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Accounting for Fetal Origins: Health Capital vs. Health Deficits

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Abstract. The Fetal Origins hypothesis has received considerable empirical support, both within epidemiology and economics. The present study compares the ability of two rival theoretical frameworks in accounting for the kind of path dependence implied by the Fetal Origins Hypothesis. We argue that while the health capital model due to Grossman (*Journal of Political Economy*, 80(2), 223-255, 1972) is irreconcilable with Fetal Origins of late-in-life health outcomes, the more recent health deficit model due to Dalgaard and Strulik (*Journal of the European Economic Association*, 12(3), 672-701, 2014) can generate shock amplification consistent with the hypothesis.

Keywords: Fetal Origins; Health Capital; Health Deficits

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1. Introduction

Half a century ago epidemiologists would tend to view the fetal state as a protected one. Since then epidemiological evidence has been accumulating that this appears not to be the case, which has spawned the fetal origins hypothesis. The fetal origins hypothesis suggests that morbidities *in utero* may cause epigenetic changes in the fetus that instigate morbidities late-in-life though without being directly visible for most of the life course (e.g., Almond and Currie, 2011).

Within economics, research has considerably strengthened the case that *in utero* (or early-in-life) shocks indeed appear to impact on late-in-life health (e.g., Almond, 2006; Van den Berg et al., 2006; Almond and Mazumder, 2011; Lin and Lui, 2014; Bhalotra and Rawlings, 2011). In addition, research has demonstrated effects beyond late-in-life health, including human capital and labor market outcomes (e.g. Bleakley, 2007; Almond et al., 2009; Nelson, 2010; Bhalotra and Venkataramani, 2016); welfare dependence (Almond, 2006; Oreopoulos et al., 2008), and even investment behavior (Cronqvist et al., 2016). From a broader perspective, the fetal origins hypothesis thus seems to be a promising avenue through which to gain further insights into the causes, and intergenerational transmission, of inequality (e.g., Currie, 2011).

From a theoretical perspective, however, the fetal origins hypothesis poses a problem for the current workhorse model within health economics: the Grossman (1972) model. At the heart of the model lies the concept of "health capital", which is a stock that depreciates but can be augmented by health investments analogous to a stock of physical capital. Herein lies the key problem: The fact that health depreciation is assumed proportional to the stock of health implies that the late-in-life health stock becomes largely unaffected by initial conditions, such as those prompted by *in utero* shocks, as the initial conditions "depreciate away" over the life course. The problem is further aggravated by the need to assume an accelerated rate of health depreciation with the passing of time in order for the Grossman model to account for mortality.[1]

These difficulties are avoided, however, in the framework developed by Dalgaard and Strulik (2014). Based on research in the natural sciences the process of aging is conceptualized as a gradual loss of redundancy in the human body, which causes increasing frailty and ultimately death. This conceptualization of aging is anchored in the biological literature, and can be

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[1]Assuming that the depreciation rate is age-dependent has other counterfactual implications. 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, and not the year on the birth certificate, is what matters to health investments.

given strong micro-foundations, as discussed below. Moreover, it leads to a law of motion for human frailty, which depends on physiological parameters and health expenditures. While the process of increasing frailty, measured by health deficits, is accelerating with age it may be slowed by health investments. In the context of the issue at hand the theory implies that health deficits accumulate exponentially over the life course, a prediction that has been repeatedly verified in research within gerontology, which means that small differences in initial conditions between individuals are *amplified* with the passing of time. As a consequence, this framework is well positioned to explain life course dynamics associated with the fetal origins hypothesis (and shocks in early childhood) in the context of long-run health outcomes as well as other socio-economic outcomes. Indeed, the basic model has been adapted to the study of the link between aging and human capital accumulation (Strulik, 2016); years in retirement (Dalgaard and Strulik, 2017); the gender-gap in mortality (Schünemann et al., 2017a) and the health gap between married and unmarried individuals (Schünemann et al., 2017b).

