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Optimal Aging and Death*

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Abstract. This study introduces physiological aging into a simple model of optimal intertemporal consumption. In this endeavor we draw on the natural science literature on aging. According to the purposed theory, the speed of the aging process and the time of death are endogenously determined by optimal health investments. At the same time, physiological aspects of the aging process influence optimal savings and health investment. We calibrate the model for the average US male in 2000 and proceed to show that the calibrated model accounts well for the cross-country link between labor productivity and life expectancy in the same year ("the Preston curve"); cross-country income differences can explain differences in life expectancy at age 20 of up to a decade. Moreover, technological change in health care of about 1.1% per year can account for the observed shift in the Preston curve between 1980 and 2000.

Keywords: Aging, Longevity, Health Investments, Savings, Preston Curve.
JEL: D91, J17, J26, I12.

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1. Introduction

The world population is growing older at an unprecedented rate. Indeed, in a few years time there will be more people above the age of 65 than below the age of 5, likely for the first time in human history (Kinsella and He, 2009). Population aging is sure to become the dominant demographic trend during the first decades of the 21st century. In order to assess how this demographic shift will affect the economy, it is necessary to gain an understanding of how *aging* influences the behavior of individuals. The aim of the present study is to offer some progress in this regard.

It is perhaps useful to begin by clarifying our notion of "optimal aging and death". To be sure, few people would regard aging and death as particularly "optimal". But from a biological standpoint aging and death are inevitable consequences of living; we have no say in matter. What we can do, though, is to influence the *speed* of aging, by making appropriate investments. Investments that slow down aging will also serve to influence our "date of expiry" (though not make us live forever). This provides ample motivation for costly health investments. At the same time, however, we value other expenditures that are not particularly life prolonging. So how should our lifetime income be divided between the two forms of expenditure? If lifetime resources are chosen such that utility is maximized, subject to economic *and* relevant *physiological* constraints, we will think of the aging process (and time of death) as "optimal".

The proposed theory is centered around a novel approach to the modeling of aging. Based on research on aging from the natural sciences, we conceptualize the process of aging as one whereby the human organism gradually looses redundancy and thus becomes more *fragile*. This leads us to a law of motion for human frailty, which depends on physiological parameters and health investments. The process of increasing frailty is relentless and accelerating over time, but it may be slowed by health investments. The incentive to slow down aging is a longer life, which facilitates greater consumption and thus utility. In a setting where individuals are maximizing lifetime utility from consumption we examine optimal intertemporal choice with respect to savings and health investments. Subject to the physiological constraints faced by humans, and the standard budget constraint, this allows us to characterize the optimal speed of aging and longevity. The aging process (and time of death) is thus endogenously determined

by optimal health investments. But physiological parameters related to the speed of aging also affect optimal choice in regards to saving and (health) investment.[1]

An interesting issue is how health expenditures should optimally change across the life cycle. If it is optimal for older citizen's to spend more on health than younger citizens, then what is the optimal growth rate of spending across life? The answer is of interest to the current debate on how health expenditures are likely to evolve in the decades to come, as the population is growing older (e.g., Hall and Jones, 2007; Fonseca, Michaud, Galama and Kapteyn, 2009). Unfortunately, cohort data on health expenditure are not yet available. But average (total) spending per capita by age group may give some indication of life cycle patterns.[2] Figure 1 illustrates such data for four "Western Offshoots": Australia, Canada, New Zealand and United States. The immediately visually arresting theme is that the four trajectories appear to be more or less parallel, suggesting similar per capita spending growth. Indeed, if one calculates the slope of the trend, the average annual growth rates across the life cycle are nearly identical: 2.0% (Australia), 2.1% (Canada), 1.9% (New Zealand) and 2.0 % (USA). Hence, while the four countries differ in terms of health care institutions the rise in health spending per capita across life is nevertheless strikingly similar.[3]

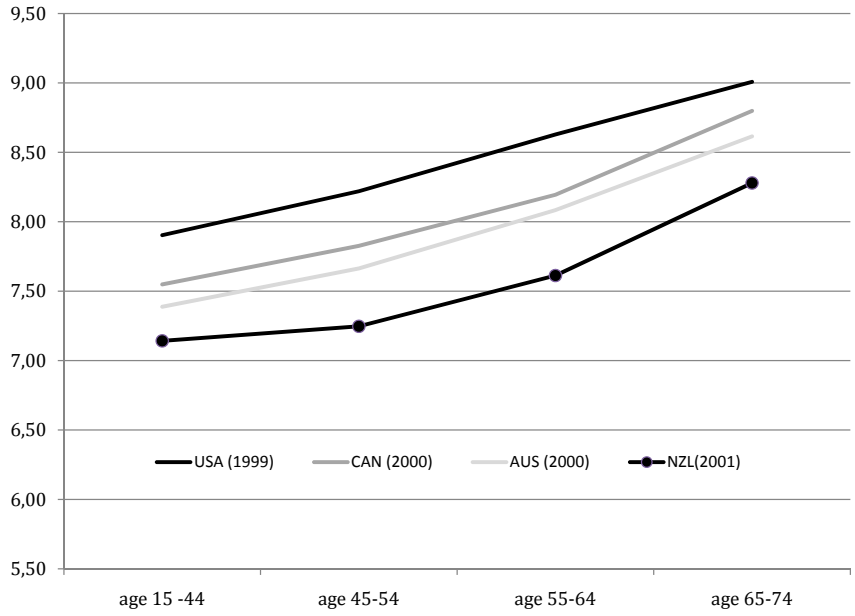
Our model delivers an optimal health spending rule for the life cycle: A "Health Euler equation". The Health Euler predicts that growth in health spending across life is determined by three factors: The real rate of interest (higher rate means faster growth in spending), health care technology (more diminishing returns to investments provides incentive for investment smoothing) and finally, the human physiology. The physiological parameter in question reflects the speed of aging. In populations where the speed of aging is "fast", late-in life health investments are relatively ineffective in prolonging life. Consequently, the optimal policy will involve slow growth in health expenditure. In theory, optimal spending could even involve *declining* health

[1]Hence, in our model health matters for the time horizon, but does not enter the per period utility function. Nor do we assume that health influences productivity. Both assumptions simplify the analysis and limit our degree of freedom when comparing the models' predictions with the data. To be sure, abandoning both assumptions would add flexibility to the model, making it far easier to map it into US data on longevity etc. At the same time it would be in conflict with the "Stigler-Becker dictum" of not appealing to preferences when explaining phenomena of interest (Stigler and Becker, 1977, p. 76.).

[2]This is also the assumption made in e.g., Hall and Jones (2007) and Fonseca et al. (2009).

[3]That the countries otherwise seem to behave differently in the context of health is nicely illustrated by their aggregate health shares. In 2006 the share of total health spending in GDP was 8.7% in Australia, 10.0% in Canada, 9.4% in New Zealand and 15.3 % in the US.

Figure 1: Health expenditures per capita across age groups: Selected countries 1999-2001



The figure shows log health expenditures per capita by age-group in Australia, Canada, New Zealand and the USA. *Sources*: US from Keehan, Lazenby, Zezza, and Catlin (2004); Canada from Health Canada (2001); Australia from Australian Institute of Health and Welfare (2004); for New Zealand from New Zealand Ministry of Health (2004). *Notes*: (i) In order to consolidate the age intervals for the purpose of illustration simple averages across age intervals and gender have been invoked. (ii) For the USA the first age group concerns individuals aged 19-44. (iii) The year of data collection varies slightly, as indicated by the legend to the figure: The data for the US is from 1999; data for Australia and Canada is from 2000 and data for New Zealand concerns 2001.

spending across the life cycle. One may think about this result as saying that early-in-life "prevention" is increasingly attractive, relative to late-in-life "treatment", if the speed of aging is rapid. Conversely, in societies were the speed of aging is slow, it will be more attractive to plan for rising health expenditure, *ceteris paribus*. From a positive perspective it is interesting to observe that the physiological parameter in question already has been estimated for three of the four countries in Figure 1 (all except for New Zealand), and has been found to be essentially the same (See Rockwood and Mitnitski, 2007, and the discussion below). This is consistent with

the fact that health spending per capita seems to rise across age groups at more or less the same rate in Australia, Canada and the United States.[4]

The theory also predicts that even if health expenditures are rising over the life cycle the expenditure *share* for health may nevertheless remain constant or even decline during life. We derive the condition under which the health expenditure share should rise, decline or remain constant across the life cycle.

The model developed below determines the speed of the aging process and its culmination – death – endogenously. Thus, we are in a position to inquire into the sources of cross-country inequality in longevity. In particular, we can ask how much of the observed differences in life expectancy that can be attributed to differences in the level of per capita health investments prompted by differences in income.

Hence, as an application of the model we revisit the celebrated "Preston curve" (Preston, 1975). More than three decades ago Preston drew attention to the fact that cross-country data reveal a positive (concave) association between income per capita and life expectancy; subsequent research has labeled this mapping "the Preston curve" (see e.g., Deaton, 2003; Bloom and Canning, 2007). Moreover, Preston documented that the curve had been shifting upwards over time and that most of the improvements of life expectancy worldwide seemed to be due to these shifts, rather than income growth (i.e., movements along the curve). In spite of its influence (also outside economics), controversy remains as to its proper interpretation (e.g., Bloom and Canning, 2007). Does the curve represent the influence from income on life expectancy, or the other way around? Maybe income is merely a proxy for other variables of relevance to longevity; education for example? Are the shifts in the curve due to technological change?

Our physiologically founded theory of aging is uniquely suited for inquiries into the causes of rising longevity. Accordingly, we contribute to the above debate by using our model to shed light on the Preston curve. The proposed theory predicts that higher levels of income enable higher levels of (optimal) health investments, which in turn serve to prolong life. Increasing productivity of health investments reduces frailty for any given level of investment and thereby produces greater longevity. We calibrate the model to the average US male in 2000 and proceed to show that the model accounts well for the Preston curve in 2000. Hence, the shape and

[4]Since the four countries in question all share genetic linage (in regards to large parts of the populations at least) the similarity in terms of physiology of the average citizen is perhaps to be expected.

position of the Preston curve can be motivated by a theory of optimal health investments in the presence of physiological aging. By implication: a model of optimal health investments is thus able to explain why life expectancy at age 20 is about a decade shorter in low income countries compared to high income countries. Moreover, a rate of technological progress at about 1.1.% per year can account for the observed shift in the Preston curve between 1980 and 2000.

