Figure 3). Despite these quantitative differences in immediate response depending on age, individual arrive at the same steady state. This implies that the price shock has about the same consequences on health deficits in old age, irrespective of whether it was experienced in young,

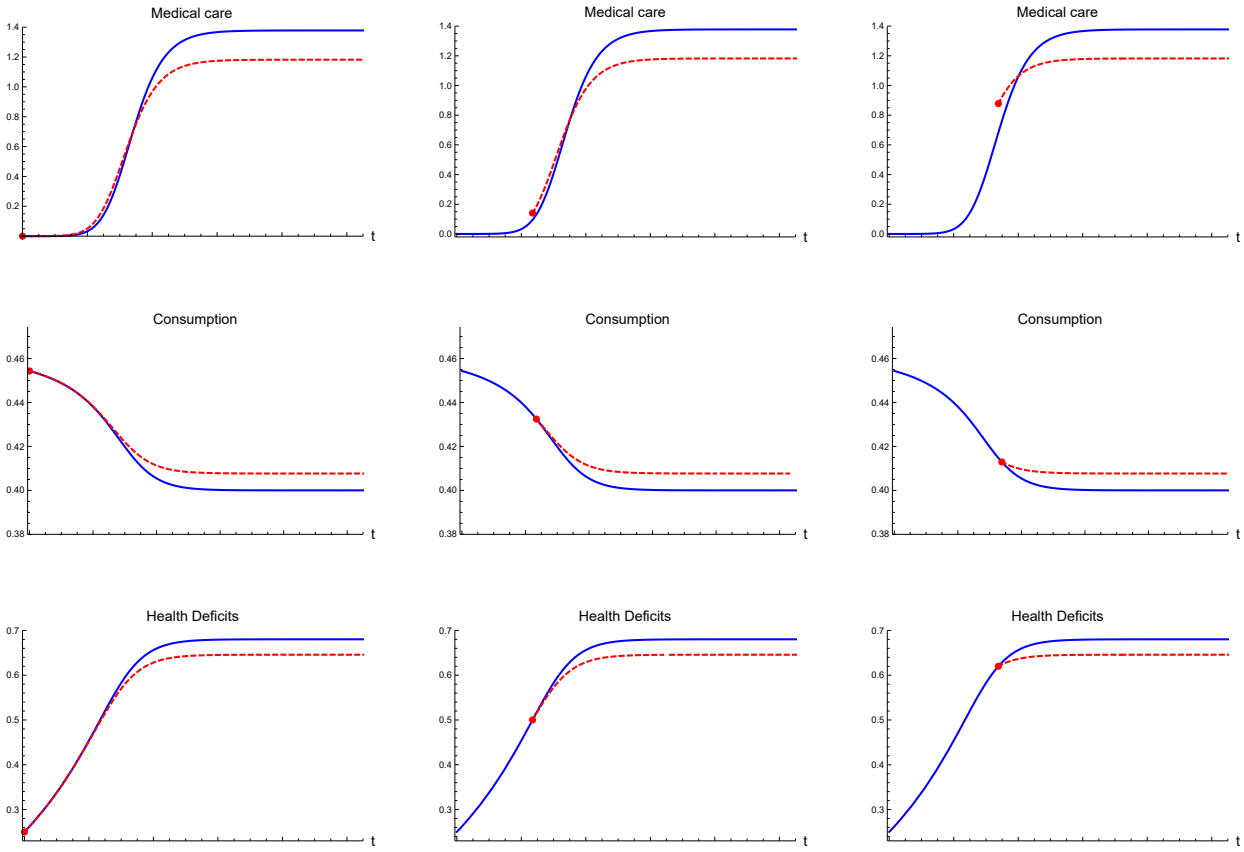
middle or old age. These results illustrate that it is important to consider and disentangle short- and long-run responses on price shocks and they demonstrate the usefulness of the here proposed method of analysis.

5.2 Improvements in medical technology

We next discuss the comparative dynamics of an improvement of medical technology. Formally, this can be investigated by considering the effect of an increase in A or in γ. The former term refers to the general power of medical care in maintaining and repairing the human body, while the latter one determines the degree of decreasing returns of health care. In the following Proposition and in Figure 6 we focus on a one-shot increase in A. We will then consider an augmented version of the model and allow for medical technology to constantly improve over time.

