



Long-Run Economic Perspectives of an Ageing Society

Aging and Frailty: What Can Be Learned From the Natural Sciences

Carl-Johan Dalgaard

Holger Strulik

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Aging and Frailty: What Can Be Learned From the Natural Sciences

Report on Exportable Tools, Methods, and Concepts

Car-Johan Dalgaard and Holger Strulik*

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Abstract. This report outlines central results from WP 2 of the LEPAS Project. It reviews our conclusions from our recent survey of the natural science literature on the modeling of aging, and provides some concrete examples on how these insights can be integrated into economic theory. Specifically, we show how the modeling of health investment and health deterioration in human capital theory can be improved by incorporating insights from reliability theory and the frailty index, and we demonstrate how the modeling of longevity in overlapping generations models can benefit from insights from the literature on metabolic activity and life-span. The background survey, which comes in four parts, is available at <http://www.lepas-fp7.de/>.

Keywords: Aging, Mortality, Life-span, Frailty, Reliability Theory, Metabolism, Body size, Overlapping Generations.

*University of Copenhagen, Oester Farimagsgade 5, bygning 26,1353 Koebenhavn K, Carl.Johan.Dalgaard@econ.ku.dk and University of Hannover, Wirtschaftswissenschaftliche Fakultät, Königsworther Platz 1, 30167 Hannover, Germany; email: strulik@vwl.uni-hannover.de. This research was funded by the European Commission within the project “Long-Run Economic Perspectives of an Aging Society” (LEPAS) in the Seventh Framework Programme under the Socio-economic Sciences and Humanities theme (Grant Agreement: SSH7-CT-2009-217275).

1. AGING, THE FRAILTY INDEX, AND THE MODELING OF HEALTH DETERIORATION

Health, a stock variable measuring the state of the body, and death, a unique event, are connected by a process that we call “aging”. Aging is defined as the intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death (Arking, 2006, Masoro, 2006). This definition suggests to measure human aging and health by accounting for the bodily deficits that we accumulate over our life.

The emphasis on functionality is also central to recent theories in gerontology, which try to explain human aging by employing basic insights and mechanisms from reliability theory, a sub-discipline of the engineering science (Barlow and Proschan, 1975). Reliability theory describes the human organism as a complex, redundant system. The basic idea is that bodily functions are provided by essential blocks (which could be thought of as organs, tissue etc), connected in series, which consist in turn of a large number of non-aging elements, connected in parallel. A particular bodily function is lost when a critically number of elements are lost. Non-aging means that elements display a constant, age-independent failure rate. It implies that with increasing age blocks lose redundancy and the probability to die increases. Aging is explained as a loss of redundancy over time. It can be shown that already relatively simple systems of parallel-serial connectivity can explain basic patterns of human aging (Gavrilov and Gavrilova, 1991). If the system is complex enough, including, for example, also a mechanism for redundancy expansion in early life it is capable to redraw with great precision actually observed human aging patterns (Milne, 2008). This notion of aging as accelerated loss of organ reserve is in line with the mainstream view in the medical science. For example, initially, as a young adult, the functional capacity of human organs is estimated to be tenfold higher than needed for survival. (Fries, 1980). A more detailed introduction to reliability theory can be found in Strulik (2010).

In order to measure human functionality the medical science and gerontology has proposed several indices of human capability or disability. One measure, the so called frailty index, is particularly related to reliability theory. The frailty index, developed by Mitnitski and Rockwood and several coauthors, counts for a large sample of (in their case Canadian) individuals the bodily impairments which are actually present out of a long list of potential impairments, ranging from mild deficits (reduced vision, incontinence) to near lethal ones (stroke, cancer). The authors then show that the relative number of deficits, i.e. the frailty index D , of an individual correlates exponentially with age t in the following way

$$D(t) = b + B \exp(\mu t). \tag{1}$$

The parameters of the frailty accumulation law are estimated with great precision. The point estimate and 95 percent confidence interval for Canadian men are $b = 0.02 \pm 0.001$, $\log(B) = -5.77 \pm 0.06$, and $\mu = 0.043 \pm 0.001$ with an R^2 of 0.97 (Mitnitski et al., 2002a, 2002b).

The exponential nature of frailty accumulation resembles the Gompertz–Makeham law of mortality for which several researchers have observed stability of the exponent, i.e. a strikingly constant pace of aging (Gavrilov and Gavrilova, 1991, Bongarts, 2005, Carnes and Olshansky, 2007, Gurven and Kaplan, 2007). This indicates that human ingenuity has not yet provided Wonka-Vite (Dahl, 1987, Kirkwood, 1999), i.e. a treatment that effectively slows down the aging process as such. The shift parameters, however, have been shown to decrease over time, indicating that the aging process has been - and probably will be - influenced through technological progress, improving income and wealth, and behavioral change.

