

Frailty, Mortality, and the Demand for Medical Care

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Abstract. This paper proposes a theory of aging and longevity according to which the probability to survive at any age depends on the number of health deficits accumulated, as suggested by modern biology. I calibrate the model for a reference U.S. citizen and exploit the fact that the association between health deficits and mortality has been estimated with great precision in gerontology. This allows me to use the model for quantitative experiments. I compare health expenditure, health deficits, and the value of life for richer and poorer individuals. The model motivates a strong socioeconomic gradient of health. It explains how income growth and advances in medical technology and the induced increase in health care demand led to large reductions in health deficits and roughly a doubling of the value of life at all ages.

Keywords: Aging, Longevity, Health, Savings, Value of Life.

JEL: D91, J17, J26, I12.

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1. INTRODUCTION

The life course of adult humans can be understood as a process of accumulation of health deficits. In gerontology, aging is defined as the intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death (Arking, 2006). A micro-foundation of human aging is provided by reliability theory (Gavrilov and Gavrilova, 1991), which explains it as consequence of the depletion of redundancy in the human body. 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). Increasing depletion of redundancy in human tissues and cells leads to an increasing failure rate of organs and is associated with increasing probability to die. Gerontology suggests one particularly straightforward metric to measure health deficits, the so called frailty index, i.e. the share of potential health deficits that a person has. Empirically, there exists a power law association of the mortality rate with the frailty index, the parameters of which have been estimated with great precision (Mitnitski et al., 2002a).

While the association of mortality and health deficits appears to be deterministic at the population level, death at the individual level is an inherently stochastic event. The probability to die is thus merely a measure to describe how members of a population (a species, a population of country) age *on average*. It is not useful to assess or explain individual survival prospects. In particular, there is no such thing as a “biological clock”. Instead, biologists and gerontologists argue that individual aging and death must be conceptualized as an event-dependent, not as a time-dependent, process (Arking, 2006).

The present paper provides a theory of longevity and health care demand that acknowledges these gerontological facts. It is based on the theory of health deficit accumulation (Dalgaard and Strulik, 2014), which integrated the frailty index into health economics. In contrast to the present study, however, Dalgaard and Strulik (2014) assumed that death is a certain event, occurring when sufficiently many health deficits have been accumulated and the human body is too frail to support life any longer. This simplification allowed them to derive many expressions analytically, a fact that has led to a rigorous understanding of the biological and behavioral mechanisms at work when the human body ages. However, in light of the gerontological facts, it could be argued that the theory is incomplete. Since death is an inherently stochastic event, the

involved uncertainty may affect human behavior. The present paper takes these considerations into account by modeling individual survival probability as a function of accumulated health deficits. The predicted association between the frailty index and mortality accords with the estimated relationship in gerontology.

The present paper suggests a new view on the association between stochastic mortality, health, and health demand. The literature so far was either built upon the idea of health capital accumulation (Grossman, 1972, 2000; Ehrlich and Chuma, 1990) or conceptualized mortality as a time-dependent event unrelated to the state of health. Studies in the health-capital tradition with a fixed time of death are provided by Dardanoni and Wagstaff (1987), Selden (1993), and Chang (1996) in a two-period framework and by Picone et al. (1998) and Laporte and Ferguson (2007) in a multi-period framework. Clearly, an approach based on pre-determined death cannot be used to explore the interaction between health investment, frailty, and mortality, which is in the focus of the present paper. Cropper (1977) proposed a model of stochastic “mild illnesses” in which the evolution of health capital and death remained deterministic and Liljas (1998) proposed a model in which health capital accumulation and thus death is partly stochastic.

The concept of health capital, however, entails a couple of problems. The idea of health capital accumulation, inspired from the economics of physical and human capital accumulation, is alien to the natural sciences. Consequently, health capital is a latent variable, which is hard to measure. A popular approach in empirical applications exploits the fact that health deficits are observable and tries to estimate health capital from a vector of (absent) health deficits (see Muurinen, 1982; Wagstaff, 1986). More importantly, at the core of the model is an equation of motion for health capital postulating that depreciation of health capital is an increasing function of its stock. This means that of two individuals of the same age, the one in better health, i.e. the one with the greater health stock loses more health capital in the next instant. As explained below, the hypothesis that healthier persons age faster is not supported by gerontology. Gerontological research suggests exactly the opposite: unhealthy persons age faster. The accumulation of health deficits is a positive function of the number of health deficits already present in a person (Mitnitski et al. 2002a, 2002b, 2005, 2006). Methodologically, the self-equilibrating force of health capital accumulation creates a number of problems. Unremedied, the model predicts convergence towards a saddle-point stable steady state of constant health (eternal life) and, if this solution is ignored, it suggests that optimal health investment should

decline with age, i.e. with declining health (see Strulik, 2015, and, for a rigorous analysis and critique, Dalgaard and Strulik, 2014b).