Proposition 5 *Suppose medical technology is good enough, $\gamma > \gamma_A$. After a positive shock on medical technology, medical care will be higher on impact, but it will be lower in the long run. Over the lifetime, biological aging will be slower.*

Figure 6 shows the adjustment paths corresponding to an improvement in medical technology. Similar to the case of a price shock, the qualitative response of medical care does not depend on the timing of the technological shock. The magnitude of the short run response depends on age: it is larger when the agent is old and has more deficits, and smaller for the young and healthy agent. For an intuition of the adjustment dynamics it may be helpful to recall that health deficits are a (slow-moving) state variable. At the point of time when the individual experiences a positive shock of health technology, the state of health is given and the individual responds to the improved efficiency of health care by increasing medical care in the short run. The short run complementarity between medical technology and medical care allows to persistently slow down the accumulation of deficits. In the long run, both medical care and the level of deficits will be lower than they would be without the technological improvement, and consumption will be higher. As consumption will be higher and deficits lower at each point in time after the technology improvement, the value of life increases.



(a) shock when young (b) shock when middle-aged (c) shock when old

Figure 6: Better medical technology. Solid lines: initial time trajectories; Dashed lines: new time trajectories after the technological improvement (from $A = 0.5$ to $A = 0.55$). Left panels: shock when young, central panels: shock when middle-aged, right panels: shock when old. Parameters and functional forms as for Figure 3.

5.3 Constant progress in medical technology

A crucial feature of the model presented in the previous sections is that medical technology needs to be powerful enough to obtain a steady state of negligible senescence. We now consider the case in which current medical technology is not yet sufficient to sustain a state of negligible senescence but individuals can foresee that it will constantly improve over time. Suppose, for example, that future medical efficiency will evolve over time according to the following equation:

$$\dot{A} = g\left(\hat{A} - A\right), \tag{29}$$

where $\hat{A}$ is an upper bound for medical efficiency and the parameter $g \in (0, 1)$ controls the rate of medical progress. Let $\bar{A}$ denote the threshold level for medical technology

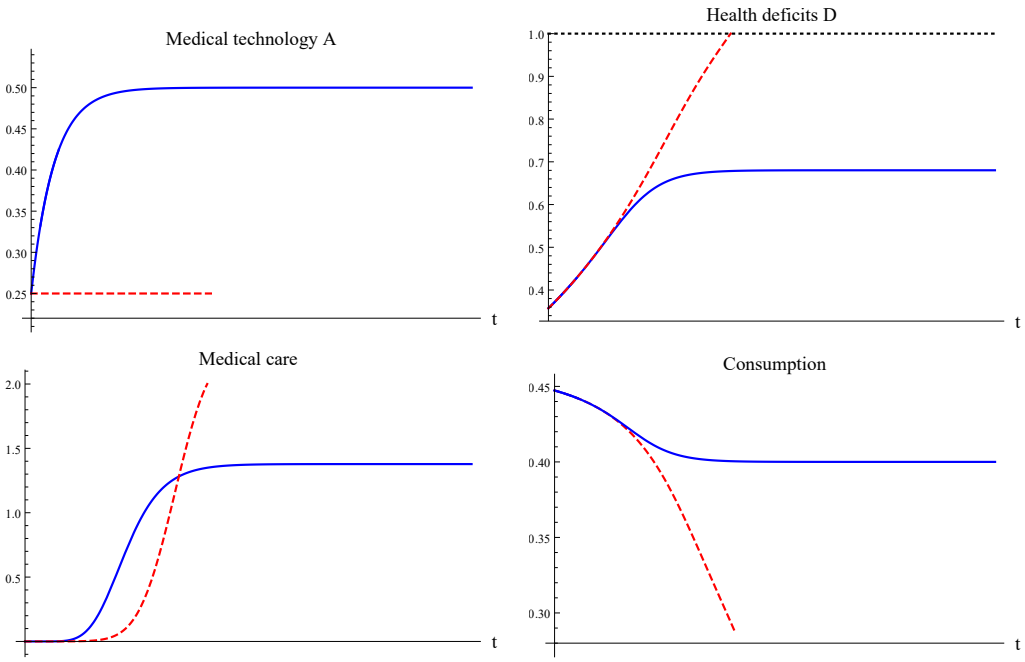


Figure 7: Constant medical progress. When medical technology is not efficient enough, progressive senescence results, as described by the dashed (red) trajectories of health deficits and medical care. The solid blue line represents the case in which, starting from a progressive senescence scenario ($A_0 = \frac{1}{4}$), medical progress is expected to improve until negligible senescence becomes feasible ($\hat{A} = \frac{1}{2}$). The two scenarios have the same initial values of deficits, consumption and medical technology. Parameters and functional forms as for Figure 3, $g = \frac{1}{5}$, $\hat{A} = \frac{1}{2}$.

above which a steady state of negligible senescence exists. The problem sketched above is captured by the initial condition $A(0) < \bar{A} < \hat{A}$, i.e. the initial medical technology is too low to guarantee a steady state of negligible senescence, but it will improve over time until eventually getting to a state where negligible senescence can be maintained.

