CESifo WORKING PAPERS

7246
2018

September 2018

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CESifo

Impressum:

CESifo Working Papers
ISSN 2364-1428 (electronic version)
Publisher and distributor: Munich Society for the Promotion of Economic Research - CESifo GmbH
The international platform of Ludwigs-Maximilians University's Center for Economic Studies and the ifo Institute
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Editors: Clemens Fuest, Oliver Falck, Jasmin Gröschl
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CESifo *Working Paper No. 7246*
Category 13: Behavioural Economics

Negligible Senescence: An Economic Life Cycle Model for the Future

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.

JEL-Codes: D910, I120, J170.

Keywords: comparative dynamics, endogenous mortality, life-expectancy, medical progress.

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September 6, 2018
We thank Martin Forster, David Slusky, Paolo Vanin and the participants of the 2016 Symposium on Human Capital and Health Behavior in Gothenburg and the 2016 Conference of the European Health Economics Association in Hamburg for useful comments and suggestions. A previous version of this paper circulated under the title “Human health and aging over an infinite time horizon”.

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 (D) optimally increase in a quasi-exponential way and the mortality- or hazard-rate (μ) also increases in such a quasi-exponential way, akin to the Gompertz (1825) law of mortality. This predicted health behavior is sketched by solid lines in Figure 1.

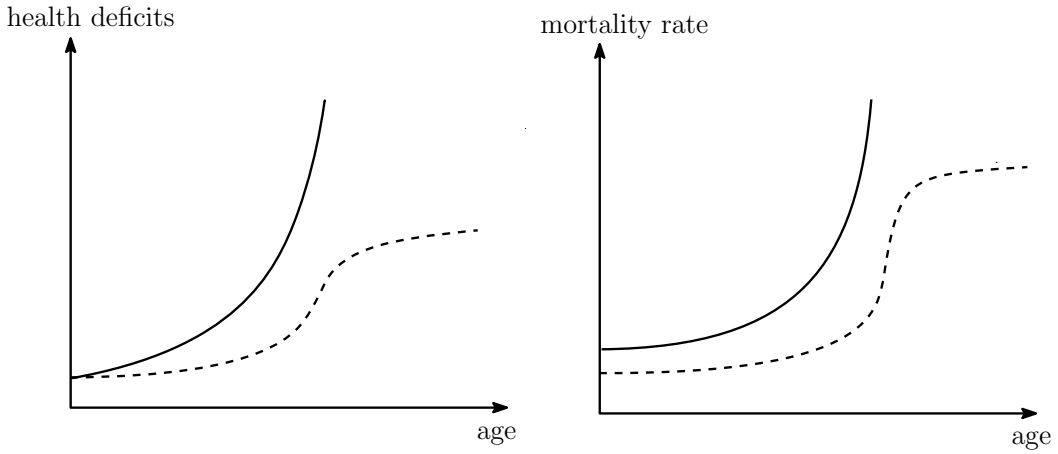

Figure 1: Stylized life cycle trajectories for health deficit accumulation (left) and mortality rate (right). Solid lines: standard model; dashed lines: existence of steady state.

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 manifold 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).

As discussed in Dalgaard and Strulik (2014), the health deficit accumulation 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 long run 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 the shock occurs, 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 set up the health deficit model with endogenous mortality. In Section 3 we provide the general formulas to perform comparative dynamics analysis in the short and in the long run. In Section 4 we consider two examples: a rise in the cost of health and an improvement in medical technology. Section 5 concludes.

2 A model of endogenous aging with uncertain lifetime

2.1 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 discussed in detail by Dalgaard and Strulik (2014), and based on research in gerontology (Gavrilov and Gavrilova, 1991, Mitnitski et al. 2002), 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 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.

The agent faces the following dynamic budget constraint,

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

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:

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

where $U\left(c\right)$ is the instantaneous utility function, ρ is the discount rate due to individual impatience, and T is the age at death.[2]

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. To account for this uncertainty, denote with

$$\mathcal{S}\left(D\left(t\right), t\right) \equiv \Pr\left(T > t\right) = \int_{t}^{\infty} g\left(D\left(\tau\right), \tau\right) d\tau \tag{5}$$

the probability that an individual will be alive at t (with $g\left(D\left(\tau\right), \tau\right)$ being the associated density function).

We assume that the survival function $\mathcal{S}$ is continuously differentiable, equal to one when $t = 0$ and in absence of health deficits, and that it is strictly decreasing to zero when time and health deficits increase $(\mathcal{S}_t, \mathcal{S}_D < 0)$.[3]

With respect to the literature, where survival functions depend on calendar time t only, here we allow the survival function $\mathcal{S}$ to depend also on biological aging, which is endogenous to individual behavior and is represented by the cumulated health deficits D. Accordingly, different combinations of biological and chronological aging determine the same survival probability $\bar{\mathcal{S}}$. The slope of such indifference curve is described, in the (D, t) space, as

$$\frac{\mathrm{d}t}{\mathrm{d}D}_{\lfloor \mathcal{S}=\bar{\mathcal{S}}} = -\frac{\mathcal{S}_D\left(\cdot\right)}{\mathcal{S}_t\left(\cdot\right)} < 0, \tag{6}$$

where $\mathcal{S}_D\left(\cdot\right)$ and $\mathcal{S}_t\left(\cdot\right)$ describe the marginal effect of biological aging and the marginal effect of the passing of time, respectively, on the survival probability. The slope of the

[2]The utility function is non negative, strictly increasing and concave. 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.

[3]Age 0 should be conceptualized as real age 20 since, by assumption, individuals are "born" as young adults. We assume that the accumulated wealth becomes an unintended bequest when the individual dies.

survival indifference curve is negative, hence an old individual with good health (i.e. few health deficits) can have the same survival probability of a younger individual with bad health (i.e. many health deficits).