Another strand of literature avoids these counterfactual implications of the health capital model by conceptualizing mortality as a direct function of age, independently of the state of health. In some cases demographic general equilibrium aspects rather than individual health are the focus of analysis and mortality is for simplicity assumed to be completely exogenous at the individual level (e.g. Heijdra and Romp, 2008, 2009; d’Albis et al., 2012; Bloom et al., 2014). Clearly this approach is not useful to discuss the interaction of health demand, aging, and longevity. In other cases it is assumed that an age-dependent mortality function can be shifted by deliberate health investments (Ehrlich, 2000; Ehrlich and Yin, 2006; Kuhn et al., 2015). While this approach is analytically convenient it is conceptually problematic since the impact of the state of health on mortality is not explicitly modeled. Ehrlich and Yin (2005), for example, assume a linear association of health investment and the age-dependent mortality rate (and a quadratic health cost function) and acknowledge that the real functional relationship is unknown. This concession is understandable because gerontologists emphasize that there should be no role for age or (calendar-) time in a models of human aging: “Only if we can substitute the operation of the actual physiological mechanism for time we have a firm idea of what we are talking about.” (Arking, 2006, p. 10).

The present paper tries to improve on this state of affairs by assuming that the probability of death is not determined by age but by the health status of individuals, as manifested in their accumulated health deficits. This approach has the advantages that health deficits are observable, that gerontology has established a metric to measure these deficits (the frailty index), that the functional form of health deficit accumulation has been estimated with great precision, and that the predicted association between health deficits and mortality can be confronted with the data. This means that there are no degrees of freedom in the numerical calibration of the model, which makes the theory potentially a useful tool for out-of-sample predictions and theoretical experiments on the interaction of health demand, aging, and mortality.

The paper is organized as follows. The next section sets up the model and discusses how the introduction of probabilistic mortality modifies the results on optimal aging from Dalgaard and Strulik (2014). Section 3 calibrates the model with US data and explains the applied solution method. Section 4 utilizes the model in order to assess how improving income and

medical technology are predicted to affect the life cycle trajectories of medical expenditure, health deficits (as a marker of biological age), and the value of life. Section 5 concludes.

2. THE MODEL

2.1. The Optimization Problem. Consider an adult maximizing utility from consumption $c(t)$ over his or her life. The initial age is for convenience normalized to zero. Let $\rho \geq 0$ denote the rate of pure time preference and $S(t)$ the probability to survive beyond age t . Facing uncertain death, rational individuals calculate the expected utility from life-time consumption by multiplying the instantaneous utility experienced at age t with the probability to survive beyond age t (Kamien and Schwartz, 1980, Section 9, Part I). The present value of expected utility experienced over the life cycle is thus given by

$$\int_0^T S(t)e^{-\rho t}u(c(t)) dt, \tag{1}$$

with instantaneous utility $u(c) = (c^{1-\sigma} - 1)/(1 - \sigma)$ for $\sigma \neq 1$ and $u(c) = \log(c)$ for $\sigma = 1$. The upper bound T can be conceptualized as human life span, i.e. the length of life of the longest living individual.

For simplicity we assume that individuals receive a fixed income stream w throughout life. Since all decisions depend on discounted permanent income, results would not change by introducing a variable wage-for-age curve or a fixed retirement age (see Strulik, 2011, for the extension of a deterministic health-deficit models towards education and life cycle wages, see Dalgaard and Strulik, 2012, for an extension towards endogenous retirement decisions). Income can be spent on consumption goods c or on health care h . The relative price of health care is p . Besides spending income on final goods, the individual may invest in capital k and receive a net interest rate r . The individual takes all prices as given and – in order to keep the problem tractable – there exists no other uncertainty except the time of death. The law of motion for individual wealth is thus given by (2).

$$\dot{k}(t) = w + (r + m)k(t) - c(t) - ph(t). \tag{2}$$

Here we have assumed perfect annuities such that the interest rate is the sum of the rate of return on capital plus the instantaneous mortality rate $m = -\dot{S}/S$. Given the annuity market, individuals inherit no wealth and leave no bequests. Capital left over at death is distributed

among the survivors by the annuity supplier. We thus implicitly assume that the individual is surrounded by sufficiently many other individuals of the same age.

The gerontological literature suggests an affine linear equation of motion for the change of health deficits as a function of the number of health deficits that are already present in a person, denoted by $D(t)$ at age t . As in detail derived in Dalgaard and Strulik (2014), health care investments work in order to reduce the accumulation of health deficits such that, formally,

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

Here μ denotes the force of aging (the rate of “natural” bodily decay), a controls for environmental influences, A specifies general efficiency of the medical technology, $h(t)$ is health expenditure at age t , and $\gamma < 1$ is the health elasticity of health spending. As shown below, this function describes the actual accumulation of health deficits reasonably well. Here we keep the deterministic law of health deficit accumulation as it is. The innovation comes through the stochastic conceptualization of death.

In contrast to Dalgaard and Strulik (2014), individuals are hit randomly by death and the probability is a positive function of the health deficits already present in a person. Specifically, we assume that the probability to survive beyond age t , $S(t)$, is a simple negative function of the number of accumulated deficits (the frailty index number) at age t :

$$S(t) = \phi \left[1 - e^{\frac{D(t) - \bar{D}}{\alpha}} \right]. \quad (4)$$

Consequently, mortality does not directly depend on *chronological* age but only indirectly through the accumulated health deficits $D(t)$. Health deficits can be conceptualized as a biomarker for an individual’s *biological* age. The parameter α controls how rectangular the survival curve is. With decreasing α survival becomes more and more rectangular and for $\alpha \rightarrow 0$ the model converges to the model of Dalgaard and Strulik, in which all individuals die at the moment when \bar{D} health deficits have been accumulated. Here, most individuals die earlier, with less than the upper limit of possible health deficits. This means, intuitively, that the severity of deficits is stochastic. Most individuals accumulate only mild health deficits (poor vision) early in life before they acquire severe deficits (heart disease, cancer) at a later age. But some individuals get the severe deficits first and die prematurely. Human life span T is reached when \bar{D} health deficits are accumulated and death is certain ($S(T) = 0$). While the minimum state of health at

death is predetermined, human life span is endogenous. Higher income or technological progress allows all individuals, including the longest living one, to delay health deficit accumulation such that life span increases (Bongaarts, 2005; Strulik and Vollmer, 2013). The parameter ϕ controls the probability to die without any health deficit (for example by traffic accident), $0 < \phi \leq 1$. As shown below, the survival function (4) predicts an association of mortality and frailty, which fits the empirical observation reasonably well.