Accordingly, we augment the endogenous aging problem 7 to 10 with dynamics of medical progress 29. We consider the case $r > \mu$, i.e. the case in which a saddle point stable steady state can emerge, and we discuss the features of the solution by way of a numerical example. In Figure 7, solid lines show the predicted time path of medical technology, health deficits, medical care, and consumption for evolving medical technology. For comparison, dashed lines show the solution of the same model for constant technology, $A = A_0$ for all t (this case corresponds to the one depicted in panel (a) of Figure 3). In this case, there is no reachable steady state of negligible senescence and the problem requires the transversality conditions to be fulfilled at finite time $\bar{T}$, when the agent dies. Thus,

dashed lines represent a solution of the original health deficit model (Dalgaard and Strulik, 2014; Strulik, 2015b).

When medical technology stays constant, health expenditure increases steeply, as predicted by the Dalgaard-Strulik model (lower left panel in Figure 7). Despite these health investments, health deficits accumulate with increasing age in a quasi-exponential way since medical technology is not good enough to stop the accumulation process (upper right panel). The dashed time series end when health deficits reach the maximum sustainable limit at $D^{\max}$ and death is inevitable (although for most individuals death has stochastically occurred before).

When medical technology is expected to improve over time and to eventually reach $\hat{A}$, the individual knows that she needs to invest heavily in her health in order to increase the probability to be still alive when the steady state of negligible senescence comes into existence. In mid-age the agent thus invests more in health (lower left panel) although medical technology is already much more efficient than the constant technology (upper left panel). Eventually, spending as well as health deficit accumulation (upper right panel) slow down and reach a plateau, as discussed for the benchmark model in the previous sections.

6 Conclusion

In this paper we have discussed optimal life cycle medical care in a model where individuals do not know, nor plan, when they are going to die. Formally, we have shown that this can be modeled by allowing infinite life to be a meaningful goal. While humankind had always longed for transcending death, for most time in history these aspirations were confined to religious beliefs and the afterlife. Now, in the 21st century, income and medical progress have advanced far enough that natural scientists as well as philosophers discuss for the first time seriously the possibilities and consequences of an infinite life on earth (Harari, 2016). Naturally, it are wealthy entrepreneurs who have the least problems in imagining and aspiring (infinite) life extension, see Friend (2017). Here we integrated into a simple life cycle model a gerontologically founded law of motion of human aging and showed that a reachable steady state of infinite life requires that the rate of health deficit accumulation falls short of the interest rate and that medical technology is good enough. The simple model allows to assess the steady state's characteristics and comparative dynamics analytically. We used this feature to discuss impulse responses to advances in medical technology and increasing health care costs.

The model conceptualizes biological aging as the accumulation of health deficits. Adjustment dynamics towards a steady state of constant health, $D^{ss} > D(0)$, are characterized as the continuous repair of health deficits resulting from "natural aging". This view is in contrast to the conventional model of health capital accumulation (Grossman, 1972) but in line with the notion of aging in modern gerontology. Our model with endogenous survival probability model differs from the "perpetual youth" model of conventional macroeconomics (Yaari, 1965) where people do not age and death occurs because of age-unrelated background mortality. Our approach differs also from the conventional modeling of aging in health economics where people either inevitably die by a finite T at the latest, or inevitably live forever. In the standard model of health capital accumulation (Grossman, 1972) there always exists a steady state of constant health such that individuals inevitably live forever (Strulik, 2015a). The reason is that for a given rate of health capital depreciation δ, individuals in bad health lose relatively little health, i.e. their health depreciation δH is low when health capital H is low. This creates an equilibrating force and convergence to a steady state of constant health. Typically, the health capital literature imposes a finite time horizon T and thus enforces a finite life. In the health deficit model of Dalgaard and Strulik (2014) a steady state of constant health exists not always but only for a favorable constellation of parameters such as medical technology. So far, the health deficit literature has focused on parameter constellations where the steady state does not exist and thus, by design, it has also assumed a finite life.

In contrast, optimistic scholars such as de Grey (2013) conceptualize medical gerontology as the endeavor to repair bodily deficits, which, once it succeeds sufficiently well, will end aging. Here we have proposed a simple model that integrates these ideas into an economic life cycle theory for the future.