For later reference, define the *endogenous hazard rate* $\mathcal{Z}$ as

$$\mathcal{Z}\left(D\left(t\right),t\right) \equiv -\frac{\mathcal{S}_D\left(\cdot\right)}{\mathcal{S}\left(\cdot\right)} > 0, \tag{7}$$

and the *exogenous hazard rate* $\mathcal{Q}$ as[4]

$$\mathcal{Q}\left(D\left(t\right),t\right) \equiv -\frac{\mathcal{S}_t\left(\cdot\right)}{\mathcal{S}\left(\cdot\right)} > 0. \tag{8}$$

2.2 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

$$\max_{c,h} \mathbb{E}_g \left[\int_0^T e^{-\rho t} U\left(c\left(t\right)\right) dt\right] \tag{9}$$

$$\dot{k}\left(t\right) = rk\left(t\right) + Y - c\left(t\right) - ph\left(t\right) \tag{10}$$

$$\dot{D}\left(t\right) = \mu\left[D\left(t\right) - a - A\left(h\left(t\right)\right)^{\gamma}\right] \tag{11}$$

$$k\left(0\right) = k_0, \qquad D\left(0\right) = D_0 > a. \tag{12}$$

The above problem differs from the literature considering a deterministic time of death in that the objective function is an expected intertemporal utility function, where the stochastic element is represented by the agent's uncertain time of death. However, as suggested by Yaari (1965), the expected intertemporal utility function 9 can be conveniently transformed into a more treatable intertemporal expected utility function which weighs the instantaneous utility function by the individual survival probability. Accordingly, the objective function of the agent from time t_0 onwards can be written as (see the Appendix for details)

$$V\left(t_0\right) = \mathbb{E}_g \left[\int_{t_0}^T e^{-\rho t} U\left(c\left(t\right)\right) \mathrm{d}t\right] = \int_{t_0}^{\infty} e^{-\rho t} \mathcal{S}\left(D\left(t\right),t\right) U\left(c\left(t\right)\right) \mathrm{d}t. \tag{13}$$

[4]Our notion of endogenous and exogenous hazard rate is inspired by the standard notion of a hazard rate, which describes the probability of dying at t, conditional on having survived until t, i.e. $\lim_{dt\to\infty} \frac{\Pr(t\leq T\leq t+dt)}{dt \Pr(T>t)}$. When $\Omega_D = 0$, our exogenous hazard rate and the standard definition of hazard rate coincide. The condition $\mathcal{Z}_D > 0$ is required to guarantee concavity of the Hamiltonian function with respect to health deficits, as shown in the Appendix.

Equation 13 represents the expected value of life of the agent. The goal of the agent is to maximize it under 10 to 12.

In the proceeding we will consider the following survival function

$$\mathcal{S}(D,t) = s(t)\, S(D) = e^{-qt} S(D). \tag{14}$$

The multiplicative specification allows to disentangle the chronological and biological aging components of the survival probability of the agent: the *exogenous* hazard rate $\mathcal{Q} = q$ does not depend on health deficits (with q summing up the role of environmental factors and individual characteristics that are out of the control of the agent). In contrast, the *endogenous* hazard rate $\mathcal{Z}(D) = -S_D(D)/S(D)$ does not depend on time.

Solving the model, the following system of differential equations results:[5]

$$\dot{h} = \frac{h}{1-\gamma}\left[r - \mu + \frac{\gamma\mu A}{p}\frac{U(c)}{U_c(c)}\mathcal{Z}(D)\, h^{\gamma-1}\right] \tag{15}$$

$$\dot{c} = -\frac{U_c(c)}{U_{cc}(c)}\left[r - \rho - q - \mathcal{Z}(D)\,\dot{D}\right] \tag{16}$$

$$\dot{D} = \mu\left(D - a - Ah^{\gamma}\right) \tag{17}$$

$$\dot{k} = rk + Y - ph - c \tag{18}$$

Equations 15 and 16 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 in 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 when $t \to \infty$, 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 unlikely. In other words, we take into account the (almost trivial) insight from gerontology

[5] All proofs are in the Appendix. Consistent with the literature, the time arguments used to denote the state variables $D(t)$ and $k(t)$ and the control variables $h(t)$ and $c(t)$ should be interpreted as time labels. In the proceeding these time labels will be omitted to simplify the notation.

that "however old we are, our probability to die within the next hour is never equal to one" (Jacquard, 1982).[6] 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 tomorrows *as if* there is a possibility that the time of death is infinitely far away (although death will surely occur in finite time). As a consequence, focusing on steady states is theoretically justified and, as shown in the subsequent sections, also very convenient because it allows to describe behavior and its responses to economic or technological shocks at different time horizons.

Since equations 15 to 18 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 require the candidate steady states to be feasible and saddle-point stable. Feasibility requires the steady state values of medical care, consumption, deficit accumulation and capital to be non negative. Saddle-point stability implies that there exists an optimal path of choices that can be sustained over an arbitrarily long time period, a requirement that results from the uncertainty of lifetime. When a steady state is a saddlepoint, it is a meaningful long run goal and it is optimal to choose a path of consumption and medical care directed toward it.

Using an approach often adopted in lifecycle models, in the proceeding we assume $q = r - \rho$.[7] The main consequence of this choice is that the Euler equation 16 for of

[6] 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 *yet* just because it is a very unlikely event.

[7] In the literature on partial equilibrium lifecyle 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. 36 in the Appendix). This allows to abstract from the dynamics originated by changes in individual wealth. Here we follow a similar approach. The extension to the general case is considered in the Appendix.

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:

$$\frac{\dot{c}}{c} = \frac{U_c}{cU_{cc}} \mathcal{Z}(D) \dot{D}. \tag{19}$$

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

Let $\bar{\gamma} \in (0,1)$ be a threshold level on the effectiveness of medical care in the repair of health deficits. Denoting steady states with the superscript 'ss', the following Proposition shows the conditions under which a an internal steady with saddle point stability exists.