The life cycle problem of individuals is to maximize (1) subject to (2)–(4), the initial conditions $D(0) = D_0$, $k(0) = 0$, and the terminal conditions $D(T) = \bar{D}$ and $k(T) = 0$. That is, at the maximum life-span individuals spend the last dollar before they expire without leaving any wealth to the life insurer (supplier of annuities). The problem can be solved by employing optimal control theory; the state variables are $k(t)$ and $D(t)$ and the control variables are consumption $c(t)$ and health investments $h(t)$. The individual takes the impact of health deficits on survival into account but, in line with the literature, we assume that the annuity is a price uncontrollable by the individual like all other prices.

2.2. Optimal Aging. The associated Hamiltonian is

$$H = S e^{-\rho t} \frac{c^{1-\sigma} - 1}{1-\sigma} + \lambda_k [w + (r+m)k - c - ph] + \lambda_D \mu [D - a - Ah^\gamma].$$

The first order conditions are

$$\frac{\partial H}{\partial c} = S e^{-\rho t} c^{-\sigma} - \lambda_k = 0 \tag{5}$$

$$\frac{\partial H}{\partial h} = -\lambda_k p - \lambda_D \mu \gamma A h^{\gamma-1} = 0 \tag{6}$$

$$\frac{\partial H}{\partial k} = \lambda_k (r+m) = -\dot{\lambda}_k \tag{7}$$

$$\frac{\partial H}{\partial D} = \frac{\partial S}{\partial D} e^{-\rho t} \frac{c^{1-\sigma} - 1}{1-\sigma} + \lambda_D \mu = -\dot{\lambda}_D. \tag{8}$$

The first order condition for health care (6) has a straightforward intuitive interpretation. Written as $p\lambda_k = -\lambda_D \mu \gamma A h^{\gamma-1}$, it shows that the marginal cost of health expenditure equals the marginal benefit of health expenditure. The left hand side provides the marginal cost in terms of forgone discounted marginal utility from consumption since $\lambda_k = S e^{-\rho t} c^{-\sigma}$, according to (5). Notice that λ_k , i.e. the contribution of an additional unit capital \dot{k} to the Hamiltonian is positive while λ_D , i.e. the contribution of an additional unit of health deficits \dot{D} to the Hamiltonian is

negative. Intuitively, the shadow price λ_D translates the effect of an additional health deficit into a monetary value (in terms of the value of life). The right hand side of the equation thus provides the marginal benefit from health expenditure. It consists of the marginal return of health expenditure in terms of reduced health deficits, given by $\mu\gamma Ah^{\gamma-1}$, times the benefit in monetary terms that a unit reduction of health deficits provides, given by $-\lambda_D$.

From log-differentiating (5) with respect to age and (7) we obtain the Euler equation for consumption:

$$g_c \equiv \frac{\dot{c}}{c} = \frac{r - \rho}{\sigma}. \quad (9)$$

As in the conventional literature, the presence of perfect annuities implies that the Euler equation with uncertain death is the same as under certainty. The reason is that $m = -\dot{S}/S$, implying that the interest rate and the effective discount rate rise by the same rate ($r + m$ and $\rho + m$) and thus the death rate cancels out.

From log-differentiating (6) with respect to age we obtain

$$\frac{\dot{\lambda}_k}{\lambda_k} = \frac{\dot{\lambda}_D}{\lambda_D} + (\gamma - 1) \frac{\dot{h}}{h}. \quad (10)$$

And from inserting (5) and (6) into (8) we get

$$\frac{\dot{\lambda}_D}{\lambda_D} = - \left(\mu - \mu \frac{A\gamma h^{\gamma-1}}{p} \cdot \frac{c - c^\sigma}{1 - \sigma} \cdot \frac{\partial S}{\partial D} \cdot \frac{1}{S} \right). \quad (11)$$

Inserting (7) and (11) into (10) we arrive at the ‘‘Health Euler’’ equation:

$$g_h \equiv \frac{\dot{h}}{h} = \frac{1}{1 - \gamma} \left(\frac{\dot{\lambda}_D}{\lambda_D} - \frac{\dot{\lambda}_k}{\lambda_k} \right) = \frac{1}{1 - \gamma} \left\{ r - \mu + m + \mu \frac{A}{p} \gamma h^{\gamma-1} \frac{c^\sigma (c^{1-\sigma} - 1)}{1 - \sigma} \frac{e^{\frac{D-\bar{D}}{\alpha}}}{\alpha \left[1 - e^{\frac{D-\bar{D}}{\alpha}} \right]} \right\}. \quad (12)$$