7 Appendix

7.1 Transforming the objective function

To transform the expected intertemporal utility function into an intertemporal expected utility function, exploit the definition of the expectation operator and the resulting double integral:

$$\begin{aligned}\mathbb{E}_g\left[\int_0^T e^{-\rho t}U(c)\,\mathrm{d}t\right] &= \int_0^\infty g(D,T)\left(\int_0^T e^{-\rho t}U(c)\,\mathrm{d}t\right)\mathrm{d}T \\ &= \int_0^\infty e^{-\rho t}U(c)\left(\int_t^\infty g(D,T)\,\mathrm{d}T\right)\mathrm{d}t \\ &= \int_0^\infty e^{-\rho t}\mathcal{S}(D,t)\,U(c)\,\mathrm{d}t \end{aligned} \tag{30}$$

7.2 Proof of Proposition 1

When $\mathcal{S}(D,t) = e^{-qt}S(D)$, the agent's objective function can be written as

$$\int_0^\infty e^{-(\rho+q)t}S(D)\,U(c)\,dt.$$

We can therefore construct the associated current-value Hamiltonian function is:

$$H = S(D)\,U(c) + \lambda\dot{D} + \eta\dot{k}, \tag{31}$$

where $\lambda = \lambda(t)$ and $\eta = \eta(t)$ are the costate variables associated with the dynamics of health deficits and capital, respectively. The corresponding necessary conditions for an internal solution read as (subscripts denote partial derivatives, the arguments are henceforth omitted):

$$h^* \;:\quad H_h = 0 \quad\Leftrightarrow\quad \lambda\gamma\mu A h^{\gamma-1} = -p\eta \tag{32}$$

$$c^* \;:\quad H_c = 0 \quad\Leftrightarrow\quad SU_c = \eta, \tag{33}$$

with $\eta > 0$ and $\lambda \leq 0$. Concavity of the Hamiltonian function requires $U_{cc} < 0$, $\mathcal{S}_{DD} < 0$ and

$$\Phi \equiv \mathcal{S}_D^2 U_c^2 - \mathcal{S}UU_{cc}\mathcal{S}_{DD} < 0. \tag{34}$$

Hence $U\mathcal{S}_{DD} < 0$ is required for concavity.

For later reference, note that

$$\frac{\partial c^*}{\partial D} = -\frac{S_D}{S}\frac{U_c}{U_{cc}} = \mathcal{Z}\frac{U_c}{U_{cc}} \tag{35}$$

where

$$\mathcal{Z}\left(D\left(t\right),t\right)\equiv-\frac{\mathcal{S}_{D}\left(\cdot\right)}{\mathcal{S}\left(\cdot\right)}=-\frac{S_{D}}{S}>0 \tag{36}$$

From the first order conditions 32 and 33 we obtain the optimal value of medical care h and consumption c as functions of the state variables, the costate variables and the survival probability. Note that both optimal medical care and consumption do not directly depend on capital, but they depend on its evolution through the shadow price $\eta>0$. The necessary conditions for the costate dynamics are

$$\dot{\lambda} = \lambda\left(\rho+q\right)-H_{D}=\left(\rho+q-\mu\right)\lambda-\mathcal{S}_{D}U \tag{37}$$

$$\dot{\eta} = \eta\left(\rho+q\right)-H_{k}=\left(\rho+q-r\right)\eta \tag{38}$$

plus the transversality condition $\lim_{t\to\infty}H\left(t\right)=0$. Differentiating 32 and 33 with respect to time, and using 37 and 38 yields:

$$\dot{h} = \frac{h}{1-\gamma}\left(r-\mu-\frac{\gamma\mu Ah^{\gamma-1}}{p}\frac{U}{U_{c}}\mathcal{Z}\right) \tag{39}$$

$$\dot{c} = -\frac{U_{c}\left(c\right)}{U_{cc}\left(c\right)}\left[\rho+q-r-\mathcal{Z}\dot{D}\right] \tag{40}$$

$$\dot{D} = \mu\left(D-a-Ah^{\gamma}\right) \tag{41}$$

$$\dot{k} = rk+Y-ph-c. \tag{42}$$

Using the definitions of exogenous and endogenous hazard rate the above system can equivalently written as

$$\dot{h} = \frac{h}{1-\gamma}\left(r-\mu-\frac{\gamma\mu A}{p}\frac{U}{U_{c}}\mathcal{Z}h^{\gamma-1}\right) \tag{43}$$

$$\dot{c} = -\frac{U_{c}}{U_{cc}}\left(r-\rho-q-\mathcal{Z}\dot{D}\right) \tag{44}$$

$$\dot{D} = \mu\left(D-a-Ah^{\gamma}\right) \tag{45}$$

$$\dot{k} = rk+Y-ph-c, \tag{46}$$

In the steady state(s) the above equations are equal to zero. Since $\frac{\gamma\mu A}{p}\frac{U}{U_{c}}\mathcal{Z}h^{\gamma-1}>0$, a necessary condition for an internal steady state to emerge is $r>\mu$ (eq. 43). Note also that, when health deficits are constant over time ($\dot{D}=0$), then equation 44 is zero only if $r=\rho+q$. In a macroeconomic framework one can reasonably assume that the interest rate is a function of k, in which case the steady state is reached when $r\left(k\right)=\rho+q$ (as in a Ramsey model). To retain the microeconomic flavour of this paper, we assume $r=\rho+q$. As a consequence, the dynamics of consumption is determined by the dynamics of D, i.e.