Proposition 1 *Consider the endogenous aging problem 9 to 12. If $\mu < r$, internal steady state(s) satisfy:*

$$h^{ss} = \left[\frac{\gamma \mu A}{p(r-\mu)} \frac{U(c^{ss})}{U_c(c^{ss})} \mathcal{Z}(D^{ss}) \right]^{\frac{1}{1-\gamma}} \tag{20}$$

$$r = q + \rho \tag{21}$$

$$D^{ss} = a + A(h^{ss})^{\gamma} \tag{22}$$

$$k^{ss} = \frac{1}{r}(ph^{ss} + c^{ss} - Y). \tag{23}$$

The steady states are saddle point stable if the marginal return of medical care in the repair of health deficits is high enough ($\gamma > \bar{\gamma}$), and they are unstable otherwise.

Proposition 1 shows the necessary conditions under which there exist steady states where medical care and health deficits levels are positive and constant over time. Consistent with the intuition, the proposition says that aiming at a long life is rational if the efficiency of medical care is good enough.

In Figure 2 we illustrate two possible intertemporal paths to the steady state (alternatively, the same information can be displayed as a function of calendar time, as shown in the solid lines of Figures 3 and 4).[8]

When the initial level of health deficits is low, the agent should spend most income on consumption goods, and spend little on medical care. This slows down the process of deficit accumulation, although it does not reverse it. Hence deficits accumulate until,

[8]For Figure 2 and the subsequent figures we use a CES utility function $U(c) = \frac{c(t)^{1-\sigma}}{1-\sigma} + b$ and we use the logistic function $S(D) = \frac{1+\alpha}{1+\alpha e^{\phi D}}$ for biological aging. Parameters: $\mu = \frac{3}{100}$, $r = \frac{3}{50}$, $q = \frac{1}{50}$, $\rho = \frac{1}{25}$, $p = 1$, $Y = \frac{1}{3}$, $A = \frac{1}{2}$, $\sigma = \frac{19}{20}$, $\gamma = \frac{24}{25}$, $\alpha = \frac{1}{100}$, $\phi = 10$, $\eta_0 = \frac{2}{5}$, $a = 0$, $b = 0$. At the steady state, threshold $\bar{\gamma} \simeq .518$.

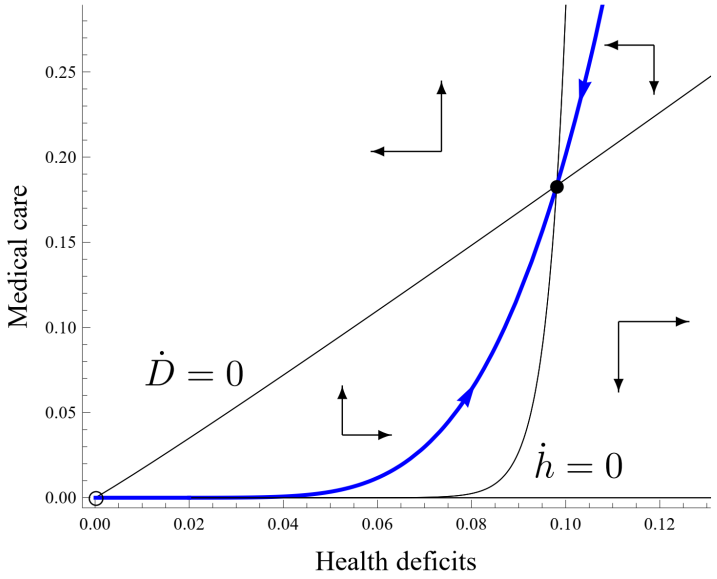

Figure 2: Phase diagram. Thick line: optimal medical care; Thin lines: nullclines $\dot{h} = \dot{D} = 0$; Full dot: long run goal (saddle point); Hollow dot: unstable steady state.

over time, the trade-off between consumption and medical care changes. 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. In this steady state, health deficits, the hazard rate and medical care levels out.

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), and with the evidence on the dynamics of hazard rates observed in supercentenarians (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; Schuenemann 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.

A second pattern depicted in Figure 2 is characterized by an agent beginning her life being very unhealthy. In such a case, her level of health deficis 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 conditions under which an internal steady state is saddlepoint stable are that (i) the force of aging is smaller than the interest rate ($\mu < r$), and (ii) that medical technology is sufficiently powerful (not too strongly decreasing returns of health investments, $\gamma > \bar{\gamma}$, see eq. 52 in the Appendix). These conditions are intuitive. The first one requires that individuals are able to accumulate savings for future health care faster than the pace at which their bodies deteriorate. The second condition requires that health care is highly effective in reducing health deficits.

This result highlights two paths to life extension, which are both discussed in medical science and gerontology: a slowdown in the force of "natural" aging μ achieved through, for example gene therapy, caloric restriction etc., or a sufficiently fast repair of health damages (sufficiently high γ) achieved through elimination of damaged cells, telomerase reactivation etc. While it can be debated whether these conditions are fulfilled already, it is likely that they will be fulfilled at some point in the future. 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.[9]

[9]The model also produces corner steady states. In the case considered in Proposition 1 ($r > \mu$), there exists a corner steady state associated with no medical care, but it is unstable and therefore not reachable under general initial conditions. If instead $r < \mu$, there exists a unique steady state in which all income is spent on consumption and nothing is spent on medical care. Despite being saddlepoint stable, it is associated to a strictly decreasing medical care path and to strictly improving health over the whole lifetime. This pattern does not match the empirical evidence and is therefore discarded in the subsequent analysis.