For certain survival up to \bar{D} health deficits we have $m = 0$ and $\partial S/\partial D = 0$ and the model collapses to the deterministic model. In a deterministic world (rectangular survival as in Dalgaard and Strulik, 2014), the shadow price of capital declines at the interest rate, $\dot{\lambda}_k/\lambda_k = -r$ and the negative shadow price of health deficits declines (in absolute terms) at the rate of deficit accumulation, $\dot{\lambda}_D/\lambda_D = -\mu$, implying that health expenditure grows at rate $g_h = (r - \mu)/(1 - \gamma)$. Intuitively, if health deficits accumulate at the rate of interest, individuals prefer a constant health expenditure profile. If the interest rate exceeds the rate of deficit accumulation, individuals prefer to save for health expenditure later in life. Additionally g_h is influenced by γ ,

the curvature of the health investment function. A smaller value of γ reduces growth in health expenditures. Intuitively, if γ is small, diminishing returns set in rapidly, which makes it optimal to smooth health expenditure to make the deficit-reducing effect as large as possible.

With stochastic survival two more elements enter the g_h equation. First, the interest rate on annuities equals the return on capital plus the mortality rate, the third term in (12). Secondly, the shadow price of health deficits declines at a rate higher than μ . This can be seen from (11), in which the second term on the right hand side captures the effect of increasing deficits on the survival rate. Notice that $\partial S/\partial D$ is negative: additional deficits reduce survival. With declining survival an additional health deficit becomes, *ceteris paribus*, more costly in monetary terms, which increases the rate $\dot{\lambda}_D/\lambda_D$ in absolute terms. This effect reappears as the last term on the right hand side of (12). A more rapid decline of the shadow price of health deficits reduces the rate at which individuals would like to increase their health expenditure. In simple words, the deficit-induced probability of death motivates individuals to spend more on health now, in order to increase their survival, such that the health expenditure profile gets flatter. Quantitatively, however, it turns out that for young and middle-aged individuals the positive effects of mortality on the interest rate (the third term) is more important than the survival effect (the last term) such that g_h is larger than for the deterministic model.

At terminal time T we have $D(T) = \bar{D}$ and the associated Hamiltonian assumes the value of zero indicating that – taken the costs into account – it is not worth to live any longer. Inserting the costate variables from (5) and (6) into the Hamiltonian and noting that $S(\bar{D}) = 0$ shows that the condition that $H = 0$ is automatically fulfilled at the terminal state. In contrast to the deterministic model, terminal time T captures no longer life expectancy but maximum human life span. Almost all individuals expire before age T .

3. SOLUTION METHOD AND CALIBRATION

Identification of the optimal life cycle trajectories is somewhat harder than for the deterministic model of health deficit accumulation. The reason is the complex expression for optimal health expenditure (12), which prevents a closed-form solution for the time paths for $h(t)$ and $D(t)$. I thus approximate the solution in the following way. I begin with obtaining the optimal T under certainty. This value serves as a first estimate of optimal life-span. I then start at the terminal state and integrate the four dimensional system (2), (3), (9), and (12) backwards using

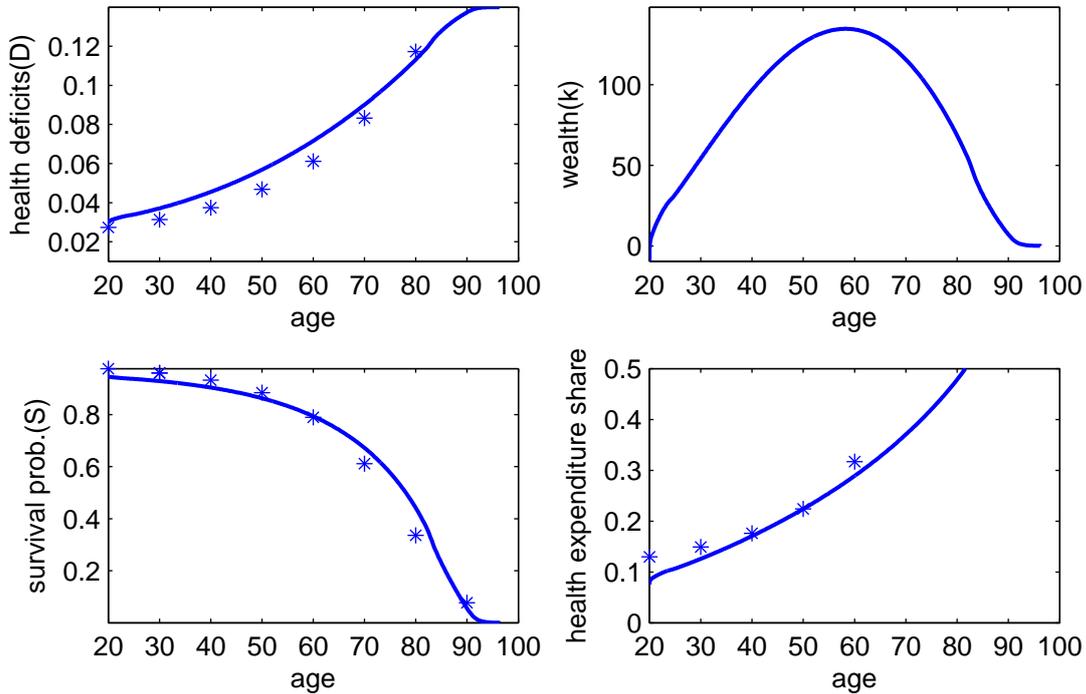
the method of Brunner and Strulik (2002). Since the trajectories representing the optimal solution of the stochastic problem originated from the endpoint of the deterministic solution they do not arrive after T years at the given initial conditions $D(0)$ and $k(0)$ in the first try. I thus iteratively adjust the estimated terminal time T until the backward trajectories approximately arrive at $D(0)$ and $k(0)$. In a final step I revert time to get the actual solution of the problem.