$\dot{c} = \frac{U_c}{U_{cc}}(\mathcal{Z}\dot{D})$, and the determinant of the following Jacobian matrix, when computed at the steady state, is:

$$\mathcal{J}_0 = \begin{bmatrix} \frac{\partial \dot{D}}{\partial D} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial h} & \frac{\partial \dot{D}}{\partial c} \\ \frac{\partial \dot{k}}{\partial D} & \frac{\partial \dot{k}}{\partial k} & \frac{\partial \dot{k}}{\partial h} & \frac{\partial \dot{k}}{\partial c} \\ \frac{\partial \dot{h}}{\partial D} & \frac{\partial \dot{h}}{\partial k} & \frac{\partial \dot{h}}{\partial h} & \frac{\partial \dot{h}}{\partial c} \\ \frac{\partial \dot{c}}{\partial D} & \frac{\partial \dot{c}}{\partial k} & \frac{\partial \dot{c}}{\partial h} & \frac{\partial \dot{c}}{\partial c} \end{bmatrix} \tag{47}$$

$$= \begin{bmatrix} \mu & 0 & -A\mu\gamma\left(h^{ss}\right)^{\gamma-1} & 0 \\ 0 & r & -p & -1 \\ -\frac{\gamma}{1-\gamma}\frac{A(h^{ss})^{\gamma}\mu}{p}\frac{U}{U_c}\mathcal{Z}_D & 0 & \frac{1}{1-\gamma}\left(r-\mu-\frac{A(h^{ss})^{\gamma-1}\mu\gamma^2}{p}\frac{U}{U_c}\mathcal{Z}\right) & \frac{A(h^{ss})^{\gamma}\mu\gamma}{p(1-\gamma)}\frac{UU_{cc}-U_c^2}{U_c^2}\mathcal{Z} \\ \mu\mathcal{Z}\frac{U_c}{U_{cc}} & 0 & \left(Ah^{\gamma-1}\mu\gamma\right)\frac{U_c}{U_{cc}}\mathcal{Z} & 0 \end{bmatrix}$$

However, the determinant of the above Jacobian is nil ($|\mathcal{J}_0| = 0$). Hence we exploit the fact that consumption tracks health deficits to reduce the dimensionality of the problem. Replacing $c^* = C(D)$ in 43, 45 and 46 yields:

$$\dot{D} = \mu\left(D - a - Ah^{\gamma}\right) \tag{48}$$

$$\widetilde{\dot{k}} = rk + Y - ph - C(D) \tag{49}$$

$$\widetilde{\dot{h}} = \frac{h}{1-\gamma}\left(r - \mu - \frac{\gamma\mu A}{p}\frac{U\left(C(D)\right)}{U_c\left(C(D)\right)}\mathcal{Z}h^{\gamma-1}\right) \tag{50}$$

Note that, since there are three equations and four variables (h, c, D and k), the steady state of the Frisch problem is in principle indeterminate. This can be solved by assuming that assets go to some non negative value in the infinite limit. Under this assumption it is possible to pin down the life-cycle budget constraint (or, equivalently, η) and the steady state value can be uniquely determined.

To obtain the steady state, set $\dot{D} = \widetilde{\dot{k}} = \widetilde{\dot{h}} = 0$. When $\mu > r$, there exists no internal steady state. When instead $\mu < r$, steady states can exist, depending on the functional specifications and parameters. When an internal steady state exists, checking for saddle point stability requires computing the 3×3 Jacobian matrix $\mathcal{J}$ at the steady state. Exploiting the definitions of Φ and $\mathcal{Z}$ (eqs. 34 and 36) implies:

$$\mathcal{J} = \begin{bmatrix} \frac{\partial \dot{D}}{\partial D} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial h} \\ \frac{\partial \widetilde{\dot{k}}}{\partial D} & \frac{\partial \widetilde{\dot{k}}}{\partial k} & \frac{\partial \widetilde{\dot{k}}}{\partial h} \\ \frac{\partial \widetilde{\dot{h}}}{\partial D} & \frac{\partial \widetilde{\dot{h}}}{\partial k} & \frac{\partial \widetilde{\dot{h}}}{\partial h} \end{bmatrix}$$