3 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 exercises 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 15 to 17. Let $\mathcal{J}$ denote the Jacobian matrix associated to 15 to 17 and define

$$\mathcal{J}_{h,\omega} \equiv \begin{bmatrix} \frac{\partial \dot{h}}{\partial \omega} & \frac{\partial \dot{h}}{\partial k} & \frac{\partial \dot{h}}{\partial D} \\ \frac{\partial \dot{k}}{\partial \omega} & \frac{\partial \dot{k}}{\partial k} & \frac{\partial \dot{k}}{\partial D} \\ \frac{\partial \dot{D}}{\partial \omega} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial D} \end{bmatrix}, \qquad \mathcal{J}_{D,\omega} \equiv \begin{bmatrix} \frac{\partial \dot{h}}{\partial h} & \frac{\partial \dot{h}}{\partial k} & \frac{\partial \dot{h}}{\partial \omega} \\ \frac{\partial \dot{k}}{\partial h} & \frac{\partial \dot{k}}{\partial k} & \frac{\partial \dot{k}}{\partial \omega} \\ \frac{\partial \dot{D}}{\partial h} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial \omega} \end{bmatrix}. \tag{24}$$

After a permanent shock on a general parameter ω, the steady state level of medical care and health deficits change as follows:

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

$$h^{ss}_{\omega} = -\frac{|\mathcal{J}_{h,\omega}|}{|\mathcal{J}|}, \qquad D^{ss}_{\omega} = -\frac{|\mathcal{J}_{D,\omega}|}{|\mathcal{J}|}, \tag{25}$$

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 saddlepont 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 25. 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 does it rely on numerical simulations.

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

$$h^0_\omega = h^{ss}_\omega - xD^{ss}_\omega - \int_{D_0}^{D^{ss}} \frac{\partial}{\partial \omega}\left(\frac{d\hat{h}}{dD}\right) dD. \tag{26}$$

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.

Corollary 1 (Sufficient conditions) *On impact, after an exogenous permanent shock:*

- *Medical care increases if $h^{ss}_\omega - xD^{ss}_\omega > 0$ and $\frac{d\hat{h}}{dD} < 0$ for all $D \in (D_0, D^{ss})$;*
- *Medical care decreases if $h^{ss}_\omega - xD^{ss}_\omega < 0$ and $\frac{d\hat{h}}{dD} > 0$ 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 two bits of information: (i) how the steady state responds to the shock, and (ii) how the slope of the policy function changes. The former information is obtained using Proposition 2. The latter information 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 15 and 17, and studying how the ratio changes when ω increases (see the Appendix for details).

To understand the applicability of equation 26, 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 ($h^{ss}_\omega = D^{ss}_\omega = 0$). Hence, if $\partial(\mathrm{d}\hat{h}/\mathrm{d}D)/\partial\omega$ can be shown to be, say, positive, the sign of h^0_ω is negative. Hence, on impact medical care is predicted to decrease. If, instead, also the steady state changes, the sign of the right hand side of 26 will be assessed by taking into

account both the change in the steady state values, $h^{ss}_{\omega} - xD^{ss}_{\omega}$, and the change in slope of the policy function over the range (D^0, D^{ss}), i.e. $\int_{D^0}^{D^{ss}} \frac{\partial}{\partial \omega}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right)\mathrm{d}D$. Corollary 1 describes sufficient conditions under which a non-ambiguous prediction on the impulse response can be made.

4 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. For both exercises we use the formulas presented in the previous Section and we apply them to the case of a CES utility function $U(c) = \frac{c(t)^{1-\sigma}}{1-\sigma} + b$ (where σ is the constant elasticity of marginal utility and $b \geq 0$ is a base level utility, see, e.g. Hall, Jones, 2007). For biological aging we use the logistic function $S(D) = \frac{1+\alpha}{1+\alpha e^{\phi D}}$. The parameters α and ϕ are positive.[10]

4.1 Increasing medical care costs

In the following we consider the case in which medical care (e.g. medicines) becomes more expensive. All statements will be reversed in sign in case medical care becomes cheaper.

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.*

As an illustration of the results of Proposition 4, Figure 3 depicts the effect of 10% increase in the cost of medical care (from $p = 1$ to $p = 1.1$). With respect to Figure 2, 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.

In the left panels of Figure 3 we show the time paths of optimal medical care, consumption and expected utility (measured as $S(D)U(c)$). In the right panels we plot the associated time paths of health deficits, hazard rate, and the percentage difference in the value of life over time (evaluated using equation 4 for t_0 going from zero to 100).

[10] We consider the case where an internal steady state exists and is saddlepoint stable, and we focus on trajectories where the initial level of deficits is lower than the steady state.