Our analysis is primarily related to the seminal work of Grossman (1972) on demand for health and to Ehrlich and Chuma's (1990) important work on optimal longevity.[5] The key difference between our work and previous contributions lies in our law of motion for frailty; the counterpart to the law of motion for health capital. Our physiologically founded frailty dynamics implies that "health depreciation" accelerates during life, but may be slowed by investments in health. The law of motion is derived from the law of increasing frailty, implying that the key (physiological) parameters entering the equation all have been estimated with great precision in the medical science literature. In the standard model, by way of contrast, an accelerating depreciation rate needs to be imposed. Even then the standard model contains the counterfactual implication that an improvement in health will lead to faster, not slower, health depreciation. We elaborate on the difference between the standard approach to health capital accumulation and our approach below.

The paper proceeds as follows. In the next section we derive the key law of motion that characterizes the aging process. We also discuss how this law of motion differs from the standard health accumulation equation. Section 3 contains the main analysis of optimal aging and death. In Section 4 we calibrate the model to US data and examine its predictions as well as its dynamical properties in the presence of shocks to income, technology, health prices and more. Then, in Section 5, we apply the model to the study of the Preston curve. Finally, Section 6 is reserved for concluding remarks.

2. Introducing Biological Aging

2.1. **Modeling Human Aging as a Process of Deficit Accumulation.** Aging is defined as the intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death (Arking, 2006). At the individual level the aging process exhibits great heterogeneity, and is only imperfectly captured by chronological age; some 60 year-olds are as fit as some 40

[5]Other notable contributions is Reid (1998), Eisering (1999) and Foster (2001).

year-olds and vice versa. Indeed, biologists and gerontologists stress that individual aging should be viewed as an event-dependent process, rather than as a time-dependent process. There is no such thing as a "biological clock", which determines the speed of individual aging and timing of death.

At the population level, however, age is a better predictor of aging and death. The so-called Gompertz-Makeham law of mortality implies that the death rate rises exponentially with age.[6] The fact that the age-specific mortality rate increases is obviously a manifestation of the aging process. But then why is the death rate increasing?

A reductionist approach might suggest that the organism ages because of aging organs, which in turn is caused by aging tissue, brought on by the aging of cells and so on. Such an approach, however, is a dead end. The reason is that, eventually, a level of "disaggregation" is reached, which consists of non-aging entities: atoms, for example. The aging process can not be understood in this manner. But how can a system constructed from non-aging components age in the manner suggested by the Gompertz-Makeham law of mortality? Why is the death rate (on average) rising with chronological age?

A promising strand of literature in biology has sought an answer by drawing on reliability theory from engineering.[7] To understand the basic idea, consider the following model (Gavrilov and Gavrilova, 1991). Suppose we view the organism as a whole as consisting of a fixed number of individual parts, which we will refer to as "blocks". Each block does not age. That is, the failure rate of a block is constant. This assumption captures that the human organism is ultimately constructed from non-aging components, as noted above. Next, assume that the blocks are connected in parallel, and that the system as a whole is assumed to survive as long as there is one functioning block remaining. This assumption is motivated by the physiological fact that the human organism is characterized by a great deal of redundancy; as young adults the functional capacity of our organs is estimated to be tenfold higher than needed for mere survival (Fries, 1980). Though each block does not age the passing of time will reduce redundancy in the system as a whole, which leads to an increasing failure rate of the system. Hence, the simple model successfully reproduces an exponentially rising death rate with age. Many

[6]More precisely, the Gompertz-Makeham (GM) law states that the age specific mortality rate, $\pi(t)$, evolves with age, t, in accordance with the formula $\pi(t) = a + b\, e^{\alpha t}$ where a, b and α are parameters. The fit of the GM law, for the population aged 20 and above (children and teen-ages are "special") is extremely good, always featuring an R^2 in excess of 0.95. See e.g., Arking (2006)

[7]Reliability theory is used in engineering to understand the failure rate of mechanical devices. Gavrilov and Gavrilova (1991) were the first to introduce reliability theory into biology.

extensions of this basic model have been developed, which have lead to new insights into the aging process.[8] But for present purposes the key point of reliability theory is conveyed by the simple model: senescence can be conceptualized as the gradual loss of redundancy, ultimately leading to organism collapse.

Following the underlying reasoning of reliability theory one may therefore think of aging as being characterized by increasing *frailty*. That is, as the redundancy of the human organism shrinks we become more fragile. An empirical measure of human frailty has been developed by Mitnitski and Rockwood and various coauthors in a series of articles (e.g., Mitnitski et al, 2002a,b; 2005; 2006).

As humans age we develop an increasing number of disorders, which Mitnitski et al. (2002a) refer to as "deficits". Some of these deficits may be viewed as rather mild nuisances (e.g., reduced vision) while others are more serious in nature (e.g., strokes). Nevertheless, the notion is that when the number of deficits rises the body becomes more frail. A frailty index can then be estimated as the proportion of the total potential deficits that an individual has, at a given age.[9]

Mitnitski et al. (2002a) show that the following equation fits data on the proportion of deficits, $D(t)$, of the representative individual at age t very well

$$D(t) = E + Be^{\mu t}.$$

This "law of increasing frailty" explains around 95% of the variation in the data, and its parameters are estimated with great precision. The parameter E turns out to be common for men and women; using a data set encompassing 66,589 Canadians, aged 15 to 79, Mitnitski et al. (2002a) estimate E to 0.02, with a standard error of 0.001. The parameters B and μ are, however, gender specific. For Canadian men (women) $\log(B)$ is $-5.77\pm$ 0.06 (-4.63 ± 0.06), while μ is $0.043\pm$ 0.001 (0.031 ± 0.001). Interestingly, very similar estimates for B and μ are obtained on data for

[8]For example, if one extends the framework we have just sketched by assuming that the body consists of a number of *essential* blocks (so that if one fails the organism fails), each of which consists of non-aging elements of which some initially are defect, the model reproduces the Gompertz-Makeham law; see e.g., Gavrilov and Gavrilova (1991).

[9]Some methodological notes. To be in the index the deficit must have been demonstrated to be a group and individual indicator of health, and an important correlate of survival (Mitnitski et al., 2002a). " The total number of potential deficits" are in practise determined by the survey at hand. This may seem arbitrary. But according to Rockwood and Mitnitski (2007) the exact choice of deficits is usually not crucial. Provided sufficiently many indicators (40 or more) are present in the index, results tend to be relatively unaffected. Note that the construction of the index does not preclude that its value declines on occasion.

Australia, USA and Sweden (Rockwood and Mitnitski, 2007). Hence, in these four developed countries (in spite of differences in samples, the precise contents of the frailty index etc.) the average individual accumulates 3-4% more deficits from one birthday to the next.

The strong ability of the deficit model to fit the data is encouraging. But a more intriguing finding is that there appears to be a very strong link between the path of frailty and the path of mortality. If average deficits of individual age groups are plotted in a $(t, \log[D(t)])$ diagram, the resulting lines for men and women intersect. While men start out with lower deficits ($D(0) = 0.023$ compared to 0.029 for women) they tend to accumulate deficits at a higher rate. Eventually, therefore, the proportion of deficits converge. The age at which deficits coincide - based on the estimates reported above - is 94. Now, when the age-specific mortality rate (the Gompertz-Makeham equation; see footnote 6) is depicted, a similar intersection point is found. Hence, in terms of mortality there is also a trade-off between the initial death rate and the speed at which the age specific mortality rate goes up. What is remarkable is that the intersection for mortality occurs at roughly the same age as that of frailty: at age 95. In fact, even when one compares mortality patterns across different countries the same "age of convergence" is found. In biology this fact is referred to as "the law of compensating mortality"; initial high mortality appears to be "compensated" by a more slowly rising mortality rate during life.[10] Similar intersection points are also found in other species, albeit the age of intersection naturally is different from species to species; biologists refer to the age at which an intersection is found as the "species-specific lifespan" (e.g., Gavrilov and Gavrilova, 1991, p. 148-56).

Hence, data on frailty and mortality independently predict gender convergence of aging, at roughly the same chronological age (for the average individual). This result suggests that the process of deficit accumulation indeed is capturing important aspects of the aging process. Indirectly, the results support the theoretical proposition of reliability theory. Namely, that aging involves increasing frailty, which eventually leads to organism collapse and death.

We can restate the law of increasing frailty in flow form by differentiating with respect to age t:

$$\dot{D}(t) = \mu D(t) - \mu E, \tag{1}$$

[10]The law of compensating mortality was first documented by Strehler and Mildvan (1960). The - immensely strong - correlation between initial mortality and the rate at which it rises with age is therefore usually referred to as the "Stehler-Mildvan correlation".

where E exhibits no gender variation, and works to slow down the speed of deficit accumulation. The parameter is interpreted, in the natural science literature, as capturing non-biological factors impact on deficit accumulation (Mitnitski et al., 2002a). Accordingly, we will assume that E is amendable to change by way of deliberate investment. This is the way in which the individual may attempt to slow down aging in the model below.

Specifically, we propose the following parsimonious refinement of the process of deficit accumulation:

$$\dot{D}(t) = \mu D(t) - a - Ah(t)^{\gamma}, \tag{2}$$

where $D(0)$ is given. The parameter a captures environmental influence on aging beyond the control of the individual (pollution, say), the parameters $A > 0$ and $0 < \gamma < 1$ reflect the state of the health technology, and h is health investment. While A refers to the general power of health expenditure in maintenance and repair of 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 highly deteriorated human bodies.