$$= \begin{bmatrix} \mu & 0 & -A\gamma h^{\gamma-1}\mu \\ -\frac{U_c}{U_{cc}}\mathcal{Z} & r & -p \\ -\frac{A\gamma h^{\gamma}\mu}{1-\gamma}\frac{\Phi}{pS^2U_cU_{cc}} & 0 & \frac{1}{1-\gamma}\left(r-\mu-\frac{A\mu\gamma^2h^{\gamma-1}U}{pU_c}\mathcal{Z}\right) \end{bmatrix}. \tag{51}$$

The associated determinant is

$$|\mathcal{J}| = \frac{r\mu}{1-\gamma}\left(r-\mu-\frac{Ah^{\gamma}\Phi - SUS_DU_{cc}}{pS^2U_cU_{cc}}A\mu\gamma^2h^{\gamma-1}\right). \tag{52}$$

For saddlepoint stability, $|\mathcal{J}| < 0$ must hold at the steady state. Replacing $h = h^{ss}$ from (21) and manipulating (52) yields

$$|\mathcal{J}^{ss}| = \mu r\,(r-\mu)\,(1-K) \tag{53}$$

where

$$K = -\frac{A\gamma h^{\gamma}\Phi}{(1-\gamma)\,SUS_DU_{cc}} > 0 \tag{54}$$

Hence $|\mathcal{J}^{ss}| < 0$ if and only if $K > 1$. When this is the case, the steady state is saddle point stable and one eigenvalue associated with the 3×3 Jacobian matrix $\mathcal{J}^{ss}$, denoted with ε, is negative. Let (x_1, x_2, x_3) be the associated eigenvector at the steady state. Then the slope of the policy function in the (D, h) space, in the neighborhood of the steady state, is

$$x \equiv \frac{x_1}{x_3} = \frac{\mu-\varepsilon}{\gamma\mu A}\,(h^{ss})^{1-\gamma} > 0. \tag{55}$$

This completes the proof of Proposition 1.

Note that expression 53 can be used to determine zones in the (D, h) space where an internal steady state, if it exists, is saddle point stable ($|\mathcal{J}^{ss}| < 0$). In the text and in section 5.3 we refer to the role of medical technology A. Solve $|\mathcal{J}^{ss}| = 0$ for A and let $\bar{A}$ be the largest of the two roots (the smaller root is negative). Then one can establish that $|\mathcal{J}^{ss}| < 0$ if $A > \bar{A}$.

7.3 Existence and stability of internal steady states for a negative utility function

In the main text we normalize the utility after death to zero and consider the case of positive utility values. However, existence of a steady state of negligible senescence is possible also when U is negative. In fact, from (60), condition $h^{ss} > 0$ implies the following

Corollary 3 *$U(c^{ss})(r-\mu) > 0$ is a necessary condition for a steady state of negligible senescence to exist.*

Hence, with negative utility a necessary condition to hold is $\mu > r$, which is the opposite condition with respect to the case of positive utility. Suppose a steady state exists. It is saddle point stable if

$$|\mathcal{J}^{ss}| = \mu r\,(r-\mu)\,(1-K) < 0. \tag{56}$$

When $U < 0$, then $K < 0$ (see eq. 54) and the above condition is always satisfied. Hence the following holds:

Proposition 6 *Consider $U < 0$. If $\mu > r$ and conditions (21) to (24) hold, a steady state of negligible senescence exists and is always saddle point stable. If $\mu < r$, no steady state of negligible senescence exists.*

To sum up, the case in which the utility function has negative values is compatible with existence of a reachable state of negligible senescence. This requires the force of aging being larger than the interest rate ($\mu > r$) and, for concavity to hold, a convex survival function, so that $U(c) S_{DD}(D) < 0$.