For the calibration I try to keep as many parameters as possible from the calibration of the associated deterministic model (Dalgaard and Strulik, 2014). This means that we consider an initially 20 years old white male US American in the benchmark run (the Reference American). We take GDP per worker in the U.S. in the year 2000 (PPP\$ 77,003) and assume a capital share of $1/3$, which implies an annual labor income (in international dollars) of \$ 51,335. The relative price of health in the year 2000 is normalized to unity, and the “physiological-medical” parameters, a , A , and μ are also taken from the deterministic model. This means that the Reference American develops 4.3 percent more health deficits from one birthday to the next. When medical technology is unable to improve adult life expectancy, the model generates a life expectancy of 42 (death at 62). This value accords with the life expectancy of 20 year old U.S. American in the year 1900 (NCHS, 1980).

In order to improve the fit of the survival curve for the year 2000, I mildly adjust σ to 1.1 (which was 1.0 in the deterministic model) and r to 0.059 (which was 0.06). Keeping the estimate of $g_h = 2.1$ percent from Dalgaard and Strulik, this implies a mild adjustment of the return on health to 0.24 (which was 0.19). Most importantly, the value for terminal health deficits \bar{D} needs adjustment because it no longer signifies the number of health deficits with which the average US American dies but the health deficits that the longest living individual in the population accumulates. In other words, the former \bar{D} value of 0.1 from the calibration of the deterministic model is now the frailty index at which 20 year old individuals *expect* to die. The new upper bound \bar{D} is higher. I estimate \bar{D} together with the parameters α and ϕ from the survivorship function $S(t)$ such that the implied survival rates approximate the real distribution of survival in the US. This approach utilizes the notion that what appears from the individual viewpoint as the probability to survive up to age t is, from the aggregate viewpoint, the population share of 20 year old males surviving to age t . I calculate this value from the Gompertz Makeham estimate for US males in Strulik and Vollmer (2013). This provides the estimates $\bar{D} = 0.14$, $\alpha = 0.05$, and $\phi = 1.065$.

For an interpretation it is important to note that the model does not deliver survivorship as a function of age (as other economic models of probabilistic survival). Instead survival is an endogenous function of the health state of the individual, i.e. the number of the accumulated health deficits. As explained in the Introduction, this notion is in line with the modern biological theory of aging (see e.g. Arking, 2006, Gavrilov and Gavrilova, 1991): while the process of dying is inherently stochastic, the expected time of death of an individual is not determined by his *age* but by his *health status*, that is by the number of accumulated health deficits.

FIGURE 1: OPTIMAL AGING AND DEATH: LIFE TRAJECTORIES



Parameters: $a = 0.013$, $A = 0.0014$, $\gamma = 0.24$, $\mu = 0.043$, $\rho = 0.06$, $r = 0.059$, $w = 51335$, $\sigma = 1.1$, $D_0 = 0.0274$, $\bar{D} = 0.14$, $p = 1$, $\alpha = 0.05$, $\phi = 1.065$. Stars: data. See main text for details.

Figure 1 shows the implied life trajectories for the Reference American. Stars in the health deficit panel indicate the original estimates of Mitnitski et al. (2002a). The model mildly overestimates the health deficits present in middle-age but explains the overall trend of health deficit accumulation quite well. The survival probability is shown in the left bottom panel. Stars indicate the estimate of the “true” survival function by Strulik and Vollmer (2013). We see that the survival curve is still quite far away from being rectangular and that the calibrated model approximates the actual survival rate by age in the US quite well.

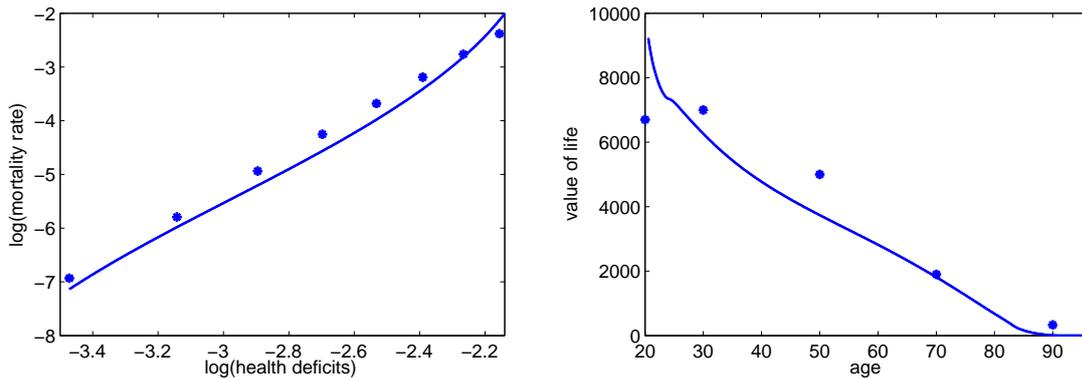
The lower right panel shows the predicted expenditure share of health care. The data for health expenditure by age is taken from Meara et al. (2004) and the data for total expenditure is taken from BLS (2002), both are from the year 2000. I converted the household expenditure data from the BLS into age-specific consumption per adult by following Deaton (1997) and computing equivalence scales. Specifically I assigned household members under 18 a weight of 0.5 of adult consumption (The BLS data does not differentiate between children of different ages in the household). The implied expenditure share is indicated by stars. The model gets the overall increase of health expenditure with age about right. In particular, the health expenditure share of the elderly is better approximated than by the deterministic model of Dalgaard and Strulik (2014). The reason is that, given the annuity market, individuals surviving to old age have the funds to spend a lot on health. Notice, however, that the solution method starts close to but not exactly at T . It misses a few oldest old individuals for which health expenditure may actually decline again (see the discussion of equation 12).