The paper proceeds as follows. In the next section we compare basic versions of the Grossman model and the Dalgaard-Strulik model in their ability to account for the fetal origins hypothesis. In Section 3 we provide a fuller discussion of the impact of initial conditions on lifetime health outcomes within the health deficits model where investments are optimally determined. Subsequently, in Section 4, we lay out micro-foundations behind the process of increasing frailty as adapted in the health deficit framework. Section 5 concludes.

2. Basic Models

2.1. **Health Capital Accumulation.** The survey by Almond and Currie (2011) provides an illustration of the inability of the Grossman (1972) model to account for fetal origins. The illustration has the following law of motion for health capital as main ingredient:

$$H_t = (1 - \delta)H_{t-1} + I_t, \ H_0 \text{ given}, \ H > \underline{H} \tag{1}$$

in which H is the stock of health capital, δ is a constant rate of health capital depreciation, I represent health investment and $\underline{H}$ is a hypothesized lower boundary for health beyond which individuals expire. Repeated substitution leaves us with the following expression for the stock of health at time t:

$$H_t = (1 - \delta)^t H_0 + \sum_{i=0}^{t-1} (1 - \delta)^i I_{t-(i+1)}.$$

The key observation to make is that shocks *in utero* that influence initial health, H_0, depreciate away with the passing of time. In general, events in the past are far less important to current health than recent events. This is an inevitable consequence of the basic assumption in the Grossman model that health depreciates in proportion to the stock of health. In principle, the model therefore imposes that healthy individuals age faster than unhealthy (or elderly) individuals, ceteris paribus. Consequently, initial conditions will be of little consequence later in life.

The panel on the left hand side of Figure 1 provides some numerical illustrations of this point, replicating Figure 1 of Almond and Currie (2011). It shows how an initial shock, which creates a 25 percent deviation in initial health to a reference individual, depreciates with age for three different rates of health capital depreciation. At five percent depreciation the initial 25 percent deviation is melted down to about a 5 percent deviation at age 30. At 15 percent, initial differences are basically equalized at age 30.[2]

FIGURE 1: SHOCK PERSISTENCE BY AGE: HEALTH CAPITAL VS. HEALTH DEFICITS ACCUMULATION
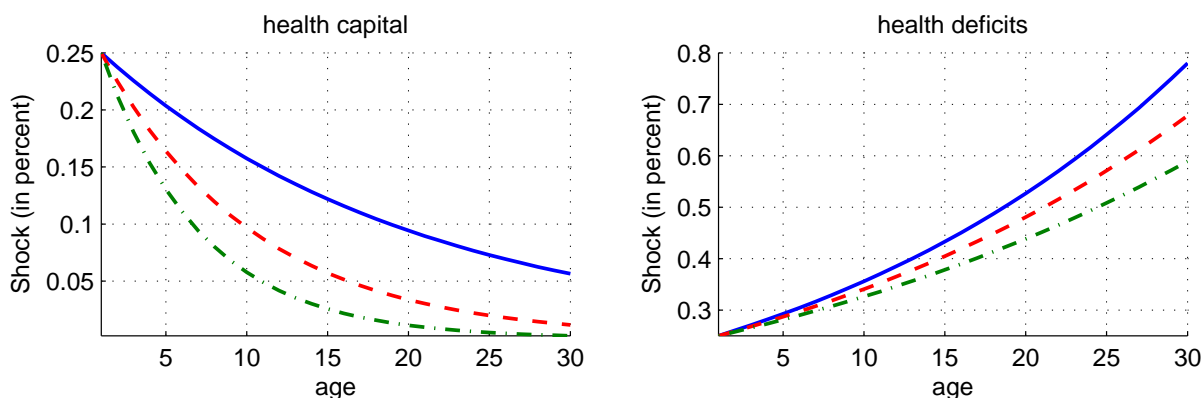
The Figures show how persistent a 25 percent negative shock to the birth endowment would be given alternative annual depreciation rates. Left: blue (solid) line: 5 percent depreciation; red (dashed) line: 10% depreciation; green (dash-dotted): 15% depreciation. Right: blue (solid) line: $\mu = 0.04$; red (dashed): $\mu = 0.035$; green (dash-dotted): $\mu = 0.03$ ($E = 0.02$ and $D_0 = 0.02$).