7.4 Proof of Proposition 3

To assess the impulse response to a generic parameter change, i.e. how the optimal path of medical care $\hat{h}$ leading to the steady state changes when the parameter ω changes, we proceed in three steps.[13] First, recall that the path of both optimal medical care and optimal consumption converging to the steady state is in principle a function of the two state variables D and k. Due to the Frisch compensation, however, $\hat{h}$ depends on the state variable variable D only, as it can be appreciated from inspection of 43. Using Taylor's rule to approximate the policy function in the neighborhood of the steady state, one can write

$$\hat{h}(D) = h^{ss} + (D - D^{ss})\, x \tag{57}$$

where x, defined in 55, is the slope in the (D, h) space of the eigenvector computed at the steady state and associated with the negative eigenvalue of the Jacobian matrix (Dragone and Vanin, 2015). From 55 and 26 it follows that

$$\frac{\partial \hat{h}(D^{ss})}{\partial D} = x > 0. \tag{58}$$

Second, using the time-elimination method (Barro, Sala-i-Martin, 1995), the slope of an optimal trajectory in the phase diagram can be computed from the optimal dynamics of h and D,

$$\frac{\dot{h}}{\dot{D}} = \frac{\frac{dh}{dt}}{\frac{dD}{dt}} = \frac{dh}{dD}. \tag{59}$$

[13] Throughout the paper we maintain the assumption that the policy function is differentiable with respect to the parameter of interest. This assumption turns out to be satisfied as there are no jumps in the optimal path to the steady state.

Graphically, this method allows studying the slope of the vectors represented in the phase diagram. Hence, studying how this slope changes when perturbing parameter ω, i.e. $\frac{\partial}{\partial \omega}\left(\frac{dh}{dD}\right)$, provides qualitative information on how the slope of the optimal path changes when ω changes. The result will depend on which portion of the phase diagram is considered. Since we are interested in the optimal path leading to the steady state, we will restrict our attention to the portion of the phase diagram that contains the policy function $\hat{h} = \hat{h}(D)$.

Third, the policy function $\hat{h} = \hat{h}(D)$ must satisfy the following expression,

$$h^{ss} = h^0 + \int_{D_0}^{D^{ss}} \frac{\mathrm{d}\hat{h}}{\mathrm{d}D} dD, \tag{60}$$

where h^0 is the optimal medical care when $D = D_0$ and $\mathrm{d}\hat{h}(D)/\mathrm{d}D$ is the slope of the policy function for each D along the optimal path starting at D_0 and ending in D^{ss}. Denote with h^0_ω the response on impact of the optimal medical care when parameter ω unexpectedly and permanently changes, and take the derivative of 60 with respect to the generic parameter ω. Applying Leibniz's rule yields

$$h^{ss}_\omega = h^0_\omega + D^{ss}_\omega \frac{\mathrm{d}\hat{h}}{\mathrm{d}D}_{|D=D^{ss}} + \int_{D_0}^{D^{ss}} \frac{\partial}{\partial \omega}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) \mathrm{d}D. \tag{61}$$

Replacing $\mathrm{d}\hat{h}/\mathrm{d}D = x$ in the second term of 61 and rearranging yields Proposition 3.

7.5 Solution using a CES utility function

Using a CES utility function

$$U(c) = \frac{c(t)^{1-\sigma}}{1-\sigma} + b \text{ for } \sigma \neq 1, \tag{62}$$

and the survival function

$$S(D) = \frac{1+\alpha}{1+\alpha e^{\phi D}}, \tag{63}$$

the optimal agent's choices are

$$h^* = \left(-\frac{\mu\gamma A}{p}\frac{\lambda}{\eta}\right)^{\frac{1}{1-\gamma}} \tag{64}$$

$$c^* = \left(\frac{1}{\eta}\frac{1+\alpha}{1+\alpha e^{\phi D}}\right)^{\frac{1}{\sigma}}. \tag{65}$$

When $c = c^*$ and $h = h^*$, the optimal dynamics are

$$\dot{h} = \frac{h^*}{1-\gamma}\left(r - \mu - \frac{A\mu\gamma c^* (h^*)^{\gamma-1}}{p(1-\sigma)}\frac{\alpha\phi e^{\phi D}}{1+\alpha e^{\phi D}}\right) \tag{66}$$

$$\dot{c} = -\frac{c^*}{\sigma}\frac{\alpha\phi e^{\phi D}}{1+\alpha e^{\phi D}}\dot{D} \tag{67}$$

$$\dot{D} = \mu\left(D - a - A(h^*)^{\gamma}\right) \tag{68}$$

$$\dot{k} = rk + Y - ph^* - c^*. \tag{69}$$

Internal steady states satisfy the following conditions:

$$h^{ss} = \left[\frac{p(1-\sigma)}{A\mu\gamma c^{ss}}\frac{1+\alpha e^{\phi D^{ss}}}{\alpha\phi e^{\phi D^{ss}}}(\mu - r)\right]^{\frac{1}{\gamma-1}} \tag{70}$$

$$c^{ss} = \left(\frac{1}{\eta}\frac{1+\alpha}{1+\alpha e^{\phi D^{ss}}}\right)^{\frac{1}{\sigma}} \tag{71}$$