By way of contrast to E, the parameter μ – impressed by its empirical constancy across developed countries – is considered to be a physiological parameter. In the remaining we will refer to this physiological parameter as *the force of aging*, as it drives the inherent and inevitable process of human aging. While the force of aging is exogenous, it is unlikely to be universally constant. In fact, it probably varies across countries in different parts of the world, much like the speed at which the death rate rise with age, according to the Gompertz-Makeham law, varies across countries (see Strehler and Mildvan, 1960). However, we would simultaneously expect that countries with higher μ to be characterized by a lower value of B, which ensures the "age of convergence" is unaltered around 95 years. That is, we would expect a "law of compensating frailty" to be found in the data (Mitnitski et al., 2002a)

In order to capture death, we need to invoke an upper boundary to deficit accumulation, $\bar{D}$. In the analysis below the representative individual remains alive as long as $D(t) < \bar{D}$. Direct evidence on the existence of an upper boundary for D is found in Rockwood and Mitnitski (2006).[11]

[11] A somewhat similar assumption is found in the existing literature on optimal health investments: here individuals expire if the health index falls below a postulated lower boundary (Grossman, 1972; Erhlich and Chuma, 1990). We will turn to the differences between the two approaches shortly.

Observe that equation (2), along with the restriction that $D(t) < \bar{D}$, provides a complete description of aging until death. In this process, chronological age does *not* play a role in itself. While the model developed below concerns optimal aging and death of a representative agent of a cohort, it is nevertheless worth noting that this formulation is in concordance with a central point made by biologists and gerontologists: individual aging is *not* time-dependent. This follows since $\dot{D(t)}$, by (2), is only influenced by current investments and accumulated deficits; t (chronological age) plays no independent role.

This completes the development of the central equation which governs aging. But before we turn to the analysis of optimal aging and death, we briefly compare our law of motion for deficits with the familiar law of motion from health, which dates back to the work of Grossman (1972).

2.2. **Deficit Accumulation vs Health Accumulation.** Usually, health is introduced as a state variable similar to human capital. In its most basic form, this would involve an equation such as $\dot{H}(t) = I(t) - \delta H(t)$, where $I(t)$ is investment in health and $H(t)$ is the stock of health capital (see e.g. Ehrlich and Chuma, 1990).

This equation has a rather unfortunate implication. It predicts that health depreciation is greater when the stock of health is large (δH), which, of course, usually would mean when individuals are relatively young. In reality, the process of aging is a process where the rate of decline in health status accelerates during life; as explained above, both health deficits and the mortality rate rises exponentially with age, implying slow aging early in life and rapid deterioration in latter stages. In practise, therefore, health losses are greater in states where the health index is *low*, which usually means when one reaches a more advanced age. A related problem is that this law of motion for health opens the door to immortality: a constant investment level that fulfills $I^*/\delta = H^*$ would keep the stock of health capital constant at H^* forever. As long as H^* exceeds the minimum health level needed for survival, H_{min}, death does not take place.

Of course, these problems have not gone unnoticed. Since the work of Grossman (1972), the standard "fix" has been to introduce an age-dependent rate of health depreciation, $\delta(t)$, with $\dot{\delta}(t) > 0$. While the rate of change in the rate of depreciation may seem intuitively appealing the question remains how *exactly* the function is to be specified. Insofar as one does not get this

choice "right" the resulting model will represent an inaccurate description of the aging process, and, by extension, its predictive power will be diminished.[12]

However, even with an age-varying depreciation rate the law of motion for health still holds a counterfactual implication: an improvement in the agent's health status (H) accelerates health depreciation ($\delta(t)H$). The model implies that investments that improve health accelerate aging. This effect tends to make health investments unattractive when the stock of health is high, which in practise means that early-in-life health investments become unappealing.

The approach proposed above, by way of contrast, provides a physiological foundation for health depreciation. To convert our equation for deficit accumulation into one for health accumulation, assume health is defined as best attainable health minus accumulated frailties: $H = \bar{H} - D$ where $\bar{H}$ is "maximum health"; the state of health of a normal 15 year old, say. This implies $\dot{H} = -\dot{D}$. Inserting equation (1) into into $\dot{H}$ and substituting D for $\bar{H} - H$ provides $\dot{H} = -\mu\left(\bar{H} - H - E\right)$, or,

$$\dot{H}(t) = \mu E - \mu(\bar{H} - H(t)). \tag{3}$$

Following the approach above, we may associate E with investments. Finally, using the terminology of Grossman (1972) and Ehrlich and Chuma (1990) we may define the minimum level of health below which life is infeasible as $H_{min} \equiv \bar{H} - \bar{D}$, where $\bar{D}$ is maximum deficits. We are thus left with a simple linear differential equation for health, which differs in one crucial respect from the one adopted in the economics literature: Consistent with the facts, the equation predicts that health loss is small at a good state of health and increasing losses are predicted when the health stock deteriorates. Hence, a meaningful description of how health deteriorates with age (as the health index erodes) is implicit in our frailty equation. Notice that according to equation (3) an improvement in health status works to slow down aging; i.e., it stimulates health accumulation.

The key advantage of our modeling approach is that various parameters, μ for example, have clear empirical counterparts. This is also the case for the frailty index itself, which has already been developed and tested in the natural sciences. These aspects are very useful in the context

[12]Making $\delta(t)$ an *explicit* function of time implies that chronological age is supposed to matter to aging per se. This is a problematic assumption from a biological perspective, as explained above. This position of biologists is supported by work of health economists. For example, Zweifel et al. (1999) demonstrate that among the elderly health expenditure is not predicted by chronological age once "time remaining until death" is controlled for. This suggest that health status (e.g., frailty), and not the year on the birth certificate, is what matters to health investments.

of calibration, and will be valuable if the presently modified version of the health model is taken to the data and tested for its economic implications.

3. A Theory of Optimal Aging and Death

3.1. **The optimization problem.** Consider an adult maximizing utility from consumption $c(t)$ over his or her life. We are considering a representative member of a cohort, for which reason the maximization problem can be viewed as deterministic to a first approximation. By considering a deterministic framework we are thus following Grossman (1972) as well as Ehrlich and Chuma (1990).[13] Initial time is for convenience normalized to zero and instantaneous utility is CRRA. Longevity T is finite and endogenous. Let $\rho \geq 0$ denote the rate of pure time preference; the rate of time preference need not be strictly positive in order for the problem to be well-defined. Summarizing, intertemporal utility is given by

$$\int_0^T \mathrm{e}^{-\rho t} u(c(t)) \ \mathrm{d}t \tag{4}$$

with $u(c) = (c^{1-\sigma} - 1)/(1-\sigma)$ for $\sigma \neq 1$ and $u(c) = \log(c)$ for $\sigma = 1$. The inverse $1/\sigma$ provides the intertemporal elasticity of substitution.

It is perhaps tempting to allow frailty to enter the per period utility function as well. We will nevertheless refrain from doing so. In this manner we are much more constrained when trying to match the cross-country data on longevity and income in Section 5 below. Nevertheless, as will become clear, the simple model matches the cross-country data remarkably well. In only allowing health investments to affect utility via longevity, our approach is similar to that of Becker (2007).

The individual receives a wage income w. We assume the wage rate is constant during life; a simplifying assumption which serves to highlight the central workings of the model. Income can be spend on consumption goods c or on health goods h. The relative price of health goods is p. While consumption goods are directly utility enhancing, health goods (e.g. a hip replacement, a weekend at the spa) are instrumental in repairing or delaying bodily decay and, ultimately, in prolonging the period during which consumption goods can be enjoyed. Accordingly, the only value of "good health" in this model is via its impact on longevity. Besides spending income

[13]Nevertheless it could be interesting (and very challenging) to add uncertainty. For instance, by admitting individual-level uncertainty one could subsequently examine whether the "deterministic solution" derived here continues to be fulfilled "on average". This difficult task is left open for future research.

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 both p and r are parametrically fixed. The law of motion for individual wealth is thus given by

$$\dot{k}(t) = w + rk(t) - c(t) - ph(t). \tag{5}$$

The individual is assumed to inherit wealth $k(0) = k_0$, and leave a bequest $k(T) = \bar{k}$ (which both could be zero).

The problem is to maximize (4) subject to the accumulation equations (2) and (5), the initial conditions $D(0) = D_0$, $k(0) = k_0$, and the terminal conditions $k(T) = \bar{k}$, $D(T) = \bar{D}$. 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 $h(t)$.

Note that optimal life-length T enters through the free boundary condition and not through the first order condition. This means that the first order condition will provide a maximum irrespective of whether the level of utility is positive or negative (i.e., whether σ is larger or smaller than one). As a result, in contrast to e.g. Hall and Jones (2007) we do not need to add a (sufficiently large) constant to per period utility in order to obtain a meaningful solution for optimal longevity (see Appendix A for details). From now on time indices are suppressed in the interest of brevity.

3.2. **Optimal Aging.** From the first order conditions we obtain the Euler equations

$$g_c \equiv \frac{\dot{c}}{c} = \frac{r - \rho}{\sigma} \tag{6}$$

$$g_h \equiv \frac{\dot{h}}{h} = \frac{r - \mu}{1 - \gamma}. \tag{7}$$

While equation (6) is the standard Consumption Euler equation, equation (7) provides a novel Euler equation for health expenditures: the "Health Euler equation". In the context of non-health expenditure a higher intertemporal marginal rate of transformation (i.e., a higher real rate of interest) calls for growing health expenditures. At the same time, growth in health expenditures is tempered by the force of aging, μ. The intuition is that if μ is high, deficits will accumulate very fast at the end of life, making late-in-life health investments a relatively ineffective way of prolonging life. Instead, the optimal strategy is to invest more heavily early in life. Conversely, if the force of aging is low (i.e., μ is "small") late-in-life health expenses

are more effective in prolonging life, for which reason it can be optimal to allow h to grow over time. Finally, growth of health expenditure is also influenced by γ, which captures the curvature of the health investment function: a larger γ increases growth in health expenditures, *ceteris paribus*. 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. Thus, in this setting where health does not enter the per period utility function it is the state of technology that determines the extent of expenditure smoothing across life.

It is interesting to note that it is not *necessarily* optimal to plan for increasing health expenditures during life. In societies where the force of mortality is sufficiently strong it may be the optimal strategy to prioritize early-in-life health spending, and thus allow health spending to *decline* over time. One way to think about this result is to associate early-in-life investments in health with "preventive measures" and late-in-life health expenses with "treatment measures". If so, then the Health Euler simply says that in societies where individuals are aging at a rapid pace ($r < \mu$) it is optimal to focus resources on prevention, rather than on treatment.