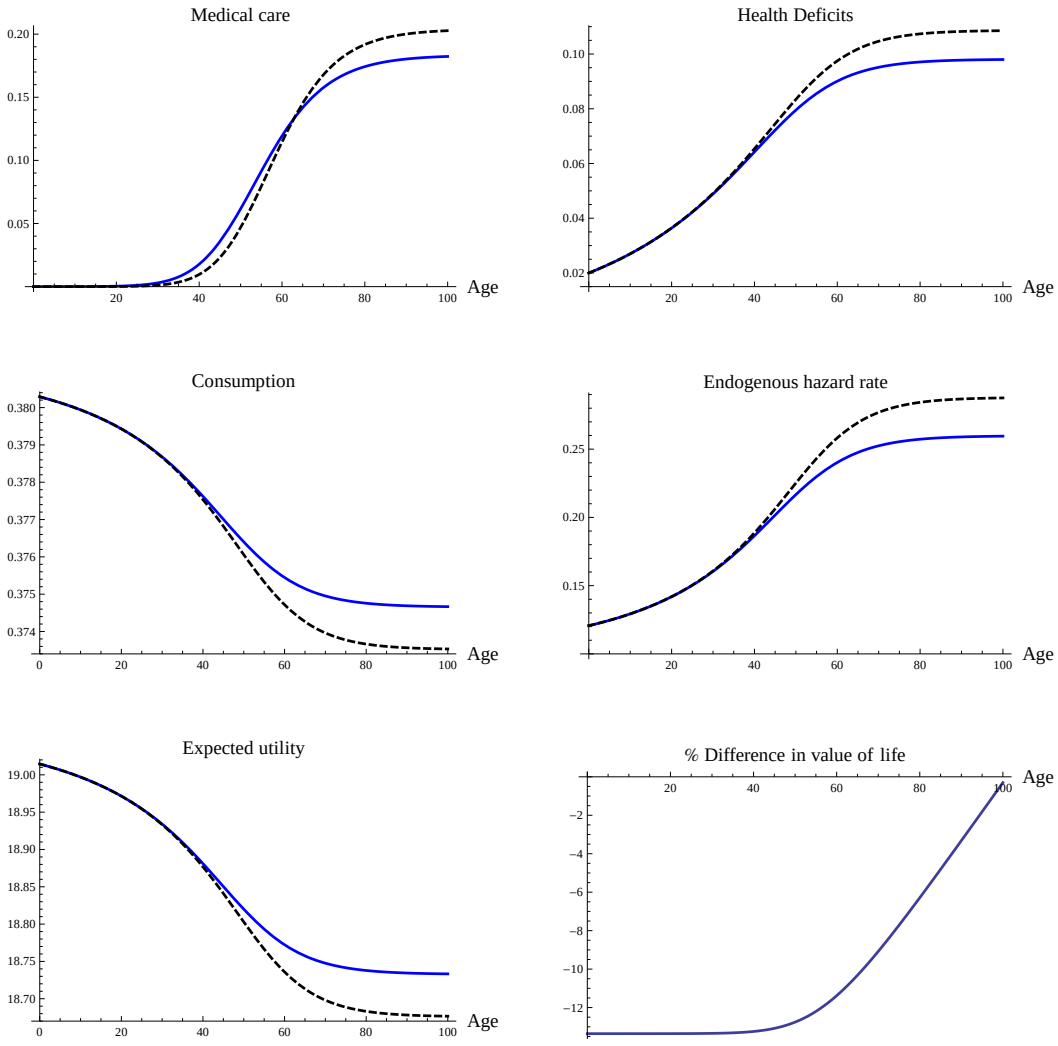

Figure 3: More expensive medical care. Solid lines: initial time trajectories; Dashed lines: new time trajectories after the price shock (from $p = 1$ to $p = 1.1$); Initial condition: $D_0 = 0.02$. Parameters as for Figure 2.

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 $S(D)U(c)$. The lower expected utility profile shown in Figure 3 is due to the joint effect of higher health deficits (which reduce the endogenous component of the survival probability) and lower consumption. Using equation 4, the bottom-right panel in Figure 3 shows the percentage difference in the value of life due to the price shock . When evaluated at time $t_0 = 0$, more expensive medical care determines a large decrease in the intertemporal expected utility profile. As time goes on (i.e. as t_0 increases), the differences in the future (expected) lifetime utility shrink. From the $t_0 = 100$ perspective, the pre and post-shock expected utility profiles are very similar.

4.2 Improvement of medical technology

We next consider 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, we focus on an increase in A.

Proposition 5 *If medical technology improves, 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 4 shows the adjustment paths corresponding to an improvement in medical technology. 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.

5 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

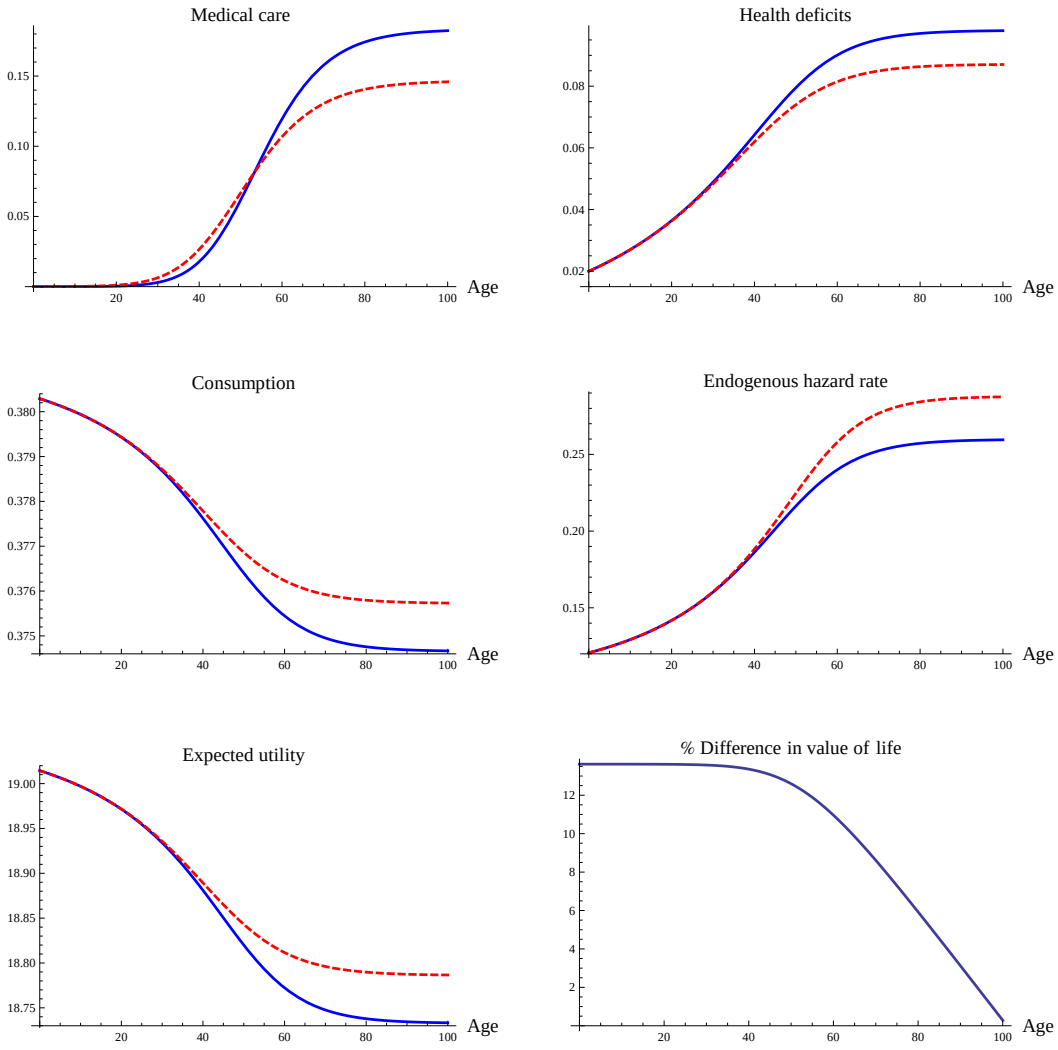

Figure 4: Better health technology. Solid lines: initial time trajectories; Dashed lines: new time trajectories after the technological improvement (from $A = 0.5$ to $A = 0.55$); Initial condition: $D_0 = 0.02$. Parameters as for Figure 2.