$$D^{ss} = a + A(h^{ss})^{\gamma} \tag{72}$$

$$k^{ss} = \frac{1}{q+\rho}\left(ph^{ss} + c^{ss} - Y\right). \tag{73}$$

7.6 Proof of Proposition 4

Using 26 we can compute the long run change in medical care and deficits as follows

$$\frac{\partial h^{ss}}{\partial p}\Big|_{(h^{ss},D^{ss})} = -\frac{r\mu^2\mathcal{Z}}{p^2|\mathcal{J}|}\frac{\gamma}{1-\gamma}\frac{U}{U_c}A(h^{ss})^{\gamma} > 0 \tag{74}$$

$$\frac{\partial D^{ss}}{\partial p}\Big|_{(h^{ss},D^{ss})} = \gamma A(h^{ss})^{\gamma-1}\frac{\partial h^{ss}}{\partial p} > 0. \tag{75}$$

Exploiting the fact that $x = \frac{\mu-\varepsilon}{\gamma\mu A}(h^{ss})^{1-\gamma} > 0$ yields the value of the first two terms of equation 28

$$h_p^{ss} - xD_p^{ss}\Big|_{(h^{ss},D^{ss})} = -\frac{\varepsilon\mu r\mathcal{Z}}{p^2|\mathcal{J}|}\frac{\gamma}{1-\gamma}\frac{U}{U_c}A(h^{ss})^{\gamma} < 0. \tag{76}$$

The integrand of the third term of equation 28 is

$$\frac{\partial}{\partial p}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) = \frac{\partial}{\partial p}\left(\frac{\dot{h}}{\dot{D}}\right) = \frac{\mu\mathcal{Z}}{p^2\dot{D}}\frac{\gamma}{1-\gamma}\frac{U}{U_c}Ah^{\gamma} > 0. \tag{77}$$

Since $\dot{D} > 0$ when $D_0 < D^{ss}$, the above expression is positive. Hence, using equation 28, the sign of h_p^0 is negative.

7.7 Proof of Proposition 5

Using 26 we can compute the long run change in medical care and deficits as follows

$$h_A^{ss} \lfloor_{(h^{ss},D^{ss})} = -\frac{r\mu^2\mathcal{Z}}{p^2|\mathcal{J}|}\frac{\gamma}{1-\gamma}\frac{U}{U_c}A\left(h^{ss}\right)^{\gamma} > 0 \tag{78}$$

$$D_A^{ss} \lfloor_{(h^{ss},D^{ss})} = \frac{\mu\left(r-\mu\right)r}{\left(1-\gamma\right)|\mathcal{J}|}\left(h^{ss}\right)^{\gamma} < 0. \tag{79}$$

Since $x > 0$, the following holds:

$$h_A^{ss} - xD_A^{ss} > 0. \tag{80}$$

To assess the change in the slope of the policy function compute

$$\frac{\partial}{\partial A}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) = \frac{\partial}{\partial A}\left(\frac{\dot{h}}{\dot{D}}\right) = \frac{\mu}{\left(\dot{D}\right)^2\left(1-\gamma\right)}\frac{hp\left(r-\mu\right)U_c - \left(D-a\right)\mu\gamma U\mathcal{Z}}{pU_c}h^{\gamma}, \tag{81}$$

whose sign is in general ambiguous. Defining

$$\bar{\gamma}_A = \frac{\left(r-\mu\right)hp}{\left(D-a\right)\mu\mathcal{Z}}\frac{U_c}{U} > 0 \tag{82}$$

then one can rewrite 81 as

$$\frac{\partial}{\partial A}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) = -\frac{\mu\left(r-\mu\right)}{(\dot{D})^2\left(1-\gamma\right)}\frac{\gamma-\gamma_A}{\gamma_A}h^{\gamma+1}. \tag{83}$$

If $\gamma > \bar{\gamma}_A$, then $\frac{\partial}{\partial A}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) < 0$ and the sign of h_A^0 is positive.

Alternatively, 81 can be written as

$$\frac{\partial}{\partial A}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) = \frac{\mu\left(r-\mu\right)}{(\dot{D})^2\left(1-\gamma\right)}\frac{\pi-\pi_A}{\pi}h^{\gamma+1} \tag{84}$$

where $\pi = cU_c/U > 0$ denotes the elasticity of consumption and

$$\pi_A = \frac{c\left(D-a\right)\mu\gamma\mathcal{Z}}{hp\left(r-\mu\right)} > 0 \tag{85}$$

Hence, an alternative sufficient condition for $\frac{\partial}{\partial A}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) < 0$ (and, consequently, $h_A^0 > 0$) is that the elasticity of consumption is lower than π_A.

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