Overall, the model predicts that countries that are technologically similar, and are inhabited by genetically similar populations should exhibit similar investment patterns across time, consistent with the parallel growth paths depicted in Figure 1 for Australia, Canada, New Zealand and USA.

Even if it is optimal to allow health expenditure to rise over time, the expenditure *share* for health, $\epsilon_h \equiv h/(h+c)$, may nevertheless fall, if pure consumption is growing sufficiently rapidly. Hence, the expenditure share for health is increasing over time if $g_h > g_c$, or (using (6) and (7)) if

$$r - \frac{1-\gamma}{\sigma} \cdot (r - \rho) > \mu, \tag{8}$$

If the ratio $\frac{1-\gamma}{\sigma}$ increases it becomes less likely that the condition is met. The interpretation is that a small σ leaves little incentive to smooth consumption, which implies (*ceteris paribus*) faster growth in consumption. Meanwhile, γ parameterizes the incentive to smooth health investments; the smaller γ is the greater the incentive to smooth health investments. Consequently, when $\frac{1-\gamma}{\sigma}$ increases it implies a greater desire, on the part of the consumer, to smooth health investments relative to consumption, which suggests a declining share of health during the life cycle as long as $r > \rho$.

A higher rate of interest will increase the rate of growth of both health investments and consumption. But whether a higher r makes it more likely that the condition is fulfilled depends on the incentive to smooth consumption and health investments, respectively. If consumers have a greater desire to smooth health investments ($\frac{1-\gamma}{\sigma} > 1$) a higher r reduces the health expenditure share.

This leaves the impact of ρ and μ. Health spending is independent of ρ since h prolongs life, but does not affect per period utility, while a higher ρ (as usual) works to slow down consumption growth.[14] Hence, it becomes more likely that the health share is rising if individuals are highly impatient. At the same time, in the presence of a greater force of aging people will act more impatiently vis-a-vis health investments, inducing them to invest early in life. If the force of aging is sufficiently strong, the expenditure share for health will therefore be declining over the life cycle.

The bottom line is that the life cycle path for the health share is ambiguous. In particular, it is not a given that the expenditure share should be rising with age. In theory one may therefore expect differences in the health share across countries that differ in terms of culture, technology and physiology.

3.3. **Optimal Death.** In contrast to the pioneering contributions on the topic of optimal health investments (Grossman, 1972; Ehrlich and Chuma, 1990), the present model is sufficiently simple to allow for an explicit solution for optimal longevity T.

In order to see this, first note that the boundary value problem with variable terminal value T requires that the boundary conditions $D(0) = D_0$, $k(0) = k_0$, $k(T) = \bar{k}$, $D(T) = \bar{D}$ and $h(T) = 0$ are fulfilled. Then integrate (5) in order to solve for $k(T)$ and integrate (2) in order to solve for $D(T)$. Finally solve the associated Hamiltonian for $H(T) = 0$. This provides the following set of equations (see Appendix A for details):

$$\bar{D} = D_0 \exp(\mu T) - \frac{Ah(0)\exp(\mu T)}{g_D}\left[\exp(g_D T) - 1\right] + \frac{a}{\mu}\left[\exp(\mu T) - 1\right], \tag{9a}$$

$$\bar{k}\exp(-rT) = k_0 - \frac{w}{r}\left[\exp((r)T) - 1\right] + \frac{c(0)}{g_c - r}\left[\exp((g_c - r)T) - 1\right] - \frac{ph(0)}{g_D}\left[\exp(g_D T) - 1\right] \tag{9b}$$

$$0 = u_T - \frac{\exp(-\sigma g_c T)}{c(0)^\sigma} \times \tag{9c}$$

[14] Appendix A offers a more detailed discussion of why g_h is independent of ρ.

$$\left\{\frac{(\mu\bar{D}-a)}{\gamma A}ph(0)^{1-\gamma}\exp((1-\gamma)g_hT)-\frac{1-\gamma}{\gamma}ph(0)\exp(g_hT)-w-r\bar{k}-c(0)\exp(g_cT)\right\}$$

where $g_D \equiv (\gamma r-\mu)/(1-\gamma)$ and $u_T \equiv \log(c(0))+g_cT$ in the case of log-utility and $u_T \equiv [c(0)\exp(g_cT)-1]^{1-\sigma}/(1-\sigma)$ otherwise. These three equations can be solved for the three unknowns: $c(0)$, $h(0)$, and T. Having found the optimal initial values and the optimal terminal time, the four-dimensional dynamic system (2) and (5) – (7) is fully specified and it can be solved for the optimal life-cycle trajectories of c, h, k and D.

4. Comparative Dynamics

4.1. **Calibration and Model Predictions for the US.** Before we turn to the experiments, we calibrate the model to USA data. In this endeavor we begin by employing the Health Euler (7) in order to calibrate the curvature of the health production function, i.e. γ. Using data from Figure 1 we put $g_h = 0.021$, to capture the growth of health spending across the life cycle. From Mitnitski et al. (2002a) we take the estimate of $\mu = 0.043$ for (Canadian) men, and finally we put $r = 0.06$ (e.g., Barro et al., 1995).[15] This produces $\gamma = 1-(r-\mu)/g_h = 0.19$, which squares well with the independent estimates obtained by Hall and Jones (2007).[16]

Mitnitski et al's regressions do not involve children. Much like the Gompertz-Makeham law for mortality, individuals below roughly the age of 20 are presumably not well described by the law of frailty (in stark contrast to the group above 20). Hence, when calibrating the model we assume individuals are "born" at the age of 20.

With this in mind we do the following in order to parameterize the deficit accumulation equation. From Mitnitski et al.'s (2002a) regression analysis we can back out $D(0) = D(20) = 0.0274$ as the relevant initial value for a 20 year old and $\bar{D} = 0.1005$ 55.6 years later; the life-expectancy of a male US American in the year 2000 was 75.6. In 1900 the life expectancy of a 20 year old U.S. American was 42 years (Fries, 1980). We will think of 1900 as characterized by virtually no health investments. Hence, we therefore set $a = 0.00055$ so that the model predicts a life-expectancy at 20 of 42 years, assuming *no* health investment.

[15] As explained in Section 2: the force of aging within the US and Canada are similar (Rockwood and Mitnitski, 2007). Thus, using the estimate from the Canadian sample should be a good approximation. While Rockwood and Mitnitski (2007) stress the similarity of their results for US and Canadian populations they do not report the detailed results for their US analysis, for which reason we are forced to rely on the results from the Canadian sample.

[16] Hall and Jones allow the curvature of the health production function to be age dependent. The average value is close to 0.2.

Since consumption tends to be essentially constant across the life cycle, once family size has been taken into account (Browning and Ejrnæs, 2009), we put $\rho = r$. The most natural specification of the intertemporal elasticity of substitution is probably unity (Chetty, 2006). So we set utility to be logarithmic for the benchmark case. In order to focus on health expenditure as a motive for savings we assume $k(0) = k(T) = 0$ for the baseline simulation.

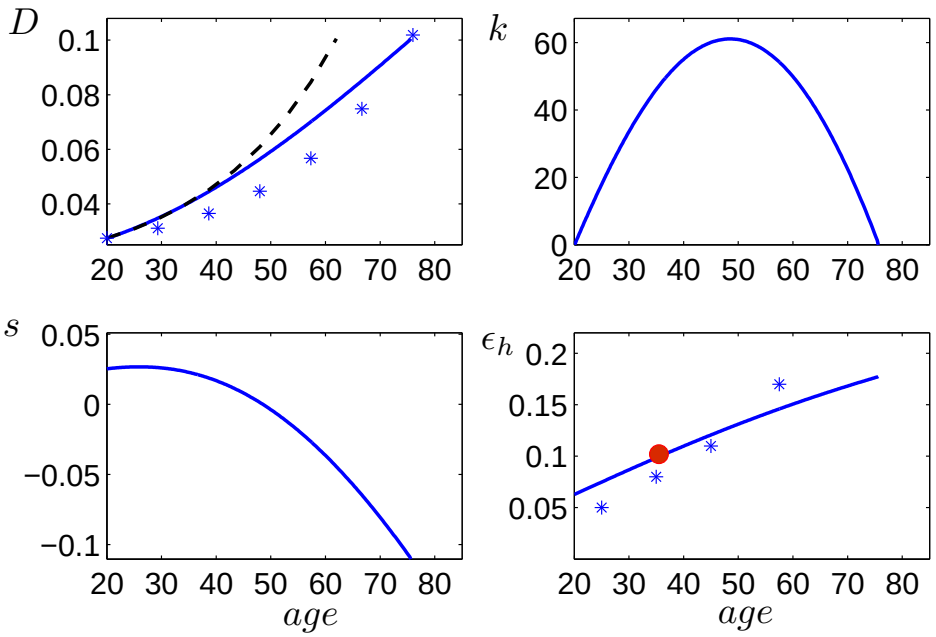
Finally, we set w equal to GDP per worker in the US in the year 2000 (PPP$ 77,003) and adjusted A such that the individual dies with deficits $\bar{D}$ at age 75.6, which was the life-expectancy of a 20 years old US American male in the year 2000. This provided the estimate $A = 5.596 \cdot 10^{-5}$.

In sum, the model is calibrated to match initial deficits, end-of-life deficits, and longevity in 2000; $D(0)$, $\bar{D}$ and T, respectively. It also matches the growth of health spending across age groups exactly. Yet the *path* of deficits (between age 20 and death), as well as the evolution of expenditure *shares* across the life cycle, are left unrestricted. This provides the opportunity for an informative consistency check of our approach: does the path of the health expenditure share and cohort frailty match the data?

Figure 2 shows the basic run of the model; including the association between model predictions and actual data. In the lower right panel of Figure 2 stars indicate actual health expenditure share by age-group inferred from Mazzocco and Szemely (2010), which can be compared to the solid line representing the prediction from the calibrated model. Admittedly, our calibration fails to match the actual expenditure for the oldest age group (0.28 for ages 65+). We conjecture this failure is mainly caused by the fact that our model does not allow wages to decline with age. Nevertheless, the model does well on average. If we calculate the age-structure weighted health expenditure share from the data it comes to 10.8%. Taking the mean age of the male US citizen in 2000 (35.4) and pugging it into the model we match this number *exactly*, as indicated by the red dot in the figure.