As it turns out, the Grossman model actually holds a stronger prediction than what is indicated by the experiment conducted in Figure 1. Observe that the absolute difference in health capital between two individuals ($i = 1, 2$ respectively) with different initial conditions (i.e.,

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[2]More formally, Figure 1 shows the impact on the long run relative level of health of two individuals (1 and 2, say) after one is hit by a shock in utero (time zero):

$$d\left(H_t^1/H_t^2\right) = (1 - \delta)^t \, dH_0^1$$

different H_0), in the absence of health investments, is given by

$$H_t^1 - H_t^2 = (1 - \delta)^t \left(H_0^1 - H_0^2 \right).$$

Hence, the Grossman model implies a stronger version of non-persistence than convergence in relative health levels: Namely, *absolute* convergence in health levels between individuals with different initial conditions, holding investments fixed.

So far we have assumed a constant rate of health depreciation, δ. Naturally, in the Grossman (1972) model the depreciation rate is not constant, as it theoretically would enable individuals to "live forever" contingent on sufficient capital investments.[3] Instead the depreciation rate is assumed to increase with the passing of time as the individual ages. Obviously, this only serves to strengthen the prediction that initial health shocks loose significance with the passing of time. In this case depreciation of initial health differences become faster than geometric.

The exercise conduced in Figure 1 and subsequent discussion sets health investments to zero. This may seem to open the door to a simple way of reconciling the Grossman model with fetal origins, namely through investments. However, as pointed out by Almond and Currie (2011, p. 158) in the context of the illustration depicted in Figure 1:

> If investments in all periods subsequent to the shock are affected by the shock, then prenatal exposures could be important for adult health in the Grossman (1972) framework. However, the fetal origins literature posits an important and persistent biological effect of the prenatal period – that is, holding investments fixed.

It is important to appreciate that fetal origins, from the point of view of the medical literature, involves a specific mechanism. The early literature spoke of how environmental shocks would "program" the fetus with a predisposition towards various diseases, like coronary heart disease (e.g., Barker, 1995). Today a widespread view is that early-in-life shocks affect late-in-life health outcomes due to epigenetic changes. That is, changes in hereditary traits brought on by environmental influence (e.g., Gluckman and Hanson, 2004; Wu et al., 2004; Hilakivi-Clark and De Assis, 2006; Dolinoy el al. 2007; Waterland and Michels, 2007; Sinclair et al, 2007; Thompson and Einstein, 2010). Animal trials have been instrumental in providing proof of the principle of the fetal origin's hypothesis (McMullen and Mostyn, 2009, for a review). Accordingly, in order

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[3]See e.g. Grossman (1972), section III.

to fully account for the fetal origins hypothesis a theory would have to allow for an influence from initial conditions on long run health outcomes *conditional* on investments, for purely biological reasons. The Grossman model does not allow for such a line of influence, as seen above.

Finally, it should be observed that while the standard Grossman model does not suggest that initial conditions influence subsequent investments, the more recent work by Heckman (2007) does.[4] The theory of human capability formation creates dynamic complementarities by assuming that health investments happens at two (or more) distinct periods in life such that health outcomes are produced with the distinct health investments as inputs. Since negative early-in-life shocks, or low initial investments, reduce the productivity of future investments, early-in-life events can have very persistent effects.[5] When these ideas are introduced into the Grossman model the resulting framework can generate persistence, which is broadly consistent with the fetal origins hypothesis. As should be clear, however, initial conditions only influences eventual outcomes *via* investments. As a consequence, the "Grossman-Heckman" framework cannot account for an impact of initial conditions on late-in-life outcomes holding investments fixed, and by extension it cannot account for the fetal origins hypothesis as it is conventionally understood in the medical literature. Combining the "Heckman mechanism" with the deficit model would not have this drawback, as will be clear from the discussion to follow.