An interesting observation is the flattening wealth curve at high ages (shown in the upper right panel of Figure 1). The reason is that most people are dead at age 90 and above. This allows the annuity supplier to pay a very high annuity premium to the survivors. High interest rates in turn mean that long-living individuals can finance their health operations with relatively little wealth. Similar as in Dalgaard and Strulik (2014), the average male US citizen at age 20 expects to expire at age 74 with a frailty index of 0.1. Actually, however, many people pass away earlier and many live substantially longer. Thanks to the early expiry of their contemporaries the long-living ones have the funds to finance health repair, a fact, which allows them to live even longer. Comparing costs and benefits, however, asymptotically everyone dies before the frailty index reaches $\bar{D} = 0.14$, which happens, according to the calibration at an age of 102 years. It should not be surprising that wealth peaks at a higher value and is less quickly depleted, compared to the deterministic model. The trajectory in Figure 1 shows the wealth of *survivors*, i.e. $k(70)$ is the wealth of individuals who reached the age of 70; *average* wealth of the cohort of persons born 70 years ago is, of course, lower since the denominator includes the already deceased persons.

4. NUMERICAL EXPERIMENTS

4.1. Frailty and the Value of Life. The frailty index can be understood as a compound measure of health, morbidity, and mortality. The association between health deficit accumulation and the mortality rate has been estimated with great precision. For Canadian men, Mitnitski et al. (2002a) estimate $\log(m) = 5.08 + 3.46 \log(D)$, with an R^2 of 0.99. The implied association is shown by stars in the left-hand side panel of Figure 2. The solid line shows the prediction from the benchmark calibration of the model. It approximates the relationship estimated from the data reasonably well. The strong association between frailty and mortality is no coincidence. It has a micro-foundation at the level of cells and organs. Using elements from reliability theory, Gavrilov and Gavrilova (1991) provide a biological explanation for the observed power law (log-log) association between accumulated health deficits and the mortality rate.

FIGURE 2: FRAILITY AND THE VALUE OF LIFE



Benchmark model from Figure 1. Stars: data. See main text for details. Value of life in 1000 dollars.

We next use the model to predict the value of life (VOL) for our Reference American at different ages and compare results with estimates and predictions from the literature. The VOL provides a monetary expression of aggregate utility experienced during life until its end. Period utility is converted by the unit value of an “util”, $u'(c)$ such that the VOL at age t is defined as $V(t) = \int_t^\infty S(v)e^{-\rho v}u(c(v)) dv/u'(c(t))$. Recall that the survival probability $S(v)$ is a function of age only through the accumulated health deficits, $S(D(v))$. This means that $S(D(v))$ may be also viewed, more generally, as a quality of life indicator, which discounts utility more strongly when more health deficits are present in a person. The model thus, indirectly, controls for the impact of health deficits on experienced utility at any age.

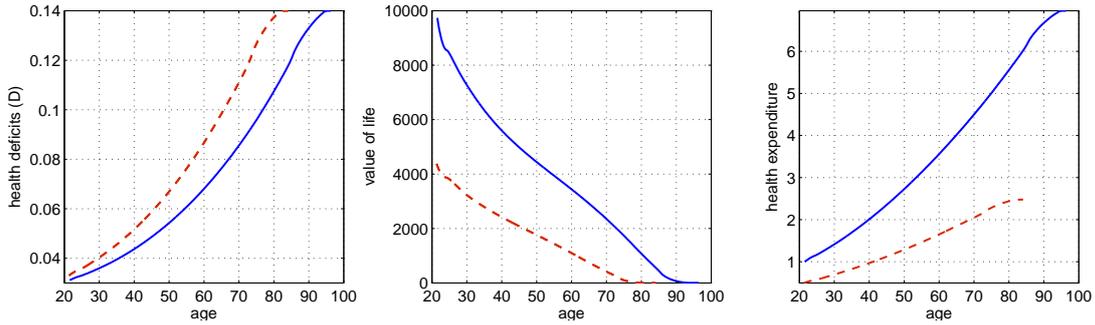
Results are shown by the solid line in the panel on the right-hand side of Figure 2. Compared to the VOL obtained for the associated deterministic model (Dalgaard and Strulik, 2014) the predictions are more plausible for the elderly. In Dalgaard and Strulik (2014), the VOL sharply declined in a concave way when age approached its terminal value. The results for the stochastic model clarify that this phenomenon was merely an artifact of deterministic lifetime. In the stochastic model the VOL smoothly fades out as individuals get older. For the young and middle-aged, however, the VOL predictions are similar, starting at 9 million dollars for 20 years old individuals and declining to about 2 million for the 65 years old. Stars in the figure show the estimates from Murphy and Topel (2006, Fig. 3). The health deficit model fails to predict an increase of the VOL from age 20 to 30 because of the flat consumption profile.¹ Otherwise, the model coincides surprisingly well with Murphy and Topel, in absolute size of the initial VOL as well as with respect to the slope at which the VOL declines with age. The coincidence is remarkable because health in Murphy and Topel's (2006) model is exogenous and its evolution is imputed from data whereas here health is endogenously explained.