In the upper left window the accumulation of deficits for the average US (male) citizen is depicted. The dotted line reflects the scenario where there are no health investments occurring; the solid line involves optimal health investments as predicted by the model. In the figure we also illustrate the law of frailty, as estimated by Mitnitski et al. (2002a) (represented by stars). As can be seen, the model's fit is rather good in that the path of deficits is fairly close to the one found in the data. We are slightly overestimating the level of deficits during life. But on the

FIGURE 2: OPTIMAL AGING: BASIC RUN



Solid lines: basic run. $T = 55.6$. Parameters: $a = 5.5 \cdot 10^{-4}$, $A = 5.59 \cdot 10^{-5}$, $\mu = 0.043$, $\rho = r = 0.06$, $D_0 = 0.0274$, $\bar{D} = 0.10$, $p = 1$. Dashed line: no health expenditure ($A = 0$) stars: data, red dot: US mean male age and age-structure-weighted expenditure share.

whole the model seems to do well in predicting the evolution of spending shares on health across the life cycle, and the evolution of deficits (i.e., the aging process) for the average US citizen.

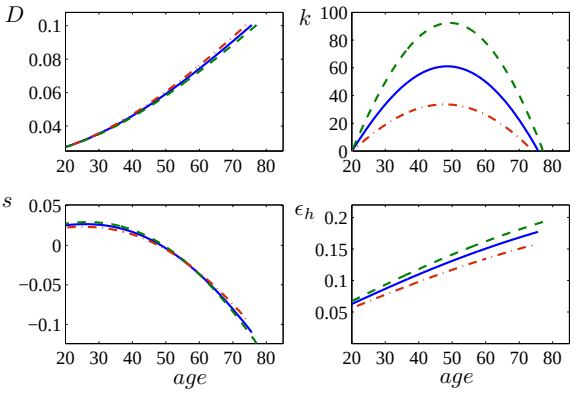
Finally, the model also holds predictions about the path of the savings rate and wealth across the life cycle. Contingent on the imposed parameter values health expenditures are rising during life, as we just saw, which requires early-in-life savings so as to finance late-in-life investments. The wealth of the representative individual therefore follows a "hump shaped" trajectory; as seen from the upper right hand side window, supported by positive savings early in life and dissaving late in life (lower left hand side window). These patterns are qualitatively consistent with the standard life cycle theory of consumption. We have not examined whether the model matches wealth and savings quantitatively, as it seems unlikely that we can predict wealth during working years without a careful discussion of e.g. bequest.[17]

4.2. **Experiment 1: Income.** Panel A of Figure 3 shows how the agent reacts if his income, w, is perturbed. The green (dashed) line is associated with an increase in w of 1/3, the red (dashed-dotted) line depicts the reaction to a reduction of w by 1/3 (in all the experiments below,

[17]The relative importance of life cycle motive vs. the bequest motives for saving is still in debate (e.g., Dynan et al., 2002).

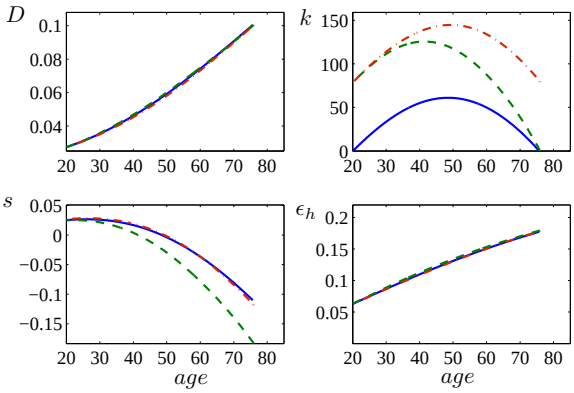
FIGURE 3: HEALTH AND WEALTH OVER THE LIFE CYCLE

A: Variation of Labor Income



Green (dashed): wage income increases by 1/3. $\Delta T = 1.66$, implied elasticity 0.09). Red (dotted): wage income decreases by 1/3 ($\Delta T = -1.60$, implied elasticity -0.09).

B: Variation of Wealth



Green (dashed lines): $k(0) = w$, $\bar{k} = 0$ ($\Delta T = 0.47$). Red (dotted): $k(0) = w$ and $\bar{k} = w$ ($\Delta T = 0.45$).

"green" is associated with increases, and "red" with reductions in the parameter of interest). As can be seen from the figure, the consequence of higher income is an increase in longevity, peak wealth and the share of health spending. In regard to the latter, note that we are keeping r, ρ, μ, γ constant. Hence the increase in the health share solely reflects a "level effect" through $h(0)/c(0)$. The intuition for why income changes entail a larger change in health spending than regular consumption is that the incentive to smooth the latter is stronger due to diminishing - per period - marginal utility. Higher income therefore leads to a larger adjustment in $h(0)$ than $c(0)$.

The issue of main interest, however, is the quantitative impact on longevity. As seen from the top left hand side corner of panel A, the impact is modest though not inconsequential. An increase of income of 1/3 (achievable in a generation with a growth rate of about 1% per year) translates into an increase in longevity of 1.7 years; the reduction involves a fall in life span of 1.6 years.[18] If we convert the impact into an elasticity - the elasticity of longevity with respect to income - we find it to be around 0.090. These effects are not outlandish in comparison to econometric estimates (e.g., Pritchett and Summers, 1996).

4.3. **Experiment 2: Wealth and Bequest.** In the baseline model we did not allow for wealth transfers. Hence, an interesting question is how longevity changes if the individual receives an inheritance, and is forced to pass on bequest. The dashed line in Figure 3, panel B, examines

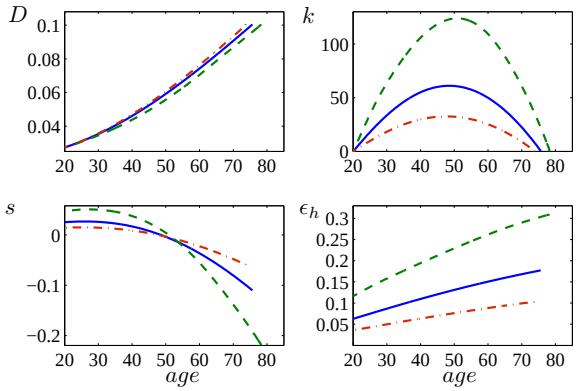
[18]The system is very non-linear, for which reason the results from our experiments is unlikely to be symmetrical.

the impact from offering the individual a transfer comparable to her annual wage, while she is not obliged to pass on any bequest. The dotted line then examines the effect from forcing the individual to pass on a bequest equal to the initial transfer.

When agents receive an inheritance they are naturally able to invest more in health than otherwise, as it increases their life time income. It is interesting to observe, however, that what really matters is the initial inheritance; if bequests are passed on the simulated increase in longevity is about half a year (0.45 years), whereas the increase is only marginally higher without the bequest requirement. Why is the bequest requirement so relatively inconsequential? The intuition is that what dominates over a lifetime is the accumulated interest on the initial bequest. A 6% annual interest will double the value of the initial inheritance almost every decade. In this light it is perhaps unsurprising that the paying of one years wages in bequest has a relatively minor impact on the results.

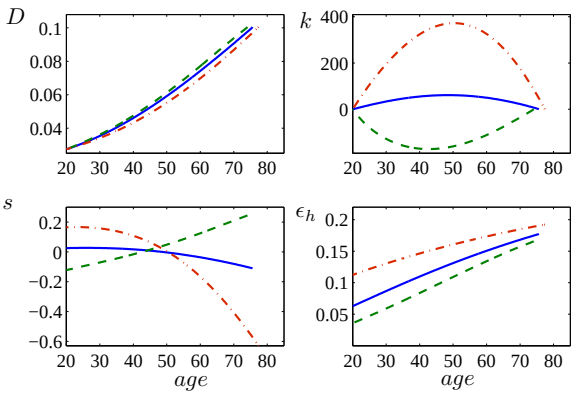
FIGURE 4: HEALTH AND WEALTH OVER THE LIFE CYCLE

A: Variation of Intertemporal Elasticity of Substitution



Green (dashed): $\sigma = 1.1$ ($\Delta T = 2.82$). Red (dotted): $\sigma = 0.9$ ($\Delta T = -1.71$).

B: Variation of Time Preference



Green (dashed): $\rho = 0.07$ ($\Delta T = -1.30$), Red (dotted): $\rho = 0.05$ ($\Delta T = 2.00$).

4.4. **Experiment 3: Preferences.** In this section we examine the influence from the intertemporal elasticity of substitution (IRS) ($= 1/\sigma$), as well as shocks to the rate of time preference, on aging and longevity.

A lowering of IRS (an increase in σ) implies that the individual has a greater preference for consumption smoothing than in the log case (See Figure 4, Panel A, dashed lines). Since we maintain a constant level of consumption throughout life, this change in preferences can therefore only manifest itself in a lower initial level of consumption. This also brings higher savings, a higher level of health investments and thus greater longevity. Quantitatively a small change

in values vis-a-vis consumption smoothing (an IRS of 0.9 rather than 1) yields an increase in longevity of about 2.8 years. Again the effect is asymmetric; an increase in IRS yields a reduction in life span of roughly 1.7 years.

In Panel B of Figure 4 we examine the consequences of an increase in "thrift"; the individual has greater preference for savings (less impatience). This change boosts the health share, though mainly because of a lowering of the consumption level. Still, due to the associated rise in savings health investments are stimulated as well, and therefore longevity. The end result is an increase in life span of 2 years. The figure also illustrates the reverse case: an increase in impatience. The ensuing reduction in longevity is 1.3 years.

Overall these experiments suggest that cultural differences across countries may have a non-negligible impact on longevity, to the extent such variations manifest themselves in preferences toward saving. This finding might shed some light on the fact that Japan is the country in the world with the highest longevity; Landes (1999), for instance, argues that "thrift" is a deeply rooted cultural trait in the Japanese society. Among rich nations, at roughly the same level of prosperity, such cultural differences could be what makes the difference.