2.2. **Health Deficit Accumulation.** The health deficit model of Dalgaard and Strulik (2014) can in its simplest form be written

$$D_t - D_{t-1} = \mu(D_{t-1} - E), \text{ for } D < \bar{D},\ D_0 \text{ given}, \tag{2}$$

where D denotes health deficits. D is measured as the relative number of health deficits that a person has out of a long list of potential health deficits. Accordingly, the index is defined on a 0 to 1 scale, and aging (declining health status) occurs as the index gradually traverses towards one. In general, individuals with a higher deficit index are to be considered more frail, and thus physiologically older. In practise the process of deficit accumulation continues until an upper boundary for deficits, $\bar{D}$, is reached at which point the individual expires. The parameter μ is the "natural" rate of aging, and E is an "environmental constant". Equation (2) derives from

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[4]See Almond et al. (2017) for a discussion.

[5]This end result is not a given, of course. At presently, the evidence in favor of dynamic complementarities seem to be largely descriptive in nature. Moreover, while some studies do find that parental investment appear to reinforce shocks, implying persistence in early-in-life shocks through investments, other studies find that parents act in a compensatory fashion. See Almond and Mazumder (2013) for a recent review.

the literature on gerontology and the underlying parameters have been estimated with great precision (see Dalgaard and Strulik, 2014, Section 2). For example, empirical estimates suggest that μ is between 0.03 and 0.045, depending on gender and country of origin (Mitnitski et al. 2002a).[6] By extension, it is worth noting that in contrast to the Grossman model where the object of interest – health capital – is an unobserved variable, the object of interest in the present model – health deficits – is empirically observable. Section 4 offers some micro-foundations for equation (2).

Repeated substitution leads to

$$D_t = (1 + \mu)^t D_0 - \sum_{i=0}^{t-1} \mu^{1+i} (1 + \mu)^i E_{t-(i+1)}$$

Hence, an inherent feature of the deficit model is that early-in-life shocks that influence the initial relative number of deficits, D_0, are amplified over time. This creates a force of divergence: initially unhealthier individuals accumulate health deficits faster than initially healthy individuals.

The panel on the right hand side of Figure 1 provides a numerical illustration of this feature. As in the previous section we study the impact a health shock *in utero* that creates a 25% deviation in initial deficits relative to a reference individual. The deviation from benchmark increases over time. For $\mu = 0.04$, the initial 25% deviation has reached 80% percent at the age of 30.[7]

As noted above, the interpretation of E in the natural science literature is that of "environmental" influences. While some such influence can be external to individuals (such as pollution), E may also be influenced by deliberate health investments. By increasing E such investments will serve to slow down the process of deficit accumulation and thus provide the prospect of a longer life. In the illustration in Figure 1, the level of E is ignored so as to provide a clean comparison with the properties of the Grossman model in the absence of health investments. Nevertheless it is also of interest to understand the consequences of allowing for optimal health

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[6]The exponential nature of health deficit accumulation as been confirms in a varieties of studies for samples from different populations, see e.g. Shi et al., 2011; Harttgen et al., 2013; Mitnitski and Rockwood, 2013, 2016; Abelianski and Strulik, 2017.

[7]More formally, Figure 1 shows the impact on the long run relative level of health deficits of two individuals (1 and 2, say) after one is hit by a shock in utero (time zero):

$$d\left(D_t^1/D_t^2\right) = (1 + \mu)^t dD_0^1$$

investments in the presence of shocks to initial deficits within the deficit model. The next section therefore studies the impact from initial deficits in the original health deficit model.