4.2. Health Expenditure, Health Technology, and the Value of Life. The model can be used to re-assess the explanation of past trends of health expenditure and gains in the value of life and compare results with Murphy and Topel (2006). The first experiment is to solve the model when labor income is half of the benchmark value. On the one hand, results would reflect choices and outcomes for an individual half as rich as the Reference American. On the other hand, results reflect the isolated impact of past income growth on health expenditure and the value of life. A doubling of labor income would, for example, be reached when productivity grows at a rate of 1.5 percent for 46 years. In the numerical experiments of Murphy and Topel (2006) income roughly doubles from the period 1950-60 to 1990-2000.

Results are shown in Figure 3. Solid lines reiterate the benchmark outcome and dashed lines show results when income is cut by half. Individuals respond to the income cut with spending less on consumption and health. The panel on the right hand side shows total health expenditure, measured relative to expenditure of a rich 20 years old individual. The model predicts that health expenditure for 75 years old individuals is about 5 times the health expenditure of 35 years old individuals when income is at benchmark level. These predictions are a bit lower

¹In Murphy and Topel (2006) the remaining net life-cycle wealth (human wealth - total expenditure) enters the VOL, implying that the there initially increasing labor income causes the VOL to rise initially.

FIGURE 3: INCOME, FRAILTY, AND THE VALUE OF LIFE



Solid lines: benchmark model from Figure 1. Dashed lines: life trajectories when income is cut by half, *ceteris paribus*.

than the estimates of Meara et al. (2004) for the year 2000. At the lower income level, health expenditure is lower at all ages but in particular for the elderly. As shown by dashed lines, poor 75 years old individuals spend about the same on health care as rich 45 years old individuals.

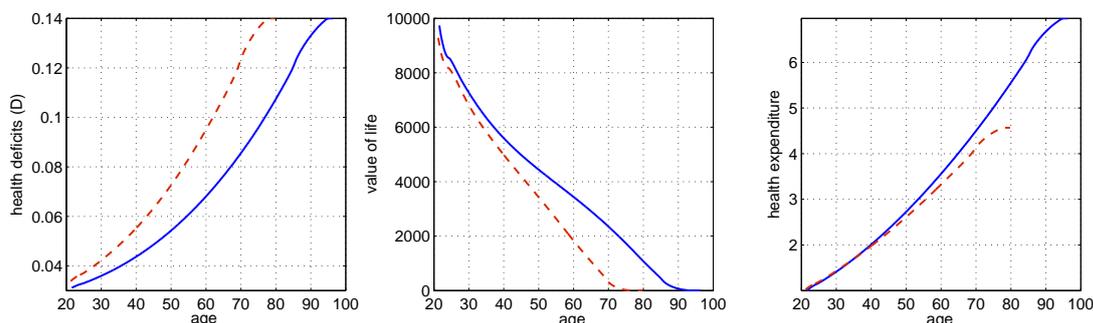
Aside from its cross-sectional interpretation, the numerical experiment can also be interpreted over time. This view shows that the rise of relative expenditure for the elderly can be partially explained as optimal response to the doubling of income over the last half century. The explanation for this outcome differs from the one offered by Hall and Jones (2007) who proposed to consider health as a luxury good. Here, the result originates from the evolution of health deficits. Since health deficits increase exponentially with age (without health expenditure), it becomes increasingly harder for aging individuals to fight against frailty accumulation with health expenditure. When income is low it is thus optimal to live a shorter life and enjoy more consumption now and in the proximate future. When income increases, in turn, relatively costly health maintenance in old age becomes affordable and, given the concavity of the instantaneous utility function, individuals optimally forgo present consumption in favor of a longer life.

The panel on the left-hand side of Figure 3 shows that the increase in income and health spending substantially improved the state of health and thus also the survival probability at any age. For example, the 70 years old at the higher income level are about as healthy as the 60 years old at the lower income level. In other words, individuals of the same age are biologically younger by about a decade through income improvements. Health expenditure thus does not only improve life expectancy but also healthy life expectancy. In its cross-section interpretation the figure is consistent with the observable large income gradient of the frailty index within countries (Harttgen et al., 2013).

The center panel of Figure 3 shows the implied gain in the value of life. Apparently, the young gain most in terms of VOL. The value of life for the 20 years old is predicted to be slightly more than twice as high when income doubles. Again, the result is surprisingly close to the estimate of Murphy and Topel (2006, Figure 4a), where the VOL for the 20 years old rises by slightly less than factor 2 from 1950 to 2000 (when income roughly doubles). Costa and Kahn (2004), using a completely different methodology based on risk premia, arrive at a much higher estimate, suggesting that the value of life increased by more than factor 3 already from 1950 to 1980 (Table 6, log specification).