4.5. **Experiment 4: Health costs.** Our next experiment concerns health costs; the relative price of h. Here we consider a doubling of the relative price of health goods. Rising relative health prices is a realistic scenario; the CPI of medical care has risen faster than the GDP deflator in the US (Cutler et al., 1998).[19]

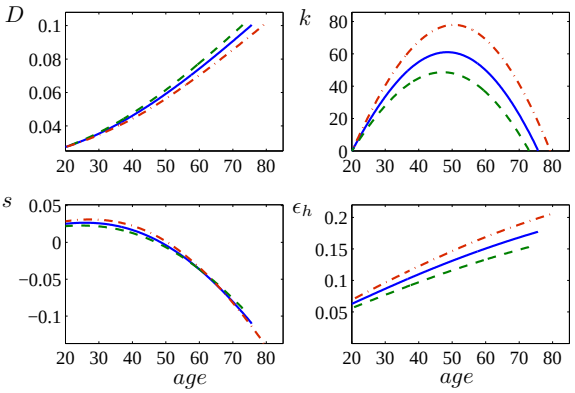
As is clear from Panel A of Figure 5, when the relative price of health increases, individuals substitute towards regular consumption. As a result, the health share declines. With less health investments, savings decline as well. The end result of a doubling of the relative health price for longevity is a reduction by 2.5 years. This amounts to a longevity-price elasticity of 0.09. Interestingly, the impact from a 1% increase in the relative price is quantitatively comparable to the impact of changes in income, as discussed above. Since the relative price of medical care has risen faster than GDP per capita, the price effect could in principle have "annulled" the potential benefits from rising income, seen through the lens of our model.

4.6. **Experiment 5: Health Technology.** In Figure 5, panel B, we examine the impact of health technologies on longevity. In the experiments above, the impact from the parameter of

[19]However, according to Cutler et al., productivity of health care has also risen. Indeed, the authors argue that relative medical prices, appropriately quality adjusted, might have been declining over time. Below we examine the influence from changes in health productivity.

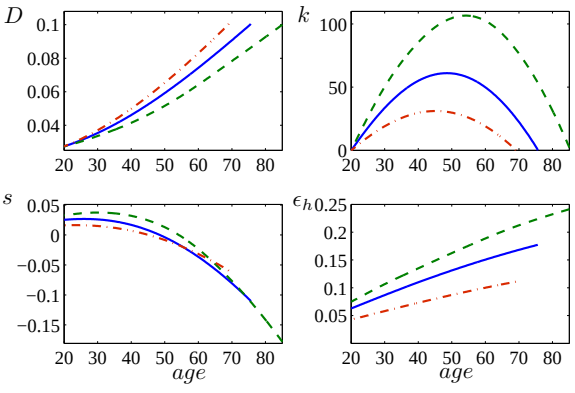
FIGURE 5: HEALTH AND WEALTH OVER THE LIFE CYCLE

A: Variation of Health Costs



Green (dashed):doubling p, $\Delta T = -2.48$, implied elasticity -0.09). Red (dotted): halving p ($\Delta T = 3.84$, implied elasticity 0.13).

B: Variation of General Medical Effectiveness (A)



Green (dashed): 33 percent increase of A ($\Delta T = 9.8$, implied elasticity 0.53). Red (dotted): 33 percent decrease of A ($\Delta T = -6.3$, implied elasticity -0.34).

interest were indirect. For instance, an increase in income translates into both higher health spending and higher consumption. Medical technologies (or the productivity of health investments more broadly), however, have a direct impact on the evolution of deficits and therefore on longevity. A larger impact is therefore to be expected.

In Panel B we examine the impact from increasing A by 1/3; an increase of a similar magnitude to that which we analyzed in Section 4.2, in terms of income. If health productivity rises by 33% the consequence is an increase in longevity of nearly a decade. The implied elasticity is about 1/2. This is a very large effect, suggesting that the impact from improvements in health productivity easily may have towered that of rising income.

5. APPLICATION: THE PRESTON CURVE

In his classic paper Preston (1975) documented two central facts: (i) there is a positive and concave cross-country relationship between GDP per capita and life expectancy at birth, (ii) the curve tends to shift upwards over time. Subsequent research has found that the Preston curve still holds, and continues to shift upwards over time (e.g., Deaton, 2003). Moreover, it has been shown that the relationship between income and longevity emerged sometime late in the 19th century (Bloom and Canning, 2001).

While the key regularities established by Preston (1975) thus continue to hold, questions remain as for their interpretation. Preston (1975) himself argued that an impact from income

on longevity could occur in various ways, including via diet, access to clean water and sanitation, as well as medical treatment. At the same time, it is hard to exclude that the same link could be attained with causality running from life expectancy to income. Or, perhaps income is simply correlated with something else that matters to longevity, like human capital (Bloom and Canning, 2007). This ambiguity also leaves the source of shifts in the Preston curve open to debate.

Our model establishes a link between income (in the form of wages) and life expectancy (in the form of longevity, T). The mediating factor is health investments, h, which would include basic investments like access to clean water and sanitation, as well as more sophisticated investments associated with medical treatment. But are these mechanisms sufficient to create the empirical income gradient Preston discovered? Can plausible changes in technology account for the changes in the position of the Preston curve over time?

In addressing these questions we perform the following exercise. We begin by feeding varying income levels through the model we calibrated to US data above; the variation in income is determined by the cross-country data. For each income level we let the model predict longevity, thereby providing a "model Preston curve" for individual years. With the predicted mapping between income and longevity in hand we can then compare it to the "income gradient" found in the data. In addition, we can investigate whether changes in health technology (A) plausibly can account for the observed shifts in the Preston curve over time.

In taking the model to the data we are not postulating that the average individual in each country around the world is actually solving a dynamic optimization problem, thus deducing optimal spending and thereby aging and time of death. But it could be that the behavior of individuals and governments gradually adjust, perhaps by way of example of others (individuals, countries), so that outcomes move towards optimality. Naturally, whether this is the case or not is an empirical matter to which we now turn.

While the original Preston curve concerns GDP per capita and life expectancy at birth, we obviously need to modify the "input data" slightly. Our model does not involve children; life expectancy at birth is therefore not the optimal empirical counterpart to T. Instead, we collected data on male life expectancy at the age of 20, to retain consistency with our calibration for the US.[20] We assembled a data set covering two points in time, 2000 and 1980, for a (only partially

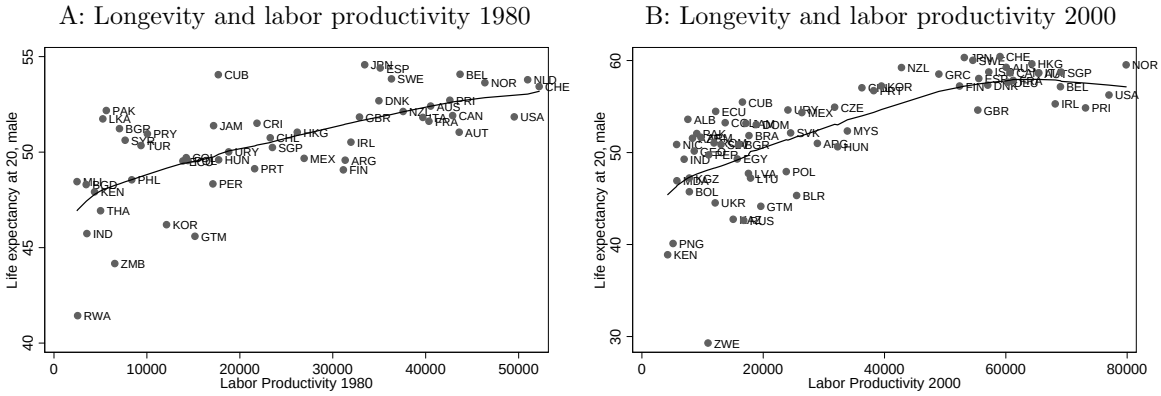
[20]All data sources are found in Appendix B.

overlapping) cross-section of countries. Unfortunately, life expectancy at age 20 is not recorded consistently each year in all countries; so our actual data involves observations circa 2000 and 1980. Our income measure is only slightly different from the one employed by Preston (1975) in that we opt for GDP per worker rather than GDP per capita. GDP per worker is arguably a more appropriate empirical proxy for wages w, the natural income measure in our model.

The country sample was restricted in two ways (beyond via availability of the basic data): following Preston (1975) we ignore countries with a population below 2 million in 1980, and in addition we omitted the OPEC countries; GDP per worker is probably a poor guide to average wages in these countries.[21] This leaves us with a sample of 51 countries in 1980, and 66 countries in 2000.

With this data in place we estimated the income gradient semi-parametrically. That is, we estimate the equation $y_i = f(z_i) + x_i\beta + \epsilon_i$, where the dependent variable is life expectancy at the age of 20, z_i is GDP per worker, x_i is the observation year for life expectancy which adjusts for the fact that life expectancy is not measured in *exactly* the same year, and ϵ_i is a noise term. The function f is assumed to be a smooth, single-valued function with a bounded first derivative, but data decides the exact relation between GDP per worker and life expectancy. This is the income gradient we subsequently compare our model's predictions to.

FIGURE 6: THE MODIFIED PRESTON CURVE



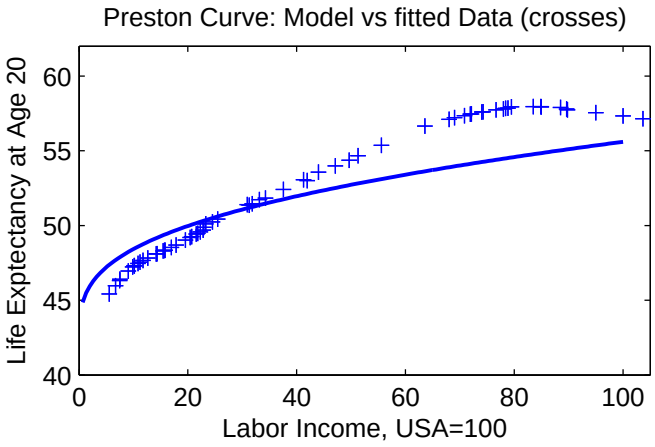
The Figure shows the cross-country link between life expectancy at age 20 and GDP per worker in 1980 (left, 50 countries) and in 2000 (right, 65 countries). *Notes:* The line is estimated semi-parametrically, with year of data collection for life expectancy being the linear control. Labor productivity is significant (p-value of 0.000). See Appendix B for data sources.