Given the strong statistical association between frailty accumulation and Gompertz law we can hypothesize that man-made progress in medical technology and, more generally, in maintaining and repairing bodily impairments manifested itself mostly in the shift parameters b and B whereas the pace of frailty accumulation is relatively stable over time. A stable μ is consistent with the notion of a “compression of morbidity” (Fries, 1980), i.e. the observation that medical progress has allowed us to live longer and to live long in good health life but was so far unsuccessful in improving the strong health deterioration that old people experience in the last years before they expire. In line with the observation by Phillipson and Becker(1998) the successful fight against one bodily impairment entails inescapably the occurrence of another impairment in the near future.

Ideas from reliability research and the frailty index can be usefully exploited to model and calibrate the evolution of human health and health investment. In order to see the value added it is helpful to briefly reconsider standard modeling of endogenous health in economics. Normally, health is introduced as a state variable similar to human capital evolving like $\dot{H} = I - \delta H$, where a dot signifies a derivative with respect to age and I is health investment, see e.g. Ehrlich and Chuma (1990). Keeping the rate of health depreciation δ constant, the model predicts, counterfactually, that health loss is largest when the state of health is best (δH is largest when

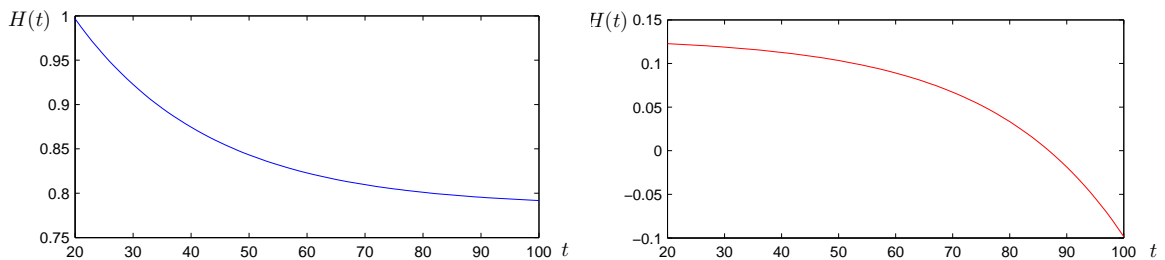
H is highest). Of course, health researchers are aware of this contradiction of the facts, and repair the health equation by introducing an age-dependent depreciation rate $\delta(t)$. But how exactly should the depreciation function be specified so that the model provides an acceptable approximation of real health deterioration? Here the notion of aging as the accumulated loss of bodily function suggests a “natural” biological foundation of health depreciation, which is intuitive and readily implemented.

Suppose actual health is defined as best attainable health minus accumulated frailties, $H = \bar{H} - D$ where \bar{H} could be the state of health of a healthy 15 year old. This implies $\dot{H} = -\dot{D}$. Taking the derivative in (1) with respect to age and ignoring sex-specific indices we get

$$\dot{D} = \beta B \exp(\mu t) = \mu D - \mu B. \quad (2)$$

Inserting this into \dot{H} and re-substituting D provides $\dot{H} = \beta (\bar{H} - H - B)$, i.e. a simple linear differential equation for health, which – in line with the facts – predicts that health loss is small at a good state of health and increasing losses are predicted when the health stock deteriorates. Moreover the health deterioration is readily quantified is, at least for Canadians, with help of the available frailty data. Figure 1 compares the implications for health deterioration diagrammatically. The left hand side re-iterates the time path based on standard human capital theory (the mainstream approach), the right hand side shows the time path based on empirical accumulation (the innovative take away).

FIGURE 1: HEALTH DETERIORATION: STANDARD MODELING IN HUMAN CAPITAL THEORY (LEFT) VS. FOUNDATION FROM GERONTOLOGY AND THE FRAILTY INDEX (RIGHT)



In order to integrate the impact of health investment on longevity one could proceed with borrowing from Ehrlich and Chuma (1990) the idea of a minimum level of health essential for survival. In the present notation, an individual expires at age T when bodily deficits exceed a threshold \bar{D} , $D(T) \geq \bar{D}$. Ideally, we would like to model stochastic deficit accumulation so that

the date of expiry is uncertain. Because this would most likely complicate the model beyond analytical tractability, a reasonable first approximation could ignore risk aversion and proceed – as Ehrlich and Chuma and the related literature – by considering a deterministic health process.

Deficit accumulation according to (2) implies that an individual’s fate is sealed at birth since initial deficits $D(0)$ predetermine the date of expiry T . The model becomes more realistic when we introduce health expenditure, i.e. costly investment into maintenance and repair of bodily function. Since medical and demographic research has shown that technology has not yet managed to manipulate the pace of aging, the natural gateway for health expenditure, h , is through the shift parameter B . This suggests the following parsimonious refinement of the process of deficit accumulation:

$$\dot{D} = \mu D - a - Ah^\gamma. \quad (3)$$

Here a constitutes a parameter fixed by nature and the parameters $A > 0$ and $0 < \gamma < 1$ reflect the state of the health technology. While A refers to the general power of health expenditure in maintenance and repair of the the human body, the parameter γ specifies the degree of decreasing returns of health expenditure. The larger γ the larger the relative productivity of cost-intensive high-technology medicine in maintaining and repairing frail human bodies. Note that, in line with the empirical evidence, the available health technology modifies “only” the constant of the equation of motion. Future research may make available the Wonka-Vite (perhaps through epi-genetic treatment) which has the power to reduce μ , the speed at which we deteriorate.