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 the marginal return in terms of health deficit repair does not decline too strongly with rising health care. 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.

Adjustment dynamics towards the steady state 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 at a finite T 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, 2015). 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 (Dalgaard and Strulik, 2014, 2015), a steady state of constant health exists as well, but only for a favorable constellation of parameters. So far, the health deficit literature has focused on situations where the steady state does not exist and thus, by design, it has 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.

6 Appendix

6.1 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:

$$\begin{aligned}\mathbb{E}_g\left[\int_0^T e^{-\rho t}U(c)\,\mathrm{d}t\right] &= \int_0^\infty g(D,T)\left(\int_0^T e^{-\rho t}U(c)\,\mathrm{d}t\right)\mathrm{d}T \\ &= \int_0^\infty e^{-\rho t}U(c)\left(\int_t^\infty g(D,T)\,\mathrm{d}T\right)\mathrm{d}t \\ &= \int_0^\infty e^{-\rho t}\mathcal{S}(D,t)\,U(c)\,\mathrm{d}t \end{aligned} \tag{27}$$

6.2 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:

$$H = S(D,t)\,U(c) + \lambda\dot{D} + \eta\dot{k}, \tag{28}$$

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 (subscritps denote partial derivatives, the arguments are henceforth omitted):

$$h^* \quad : \quad H_h = 0 \quad \Leftrightarrow \quad \lambda\gamma\mu A h^{\gamma-1} = -p\eta \tag{29}$$

$$c^* \quad : \quad H_c = 0 \quad \Leftrightarrow \quad SU_c = \eta, \tag{30}$$

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

$$\mathcal{S}_D^2 U_c^2 - \mathcal{S}UU_{cc}\mathcal{S}_{DD} < 0. \tag{31}$$

Rewriting the above equation using the definition of endogenous hazard rate

$$\mathcal{Z}(D(t),t) \equiv -\frac{\mathcal{S}_D(\cdot)}{\mathcal{S}(\cdot)} = -\frac{S_D}{S} > 0 \tag{32}$$

allows to rewrite the concavity requirement as

$$\oplus \equiv \left(U_c^2 - UU_{cc}\right)\mathcal{Z}^2 + UU_{cc}\mathcal{Z}_D < 0. \tag{33}$$

With $U > 0$ and $U_{cc} < 0$, the condition $\mathcal{Z}_D > 0$ is necessary to ensure concavity. For later reference, note that

$$\frac{\partial c^*}{\partial D} = -\frac{S_D}{S}\frac{U_c}{U_{cc}} = \mathcal{Z}_D\frac{U_c}{U_{cc}} \tag{34}$$

From the first order conditions 29 and 30 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 $\eta \geq 0$. The necessary conditions for the costate dynamics are

$$\dot{\lambda} = \lambda(\rho + q) - H_D = (\rho + q - r)\lambda - e^{-\rho t}\mathcal{S}_D U \tag{35}$$

$$\dot{\eta} = \eta(\rho + q) - H_k = (\rho + q - r)\eta \tag{36}$$

plus the transversality condition $\lim_{t\to\infty} H(t) = 0$. Differentiating 29 and 30 with respect to time, and using 35 and 36 yields:

$$\dot{h} = \frac{h}{1-\gamma}\left(r - \mu + \frac{\gamma\mu A}{p}\frac{U}{U_c}\frac{S_D}{S}h^{\gamma-1}\right) \tag{37}$$

$$\dot{c} = -\frac{U_c(c)}{U_{cc}(c)}\left[r - \rho + \frac{S_D}{S}\dot{D}\right] \tag{38}$$

$$\dot{D} = \mu(D - a - Ah^{\gamma}) \tag{39}$$

$$\dot{k} = rk + Y - ph - c. \tag{40}$$

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

$$\dot{h} = \frac{h}{1-\gamma}\left(r - \mu - \frac{\gamma\mu A}{p}\frac{U}{U_c}\mathcal{Z}h^{\gamma-1}\right) \tag{41}$$

$$\dot{c} = -\frac{U_c}{U_{cc}}\left(r - \rho - q - \mathcal{Z}\dot{D}\right) \tag{42}$$

$$\dot{D} = \mu(D - a - Ah^{\gamma}) \tag{43}$$

$$\dot{k} = rk + Y - ph - c, \tag{44}$$

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. 41). Note also

that, when health deficits are constant over time ($\dot{D}=0$), then equation 65 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 $r(k)=\rho+q$ (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:

$$\begin{aligned}\mathcal{J}_0 &= \begin{bmatrix} \frac{\partial \dot{D}}{\partial D} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial h} & \frac{\partial \dot{D}}{\partial c} \\ \frac{\partial \dot{k}}{\partial D} & \frac{\partial \dot{k}}{\partial k} & \frac{\partial \dot{k}}{\partial h} & \frac{\partial \dot{k}}{\partial c} \\ \frac{\partial \dot{h}}{\partial D} & \frac{\partial \dot{h}}{\partial k} & \frac{\partial \dot{h}}{\partial h} & \frac{\partial \dot{h}}{\partial c} \\ \frac{\partial \dot{c}}{\partial D} & \frac{\partial \dot{c}}{\partial k} & \frac{\partial \dot{c}}{\partial h} & \frac{\partial \dot{c}}{\partial c} \end{bmatrix} \\ &= \begin{bmatrix} \mu & 0 & -A\mu\gamma\left(h^{ss}\right)^{\gamma-1} & 0 \\ 0 & r & -p & -1 \\ -\frac{\gamma}{1-\gamma}\frac{A(h^{ss})^{\gamma}\mu}{p}\frac{U}{U_c}\mathcal{Z}_D & 0 & \frac{1}{1-\gamma}\left(r-\mu-\frac{A(h^{ss})^{\gamma-1}\mu\gamma^2}{p}\frac{U}{U_c}\mathcal{Z}\right) & \frac{A(h^{ss})^{\gamma}\mu\gamma}{p(1-\gamma)}\frac{UU_{cc}-U_c^2}{U_c^2}\mathcal{Z} \\ \mu\mathcal{Z}\frac{U_c}{U_{cc}} & 0 & \left(Ah^{\gamma-1}\mu\gamma\right)\frac{U_c}{U_{cc}}\mathcal{Z} & 0 \end{bmatrix}\end{aligned} \tag{45}$$

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 42, 43 and 44 yields:

$$\dot{D} = \mu\left(D-a-Ah^{\gamma}\right) \tag{46}$$

$$\widetilde{\dot{k}} = rk+Y-ph-C(D) \tag{47}$$

$$\widetilde{\dot{h}} = \frac{h}{1-\gamma}\left(r-\mu-\frac{\gamma\mu A}{p}\frac{U\left(C(D)\right)}{U_c\left(C(D)\right)}\mathcal{Z}h^{\gamma-1}\right). \tag{48}$$

We can therefore compute the following 3×3 Jacobian matrix $\mathcal{J}$ at the steady state

$$\begin{aligned}\mathcal{J} &= \begin{bmatrix} \frac{\partial \dot{D}}{\partial D} & \frac{\partial \dot{D}}{\partial k} & \frac{\partial \dot{D}}{\partial h} \\ \frac{\partial \widetilde{\dot{k}}}{\partial D} & \frac{\partial \widetilde{\dot{k}}}{\partial k} & \frac{\partial \widetilde{\dot{k}}}{\partial h} \\ \frac{\partial \widetilde{\dot{h}}}{\partial D} & \frac{\partial \widetilde{\dot{h}}}{\partial k} & \frac{\partial \widetilde{\dot{h}}}{\partial h} \end{bmatrix} \\ &= \begin{bmatrix} \mu & 0 & -A\gamma\mu\left(h^{ss}\right)^{\gamma-1} \\ -\frac{U_c}{U_{cc}}\mathcal{Z} & r & -p \\ \frac{\gamma}{1-\gamma}\frac{\mu(D^{ss}-a)}{pS^2U_cU_{cc}}\oplus & 0 & \frac{1}{1-\gamma}\left(r-\mu-\frac{A\mu\gamma^2}{p}\frac{U}{U_c}\mathcal{Z}\left(h^{ss}\right)^{\gamma-1}\right) \end{bmatrix}\end{aligned} \tag{49}$$

At the steady state, the associated determinant and trace are:

$$|\mathcal{J}| = \frac{\mu r}{\bar{\gamma}(1-\gamma)}(r-\mu)(\bar{\gamma}-\gamma) \tag{50}$$

$$Tr|\mathcal{J}| = 2r \tag{51}$$

Let

$$\bar{\gamma} \equiv 1 - \frac{SUS_D U_{cc}}{SUS_D U_{cc} + (D^{ss} - a) \oplus} \in (0, 1) . \tag{52}$$

where $\oplus < 0$ was defined in 33.

An internal steady state exists if $r > \mu$, and it has saddle point stability (one negative and two positive eigenvalues, hence $|\mathcal{J}| < 0$) if

$$\gamma > \bar{\gamma}. \tag{53}$$

When the saddlepoint stability condition holds, one eigenvalue associated with the 3×3 Jacobian matrix $\mathcal{J}$, 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

$$x \equiv \frac{x_1}{x_3} = \frac{\mu - \varepsilon}{\gamma \mu A} (h^{ss})^{1-\gamma} > 0. \tag{54}$$

This completes the proof of Proposition 1.

6.3 Proof of Proposition 2

The proof relies on an application of the implicit function theorem to equations 15, 16 and 17, computed at the steady state, as shown in Dragone and Vanin (2015).

6.4 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.[11] 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 41. Using Taylor's rule to approximate the policy function in the neighborhood of the steady state, one can write

$$\hat{h}(D) = h^{ss} + (D - D^{ss}) x \tag{55}$$

[11] 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.

where x, defined in 54, 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 54 and 24 it follows that

$$\frac{\partial \hat{h}\left(D^{ss}\right)}{\partial D} = x > 0. \tag{56}$$

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,

$$\frac{\dot{h}}{\dot{D}} = \frac{\frac{dh}{dt}}{\frac{dD}{dt}} = \frac{dh}{dD}. \tag{57}$$

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 $\hat{h} = \hat{h}\left(D\right)$.

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

$$h^{ss} = h^0 + \int_{D_0}^{D^{ss}} \frac{\mathrm{d}\hat{h}}{\mathrm{d}D} dD, \tag{58}$$

where h^0 is the optimal medical care when $D = D_0$ and $\mathrm{d}\hat{h}\left(D\right)/\mathrm{d}D$ 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 58 with respect to the generic parameter ω. Applying Leibniz's rule yields

$$h^{ss}_\omega = h^0_\omega + D^{ss}_\omega \frac{\mathrm{d}\hat{h}}{\mathrm{d}D}_{|D=D^{ss}} + \int_{D_0}^{D^{ss}} \frac{\partial}{\partial \omega}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) \mathrm{d}D. \tag{59}$$

Replacing $\mathrm{d}\hat{h}/\mathrm{d}D = x$ in the second term of 59 and rearranging yields Proposition 3.