In the next experiment we investigate the isolated impact of medical technology. Dashed lines in Figure 5 show results when technology A is $3/4$ times the technology level of the benchmark scenario, holding everything else constant. Solid lines re-iterate the benchmark case. Going from dashed to solid lines thus captures an increase of technology by $1/3$, an increase which would be achieved if technology grew at a rate of 0.58 percent for half a century (which coincides approximately with the estimate in Dalgaard and Strulik, 2012, for the period 1940 to 1990).

FIGURE 4: HEALTH TECHNOLOGY, FRAILTY, AND THE VALUE OF LIFE



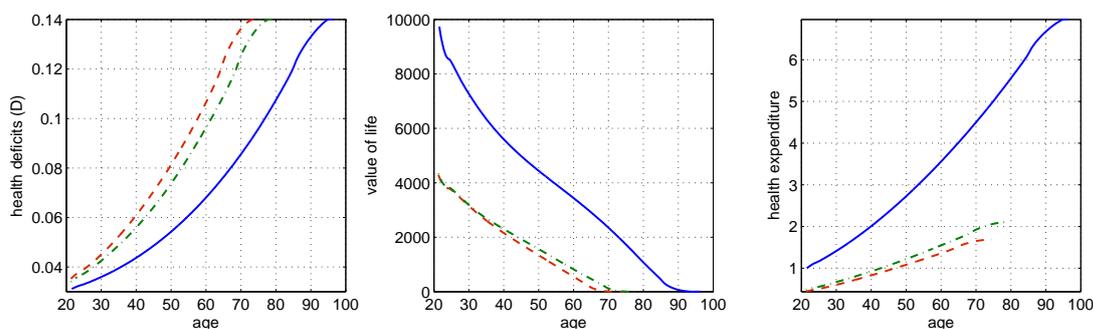
Solid lines: benchmark model from Figure 1. Dashed lines: life trajectories when medical technology (A) is reduced by factor $3/4$, ceteris paribus.

As visible in the center panel of Figure 4, medical progress – in sharp contrast to income improvements – provides large increases in the VOL predominantly for the elderly. The reason is that medical progress makes health expenditure more powerful mostly for those individuals who actually survive to an old age. Given pure time preference and uncertain survival these future gains are heavily discounted by young individuals. Elderly survivors, however, experience a large gain in the value of life. The VOL for 75 years old individuals under the high medical technology is about as high as the VOL for 60 years old individuals under the low medical technology. As shown in the panel on the left hand side, 75 years old individuals under the

high medical technology develop as much health deficits as 60 years old individuals under the low medical technology technology. Better technologies for health maintenance and repair work like a de facto reduction of the biological age of individuals at any age and in particular for the elderly.

Finally we inspect the combined effect of income growth and technology growth. Solid lines in Figure 5 re-iterate the benchmark case and dashed lines show the life trajectories when income is half as large and technology is lower by factor $3/4$. As visible in the Figure, 70 years old individuals in the high income-high technology scenario are predicted to display about as many health deficits as 53 years old individuals in the low income-low technology scenario and 50 years old individuals as much as 35 years old individuals. Income and technology growth are thus capable to explain not only large improvements in life expectancy but also in healthy life expectancy (Manton et al., 2006; Strulik and Werner, 2013). Improving health and survival conditions lead to an almost parallel outward shift of the VOL-curve at all ages. Under the high-income-high technology scenario the value of life is higher by about factor 2.3 for 20 years old individuals as well as for 50 years old individuals. These conclusions are largely robust even if one factors in an increasing relative price of health care. Dash-dotted lines in Figure 5 show the life trajectories when not only income and technology are lower than in the benchmark case but also p is only half as large.

FIGURE 5: INCOME, PRICES, TECHNOLOGY, AND THE VALUE OF LIFE



Solid lines: benchmark model from Figure 1. Dashed lines: life trajectories when income is cut by half and medical technology is lower by factor $3/4$. Dashed-dotted lines: as for dashed lines plus medical prices are cut by half.

5. CONCLUSION

This study has integrated recent insights from modern gerontology into a stochastic economic life cycle model in which the survival probability at any age depends on the accumulated health

deficits of a person. Health deficits, in turn, can be manipulated through deliberate investments in health maintenance and repair. The model allows to distinguish between chronological and biological age of persons. Health deficits are measured by the frailty index and provide an indicator of health, morbidity, and mortality condensed in one number. The model has been calibrated for a reference American using precise estimates of frailty accumulation and its association with mortality from the gerontological literature. The model predicts that income growth and medical advances of the last half of the 20th century have reduced the biological age of middle-aged persons by about 15 years and more than doubled their value of life. The estimated improvements in the value of life are largely in line with the predictions of Murphy and Topel (2006). In contrast to that study, however, the path of health care expenditure is not exogenously imposed but explained as an outcome of intertemporal choice of income-constrained individuals interested in a long and healthy life.

The model generalizes results from the associated deterministic model (Dalgaard and Strulik, 2014). The stochastic model motivates a steeper increase of health expenditure with age and the calibrated model thus leads to a better fit of the actual health expenditure pattern over the life cycle. Improved approximation of reality, however, comes at the price of analytical tractability. In contrast to the deterministic version, the model can no longer be solved in closed form but has to be simulated numerically.

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