[21] For example, Saudi Arabia's GDP per worker in 1980 is 170,908 PPP$, compared to 51,366 for the US. We seriously doubt GDP per worker of Saudi Arabia (and similar countries in the region) is a good proxy for average wages - it rather reflects oil revenues - which is why we chose to prune the OPEC oil producing countries from our sample.

Figure 6 shows the results for 1980 (Panel A) and 2000 (Panel B). In comparison to the original Preston curve the estimated functions are somewhat more linear to behold. This result is likely caused in part by the fact that we are ignoring child mortality, and in part by the fact that data is missing in many of the poorest countries. Nevertheless, the results do suggest a flattening of the income gradient at high levels of GDP per worker. Moreover, by observing the range in life expectancy in the two years it becomes clear that the 2000 curve has shifted up compared to its opposite number in 1980, though the increase is most evident for rich and middle income countries.

In order to apply our model we indexed the level of w (i.e., GDP per worker) for the US to 100, and simulated life expectancies associated with the empirically relevant range of labor productivity levels. Figure 7 compares the model's predictions regarding life expectancy in 2000, and the empirically estimated income gradient from the cross section in 2000. As can be seen,

FIGURE 7: THE MODIFIED PRESTON CURVE



the model underestimates life expectancy among the rich countries. Among the poor the concordance between the model and the empirical Preston curve is much better, although the model slightly overestimates longevity. This was to be expected. In this simulation we are keeping (among other things) the level of health technology fixed, and it seems reasonable to assume A is higher in the US than in many poor countries. The fact that the model underestimates longevity among the richest nations is perhaps more surprising. But by the same logic a potentially viable explanation is that the level of efficiency in health investments is somewhat lower in the US than in certain other rich and middle income countries. At least, this is the most straightforward

explanation for the discrepancy. It is worth bearing in mind that A likely captures more than pure medical technology, which undoubtedly is second to none in the US. Strictly speaking, A is the level of efficiency by which health investments are translated into reductions in the speed of aging. As a result, institutional inefficiencies in the health care sector will influence the size of A, by affecting the mapping between expenditure (input) and longevity (output). The present analysis cannot pinpoint the reason why A apparently is lower in the US than in other OECD countries. But it remains an observable fact that the US constitutes an "outlier" only in health expenditures, not in life expectancy.

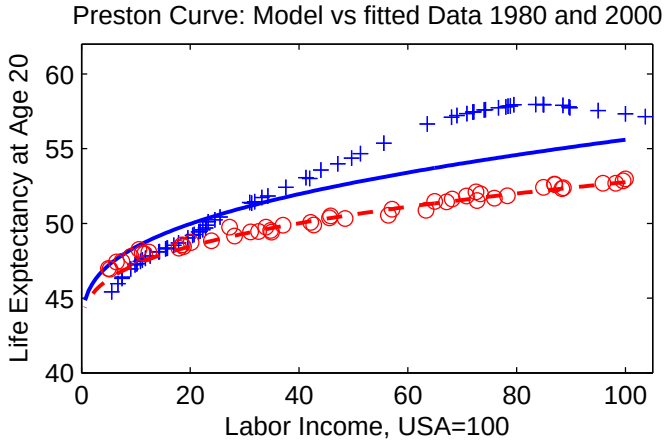
Admittedly, the fit of the model is not perfect. Still, considering the simplicity of the underlying framework it does a remarkably good job at matching the data. In particular, the model captures very accurately the range in life expectancy found in the data. This suggests that differences in health investments, prompted by differences in income, go a long way towards motivating the Preston curve. By way of contrast, in order to account for the observed income gradient there is only a limited need to appeal to differences in relative prices of health care (although they undoubtedly exits) as well as cross-country differences in A.

The next exercise is to examine whether we can match the shift in the (modified) Preston curve between 1980 and 2000. Here we proceeded as follows. According to the theory there are two forces which plausibly could cause the Preston curve to shift over time (aside from income). One is technology A, and the other is changes in the relative price of health investments p.

In an effort to take price changes into account, we calculated the difference between the rate of increase in the CPI for medical care and the overall CPI for the US, using data from the Bureau of Labor Statistics. From 1980 to 2000 this comes to a rate of 2.08 percent per year, implying that the relative price of health investments went up during the period in question. While the rate of change in p likely exhibits country variation, we stay with the basic approach and assume this change occurred in *all* countries worldwide. Hence, when simulating the Preston curve for 1980 we *lowered* the level of p compared to its value in 2000 (where it was normalized to 1). This means that p for the 1980 simulation is 0.66, rather than one. If this was the only change, the model would predict a Preston curve in 1980 which lies above the corresponding curve for 2000.

The difference between the simulated curve and the actual curve (see Figure 6) must be – according to the theory – attributable to A.[22] Thus, we finally adjust the level of A in the 1980 simulation (downward) so that we match the 1980 Preston curve as well as possible. This provides us with a level of health productivity in 1980, which we can compare to the number calibrated to the US data for 2000. The end result is depicted in Figure 8. With an appropriate

FIGURE 8: THE MODIFIED PRESTON CURVE 1980 VS 2000

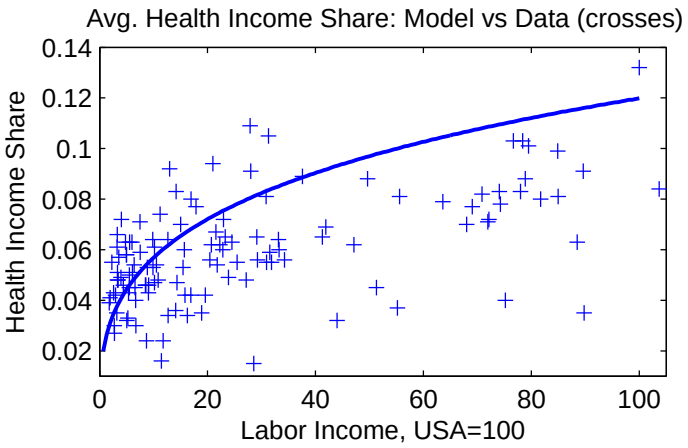


adjustment of A we can match the empirical income gradient in 1980 strikingly well; the model's prediction and the nonlinear mapping from the data almost coincide. The required drop in A, going from 2000 to 1980, is 20%, which is equivalent to an annual rate of growth in A of 1.12 percent. The only estimates which speaks to "aggregate" A, which can be compared to our calibration (that we are aware of), is the work of Fonseca et al. (2009), who find a rate of technological change of 1.8% per year from 1965-2005. Our estimate is thus perhaps on the low side, yet it remains in the ball park.

As a final consistency check we examined our model's predictions regarding spending shares across countries. As we are changing the level of income, the share of health spending changes too. Are these changes of plausible magnitudes, compared to the cross-country data? Figure 9 shows the model's prediction (i.e., the *average* spending shares across life) versus cross country data for 125 countries for which data on health expenditures as a fraction of GDP was available for 2000. Interestingly, while the model tends to underestimate longevity in the richest places on the planet, it overestimates the health shares. Once again this may be due to the calibration

[22]While the *level* of A depends on institutions, it seems reasonable to expect that most of the observed *change* in A is attributable to technological progress.

FIGURE 9: EXPENDITURE SHARES: MODEL VS DATA



of the level of productivity to that of US; if the US indeed is less efficient with respect to health investment than many of the similarly rich nations (i.e., the US tends to get less longevity per dollar than other countries) it is natural that we overestimate the expenditure shares when applying the US level of A. There is also a lot of "noise" around the model-line at lower levels of income. For example, countries like Zimbabwe and Lesotho spend a large fraction of their GDP on health care, given their level of income: 8.3% and 6.2%, respectively, in 2000. It seems possible that the HIV epidemic might be part of the explanation, which our calibrated model obviously cannot account for.[23]

Still, overall, the model produces spending shares for health that are broadly consistent with the empirical data; the predicted range for the health shares is fairly accurately captured, and the positive concave association suggested by the model is consistent with the data. Hence, the underlying intermediate step that connects income and longevity, as reflected in the Preston curve, is also captured rather well by the model.

In sum, this calibration suggests that the Preston curve can be quantitatively motivated by a theory of optimal health investments, and that the movements in the curve between 1980 and 2000 can be accounted for by technological progress in health investments, at a rate of about 1.1% per year.

The reasonable fit of the model leads to some important conclusions. People in the poorest countries are dying earlier than citizen's in rich nations. In order to explain this cross-country

[23]In both Zimbabwe and Lesotho the epidemic is rampant; in 2004 23.3 % of the 15–49 year-olds were HIV positive in Lesotho; in Zimbabwe the number was only slightly lower in 2004, at 18.1% (World Health Organization, 2009).

variation there appears to be little need to resort to explanations involving lack of technological know-how, different relative prices on health care, low levels of human capital, inefficiencies in health care etc. To a first approximation the sad reality seems to be that poor people spend less on health because they are poor and live shorter lives because of it. These conclusions mimic Preston's (1975) own conjecture regarding the underlying forces that generate his curve rather well. That is, the nonlinear link between income and life expectancy is to a large extent caused by lower health investments in several dimensions, ranging from clean water to medical treatment. Changes in relative prices and health technologies do matter. But in order to understand the income gradient, they do not seem to be the main culprits.

At the same time the model fit is certainly not perfect. How could it be, considering its stylized nature. As a result, there is ample scope for many other factors, beyond income, to be contributing to global life expectancy inequalities. It is also worth bearing in mind that our analysis does not concern infant mortality. This could be an important extension of the framework, which undoubtedly would require careful theorizing about nutritional intake and more, which is beyond the scope of the present paper.