6.5 Solution using a CES utility function and a logistic survival function

Using a CES utility function

$$U\left(c\right) = \frac{c\left(t\right)^{1-\sigma}}{1-\sigma} + b \text{ for } \sigma \neq 1, \tag{60}$$

and a logistic survival function

$$S(D) = \frac{1+\alpha}{1+\alpha e^{\phi D}}, \tag{61}$$

the optimal agent's choices are

$$h^* = \left(-\frac{\mu\gamma A}{p}\frac{\lambda}{\eta}\right)^{\frac{1}{1-\gamma}} \tag{62}$$

$$c^* = \left(\frac{1}{\eta}\frac{1+\alpha}{1+\alpha e^{\phi D}}\right)^{\frac{1}{\sigma}}. \tag{63}$$

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

$$\dot{h} = \frac{h^*}{1-\gamma}\left(r - \mu - \frac{A\mu\gamma c^* \left(h^*\right)^{\gamma-1}}{p\left(1-\sigma\right)}\frac{\alpha\phi e^{\phi D}}{1+\alpha e^{\phi D}}\right) \tag{64}$$

$$\dot{c} = -\frac{c^*}{\sigma}\frac{\alpha\phi e^{\phi D}}{1+\alpha e^{\phi D}}\dot{D} \tag{65}$$

$$\dot{D} = \mu\left(D - a - A\left(h^*\right)^{\gamma}\right) \tag{66}$$

$$\dot{k} = rk + M - ph^* - c^*. \tag{67}$$

Internal steady states satisfy the following conditions:

$$h^{ss} = \left[\frac{p\left(1-\sigma\right)}{A\mu\gamma c^{ss}}\frac{1+\alpha e^{\phi D^{ss}}}{\alpha\phi e^{\phi D^{ss}}}\left(\mu - r\right)\right]^{\frac{1}{\gamma-1}} \tag{68}$$

$$c^{ss} = \left(\frac{1}{\eta}\frac{1+\alpha}{1+\alpha e^{\phi D^{ss}}}\right)^{\frac{1}{\sigma}} \tag{69}$$

$$D^{ss} = a + A\left(h^{ss}\right)^{\gamma} \tag{70}$$

$$k^{ss} = \frac{1}{q+\rho}\left(ph^{ss} + c^{ss} - Y\right). \tag{71}$$

The condition for saddlepoint stability requires

$$\gamma > \bar{\gamma} = \frac{\sigma\left(1+\alpha e^{\phi D^{ss}}\right)}{\sigma\left(1+\alpha e^{\phi D^{ss}}\right) + \phi\left(D^{ss}-a\right)\left(\alpha e^{\phi D^{ss}} - \sigma\right)}. \tag{72}$$

6.6 Proof of Proposition 4

In the long run medical care and deficits change as follows

$$\frac{\partial h^{ss}}{\partial p}\lfloor_{(h^{ss},D^{ss})} = -\frac{rA\mu^2\gamma c^{ss}h^{\gamma}}{p^2\left(1-\sigma\right)\left(1-\gamma\right)\left|\mathcal{J}\right|}\mathcal{Z} > 0 \tag{73}$$

$$\frac{\partial D^{ss}}{\partial p}\lfloor_{(h^{ss},D^{ss})} = \gamma A\left(h^{ss}\right)^{\gamma-1}\frac{\partial h^{ss}}{\partial p} > 0. \tag{74}$$

Using the above expressions and considering the first two terms of equation 26 yields

$$h_p^{ss} - xD_p^{ss} \lfloor_{(h^{ss},D^{ss})} = -Ah^{\gamma}\mu p^2 r(1-\gamma)\gamma U U_c \mathcal{Z}|\mathcal{J}|\varepsilon < 0. \tag{75}$$

The integrand of the third term of equation 26 is

$$\frac{\partial}{\partial p}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) = Ah^{\gamma}p^2\left(1-\gamma\right)\gamma U U_c \mathcal{Z}\dot{D} > 0. \tag{76}$$

Since $\dot{D} > 0$ and $\varepsilon < 0$, the above expression is positive. Hence, using equation 26, the sign of h_p^0 is negative.

6.7 Proof of Proposition 5

In the long run, medical care and deficits change as follows

$$h_A^{ss} \lfloor_{(h^{ss},D^{ss})} = \frac{\mu\left(r-\mu\right)rh^{ss}}{A|\mathcal{J}|\left(1-\gamma\right)}\left(\frac{A\left(h^{ss}\right)^{\gamma}\oplus}{SS_D U U_{cc}} - 1\right) < 0. \tag{77}$$

$$D_A^{ss} \lfloor_{(h^{ss},D^{ss})} = \frac{\mu\left(r-\mu\right)r}{\left(1-\gamma\right)|\mathcal{J}|}\left(h^{ss}\right)^{\gamma} < 0. \tag{78}$$

Moreover:

$$h_A^{ss} - xD_A^{ss} = \frac{h}{\gamma A}\left(\frac{\varepsilon}{|\mathcal{J}|}\frac{\left(r-\mu\right)r}{1-\gamma} - 1\right).$$

Let

$$\bar{\gamma}_A \equiv 1 - \frac{\varepsilon}{|\mathcal{J}|}\left(r-\mu\right)r < 1. \tag{79}$$

Then, in the long run, the following holds:

$$h_A^{ss} - xD_A^{ss} > 0 \quad \Longleftrightarrow \quad \gamma > \bar{\gamma}_A. \tag{80}$$

To assess the value of the integrand in equation 26, assume that $\gamma > \bar{\gamma}_A$ and compute

$$\frac{\partial}{\partial A}\left(\frac{\mathrm{d}\hat{h}}{\mathrm{d}D}\right) = -\frac{h\left(r-\mu\right)}{\left(1-\gamma\right)A\dot{D}} < 0. \tag{81}$$

Since $\dot{D} > 0$, the above expression is negative. Hence, using equation 26, condition $\gamma > \bar{\gamma}_A$ is sufficient for the sign of h_A^0 to be positive.

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