2. METABOLIM AND LONGEVITY

The links between metabolism and longevity, discussed in Dalgaard (2010), could potentially be integrated into economic models. In the following we sketch one potential application, which would allow for the analysis of how savings, longevity and body stature interacts in an intergenerational setting.

We begin by asserting that maximum life span, x_t , is affected by body size in the following way (with the “microfoundations” laid out in Dalgaard, 2010):

$$x_t = \bar{x} m_t^{1-b}, \bar{x} > 0,$$

where m_t is the body mass of a member of generation t , and b in keeping with Kleiber's law would be $3/4$. "Maximum life span" does not mean actual life span. But it is reasonable to assume that the *survival probability* is increasing in x_t .

Suppose now, that life is segmented into three periods. Period one is period of body growth; individuals live off the consumption of their young parent. No economic choices are made. In period 2, the first economically active period in life, individuals work, save and consume. Consumption is shared with the (sole) offspring; for simplicity we ignore population growth, and replacement fertility is exogenous. The size of each generation is normalized to one. In period three individuals live off their savings. Not everybody makes it to retirement however; there is a survival probability $\pi_t < 1$; π_t is an increasing function of maximum life-span, and thus x_t .¹

The problem of a young adult at time t could be stated

$$\log(c_{yt}) + \pi \left[\bar{x} m_t^{1-b} \right] \log(c_{ot+1})$$

subject to

$$c_{yt} + s_t = w_t$$

$$c_{ot+1} = (1 + r_{t+1}) s_t.$$

Accordingly, we are assuming π is the survival probability, which is increasing in body size for the reasons explained in Dalgaard (2010). The solution to the problem is optimal consumption and savings²

$$s_t = \frac{\pi_t}{1 + \pi_t} w_t$$

$$c_{yt} = \frac{w_t}{1 + \pi_t}.$$

Body size of (young) individuals evolves in the way described in Dalgaard and Strulik (2010), and Dalgaard (2010)

$$m_{t+1} = ac_{1t} + (1 - d) m_t.$$

¹Certain reasonable assumptions could be introduced on π . For instance, non-decreasing, and bounded; $\lim_{m \rightarrow \infty} \pi(m) = \bar{\pi} < 1$. Details such as this would have to be worked out in a final application.

²The model is related to Chakraborty (2004) who also examines endogenous life time. In Chakraborty, however, life time depends on government supply of public goods (and body size is ignored).

In an overlapping generations model, where mortality is endogenous, there is a problem of "unused savings" to deal with. In the present context we may use the same solution as in Chakraborty, assuming the existence of a (non-profit) life insurance company, which pays out uncollected savings as additional return for the survivors. This does not influence the discussion to follow, due to the logarithmic preferences of households. That is, in this case the size of real rate of return leaves the savings rate unaffected.

Hence, at time t , body mass of the young is determined by the consumption of their parents when *they* were young, as consumption is (for simplicity) shared between adult and offspring. During period t the young consume and share consumption with their offspring, which determines the offsprings body size as of time $t + 1$; that is, m_{t+1} . Accordingly, when the young adults chose consumption, their body size is predetermined. Capital (per worker) in period $t + 1$ is given by the savings of the young. We end up with the following two-dimensional dynamical system for the economy

$$k_{t+1} = (1 - \alpha) \frac{\pi_t}{1 + \pi_t} k_t^\alpha m_t^\phi$$

$$m_{t+1} = a (1 - \alpha) \frac{k_t^\alpha m_t^\phi}{1 + \pi_t} + (1 - d) m_t$$

where we have assumed competitive markets, and that the aggregate per worker production technology is $y_t = m_t^\phi k_t^\alpha$, where $\phi \geq 0$ parameterizes the productivity impact from large size (see Dalgaard and Strulik, 2009 on this), and, recall $\pi_t = \pi [\bar{x} m_t^{1-b}]$.

The model determines endogenously long-run income, body size and longevity and may feature multiple steady states, with one steady state characterized by low body size, short lives, low savings and income, and a “high steady state”, characterized by high body size, long lives, high savings and income. But depending on parameters, the model may also allow for monotonic convergence; during transition body size, capital per worker, income per worker and longevity rises.³

This model could form the basis of an improved understanding of how physiological characteristics, like body size and longevity, influences the growth process, and could generate new predictions about the sources of comparative development and cross-country variation in savings per capita.

³Naturally, if growth is persistently ongoing (exogenous technological change, say), the model would need to be adjusted, in that body size otherwise would tend to infinity. Realistically, a model such as this would possibly have to involve a two sector set-up (agriculture and industry), where preferences are such that Engel’s law prevail, implying that food consumption takes up a progressively smaller part of the budget as income rises. This could prevent unbounded body size. This challenge would have to be dealt with appropriately in a final application.

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