6. Concluding Remarks

In the present paper we have proposed a theory of optimal aging and death. Individuals maximize lifetime utility subject to the usual budget constraint, but also taking their physiology into account. The physiological constraint concerns the gradual emergence of health deficits that constitute the aging process. While aging and death are inevitable, individuals can invest in their health which serves to slow down aging and prolongs life. Contingent on preferred health investments the aging process as well as the time of death are determined.

The model holds strong predictions regarding optimal health spending across the life cycle, as well as for the optimal evolution of health expenditure shares. Interestingly, it is not necessarily optimal for health spending, or its expenditure share, to rise during life. The analysis has worked out under what circumstances it is optimal for spending to be rising, constant, or declining during life.

The calibrated model is able to predict spending shares and frailty across age groups, in the US, fairly well. Encouraged by these findings we used the calibrated model to elicit information about the dynamic impact on frailty and longevity from shocks to technology, income and

more. One interesting outcome of the simulations were that changes in relative health prices and income have relatively modest effects on longevity. By way of contrast, the impact on longevity from changes in health productivity is much larger. This suggests that governments aiming to improve health outcomes might be better off focusing on health technology, and on the institutional set-up of the health sector, than focusing on (say) subsidizing prices on health investments.

In a cross-country setting the calibrated model is able to account well for the celebrated Preston curve (Preston, 1975). This suggests that most of the observed link between income and longevity across countries can be attributed to variations in health investments. To a first approximation we can match the income gradient in the data rather well without appealing to differences in medical technology, health prices etc. Technological change (and price changes) *do* matter though; in fact, according to our calibrations they are more or less fully responsible for the change in the position of the Preston curve between 1980 and 2000. These findings largely corroborate Preston's (1975) own interpretations of his empirical findings. By extension, they therefore also support his conclusion that the impact from technological innovations are going to be a much more important force for rising longevity in the future than economic growth in income.

We believe the framework developed above may form the basis for further research in various directions. From our simulations it is clear that small variations in the rate of time preference hold a strong impact on longevity. The same is true for the intertemporal rate of substitution. This could suggest that differences across countries in cultural values with regards to e.g. "thrift" might explain why some societies invest more in health, and consequently are inhabited by longer-lived citizens, which could be an interesting hypothesis to explore empirically. Similarly, the model predict that physiological differences across country populations should contribute to an explanation for observed differences in longevity, in as much as these physiological differences influence the force of mortality. Work has already been done, which suggest that cross-country genetic differences holds considerable explanatory power vis-a-vis longevity (Galor and Moav, 2007), but there is probably even more to be learned in this area.

While the model does a good job at matching the cross-country data on income and longevity, it remains rather stylized. Hence, there are a number of natural theoretical extensions of the basic model that seem worth pursuing. One extension would concern labor supply; optimal

retirement more specifically. It seems very plausible that the force of aging, and its interaction with health investments, will influence the choice of when to retire from the labor market. Similarly, aging should influence the costs of on-the-job skill accumulation, which ultimately may contribute with a physiological explanation for the hump-shaped path for wages across the life-cycle that is observed empirically.

Appendix A: Derivations

Derivation of (6) and (7). The Hamiltonian associated with the problem of maximizing (4) subject to (2) and (5) reads

$$H = \frac{c^{1-\sigma}-1}{1-\sigma} + \lambda\left(\mu D - a - Ah^{\gamma}\right) + \phi\left(rk + w - c - ph\right).$$

For $\sigma = 1$ the first term is replaced by $\log(c)$. The first order conditions wrt. c and h and the co-state equations are

$$c^{-\sigma} - \phi = 0 \qquad \Rightarrow \qquad c^{-\sigma} = \phi \qquad \Rightarrow \qquad \sigma\dot{c}/c = -\dot{\phi}/\phi \tag{10}$$

$$-\lambda A\gamma h^{\gamma-1} - p\phi = 0 \tag{11}$$

$$\lambda\mu = \lambda\rho - \dot{\lambda} \qquad \Rightarrow \qquad \mu - \rho = -\dot{\lambda}/\lambda \tag{12}$$

$$\phi r = \phi\rho - \dot{\phi} \qquad \Rightarrow \qquad r - \rho = -\dot{\phi}/\phi. \tag{13}$$

Equation (13) is the well known Euler equation requiring that the shadow price of consumption (ϕ) grows at the rate of the interest rate less the time preference rate. Analogously, the Euler equation (12) requires that the shadow price of health grows at the rate of health deterioration (μ) less the time preference rate.

Log-differentiate (2) wrt. time and insert (12) and (13) to obtain optimal growth of health expenditure:

$$\frac{\dot{\lambda}}{\lambda} - \frac{\dot{\phi}}{\phi} = (1-\gamma)\frac{\dot{h}}{h} \qquad \Rightarrow \qquad -\mu + \rho + r - \rho = (1-\gamma)\frac{\dot{h}}{h}.$$

Observe that ρ cancels out. Intuitively, the growth rate of health expenditure depends positively on the growth rate of the the shadow price differential, i.e. the growth rate of λ/ϕ. If the shadow price of health (λ) grows at higher rate than the shadow price of consumption (ϕ), it indicates that the future contribution of health to utility is more important than the future contribution of consumption (both measured relative to current contribution) and thus it is optimal that health expenditure increases over time ($\dot{h}/h$ is positive). Since the time preference enters both equations symmetrically, it has no significance for the *growth* of health expenditure. Of course it will affect the level of health expenditure, see main text. Since r enters only the growth rate of the shadow price of consumption and μ enters only the growth rate of the shadow price of health, they do not drop out but affect growth of health expenditure with opposite sign. Specifically solving for the growth rate of expenditure we get the "Health Euler", i.e. equation (7) of the

main text. As usual, (6) is obtained by inserting (12) into (10). Note also, from (2), that the shadow price of health ϕ is negative because the associated stock variable D is a "bad" rather than a good.

Derivation of (9a)-(9c). Begin with noting that, because g_h is optimally constant according to (7), the differential equation (2) can be rewritten as

$$\dot{D} = \mu D - a - Ah(0)^{\gamma} \exp(\gamma g_h t).$$

Given $D(0) = D_0$ the solution at time T is:

$$D(T) = D_0 \exp(\mu T) - Ah(0)^{\gamma} \exp(\mu T) \int_0^T \exp(\gamma g_h - \mu) \mathrm{d}t - a \exp(\mu T) \int_0^T \exp(-\mu t) \mathrm{d}t.$$

At the time of expiry the boundary condition requires $D(T) = \bar{D}$. Solve the integrals in the above equation to get (9a) in the text.

Next, integrate (5) and insert $k(0) = k_0$ and $k(T) = \bar{k}$ to obtain

$$\begin{aligned} \bar{k} = {} & k_0 \exp(rT) + w \exp(rT) \int_0^T \exp(-rT) \mathrm{d}t \\ & - c(0) \exp(rT) \int_0^T \exp[(g_c - r)T] \mathrm{d}t - ph(0) \exp(rT) \int_0^T \exp[(g_h - r)T] \mathrm{d}t. \end{aligned}$$

Divide by $\exp(rT)$. Note that $g_h - r = (\gamma r - \mu)/(1 - \gamma) \equiv g_D$ and solve the integrals to obtain (9b) in the text.

Finally note that optimal expiry requires that the Hamiltonian assumes the value of zero at T. Otherwise, it would have been optimal to live longer or die earlier. Also, at expiry $D(T) = \bar{D}$ and $k(T) = \bar{k}$. Thus the Hamiltonian reads

$$0 = H(T) = u(c(T)) + \lambda(T) \left[\mu \bar{D} - a - Ah(T)^{\gamma}\right] + \phi(T) \left[r\bar{k} + w - c(T) - ph(T)\right].$$

Insert $\lambda(T)$ and $\phi(T)$ from (10) and (11) to get

$$0 = u(c(T)) - \frac{p}{c(T)^{\sigma}} \left[\frac{(\mu D - a)h(T)^{1-\gamma}}{\gamma A} - \frac{h(T)}{\gamma} - \frac{w + r\bar{k}}{p} + c(T) + h(T)\right]$$

where $u_T \equiv \log(c(T))$ in the case of log-utility and $u_T \equiv [c(T) - 1]^{1-\sigma}/(1 - \sigma)$ otherwise. Noting that $c(T) = c(0) \exp(g_c T)$ and $h(T) = h(0) \exp(g_h T)$ this yields (9c) in the text. Observe that the date of expiry T is optimized via the the boundary condition $H(T) = 0$ rather than via a first order condition for T as in the related literature (see Hall and Jones, 2007 for a detailed

discussion). This implies that utility in the above equation is allowed to be negative. The boundary value approach does not need a constant that turns utility positive for all c. Essential here is that higher $c(T)$ increases $u(c_T))$ and thus $H(T)$ no matter what the absolute value of utility is.

Appendix B: Data sources

Data on life expectancy at age 20. We use two samples of data on life expectancy at the age of 20 for males: A "2000" sample and a "1980" sample. The former sample involves observations covering the period 1997–2006. In the 1980 sample we included observations if they were available sometime during the period 1970–1990; in the event several years were available we chose the data point closest to 1980. The data is available in the Demographic yearbook for 2006 (2000 sample) and in the historical supplement to the Yearbook for 1997 (1980 sample). Alternatively, the data can be obtained online at the web address:

http://unstats.un.org/unsd/demographic/sconcerns/mortality/mort2.htm

In our sample, used for estimation, the median year of observation for the "1980" sample, is 1979, with a standard deviation of 4 years. For the "2000" sample the median year of observation is 2003 with a standard deviation of 2.73 years.

Data on Labor productivity. In the regressions we employ GDP per worker (RGDPW) for 1980 and 2000, from Penn World Tables, Mark 6.3.

Data on "p". For the calibration involving shifts in the Preston curve we adjust for plausible changes in p during the period 1980–2000. We calculated the relative price as the ratio between the CPI for "Medical care" and the "all items" CPI. In this regard we obtained data from the Bureau of Labor Statistics, Consumer Price Index Detailed Report Tables; Annual Average Indexes 2000 (Tables 1A-23A), which are available from:

http://www.bls.gov/cpi/cpid00av.pdf

Data on the share of health expenditure in GDP. We obtained data on health expenditures as a share of GDP in 2000, from the World Health Organization. The data is available for free at:

http://apps.who.int/ghodata/

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