As a final remark on the properties of the basic deficit model its worth observing that it also holds radically different implications from the Grossman model in terms of the evolution of *absolute* health differences. Comparing the absolute difference in health deficits between two individuals ($i = 1, 2$ respectively) with different initial conditions (i.e., different D_0), in the absence of health investments ($E = 0$), is given by:

$$D_t^1 - D_t^2 = (1 + \mu)^t \left(D_0^1 - D_0^2 \right).$$

Hence, initial differences in health deficits are amplified and the model thus predicts absolute divergence in health holding investments fixed.

3. Shock Persistence in the Health Deficit Model

We begin by rewriting (2) for a continuous notion of age and separating E into a "real" environmental constant a and the impact of health investment on health deficit accumulation:

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

Here, 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 deteriorated human bodies. Bad health promotes death such that individuals die when $\bar{D}$ health deficits have been accumulated.

Individuals are interested only in maximizing their life time utility from consumption:

$$\int_\tau^T e^{-\rho(t-\tau)} u(c(t)) \ dt \tag{4}$$

with $u(c) = (c^{1-\sigma} - 1)/(1 - \sigma) + b$ for $\sigma \neq 1$ and $u(c) = \log(c) + b$ for $\sigma = 1$. Here σ is the inverse of the elasticity of intertemporal substitution and ρ is the rate of time preference. Allowing for death to be a stochastic event and considering health as an element in the utility function leads to some further interesting results but does not change the basic insight on the impact of initial health deficits (see Strulik, 2015a,b). We thus focus on the simpler model here.

Besides spending income on final goods, individuals may save or borrow at a net interest rate r. Individuals take all prices as exogenously given. The law of motion for individual wealth k is thus given by (5).

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

in which w is the (annual) wage, r is the interest rate, and p is the price of health goods. The problem is to maximize (4) subject to the accumulation equations (3) and (5), the initial conditions $D(\tau) = D_\tau$, $k(\tau) = k_\tau$, and the terminal conditions $k(T) = \bar{k}$, $D(T) = \bar{D}$. At the very basic level the problem is to trade-off the benefits and costs of health investments over the life cycle. The benefits consists in, by slowing down the process of aging, a longer life which allows for more consumption along the extensive margin. However, by increasing health investments, individuals forego consumption in the current period. See Dalgaard and Strulik (2014) for details on the solution of this free terminal value problem.

We take the calibration of the model for an average 20 years old male U.S. American in the year 2000 from Dalgaard and Strulik (2014). This means that we set the rate of aging μ to 0.043, the interest rate to 6 percent and γ to 0.19 such that health expenditure increases at an annual rate of 2 percent over the life cycle. We set $D(0) = 0.0274$ as the relevant initial value for a 20 year old and $\bar{D} = 0.1005$ 55.2 years later since the life-expectancy of a 20 year old U.S. American in the year 2000 was 55.2 years. We set $a = 0.013$ such that the model predicts a life-expectancy at age 20 of 42 years (life expectancy in the 19th century when adult life expectancy was only modestly affected by medical technology). Moreover we set the benchmark values $\rho = r$, $\sigma = 1$, $p = 1$, and $b = 0$. Finally we set the annual labor income w to 77,0035 and estimate $A = 0.00139$ such that the individual dies with deficits $\bar{D}$ at age 75.2, according to the life-expectancy of a 20 years old U.S. Americans in the year 2000.

In Figure 2 we replicate the benchmark run of Dalgaard and Strulik (2014), represented by blue (solid lines). We then look at an individual that is initially 10 percent less healthy than the Reference American, represented by red (dash-dotted) lines, and an individual that is initially 10 percent healthier than the Reference American. These differences in initial health deficits at age 20 can be thought of as resulting from negative health shocks earlier in life (and perhaps *in utero*). In line with observations the model predicts that unhealthier individuals spend more on health (panel on the right hand side). But the calibrated health technology is not powerful enough to equalize initial health differences. In fact initial health differences get amplified over
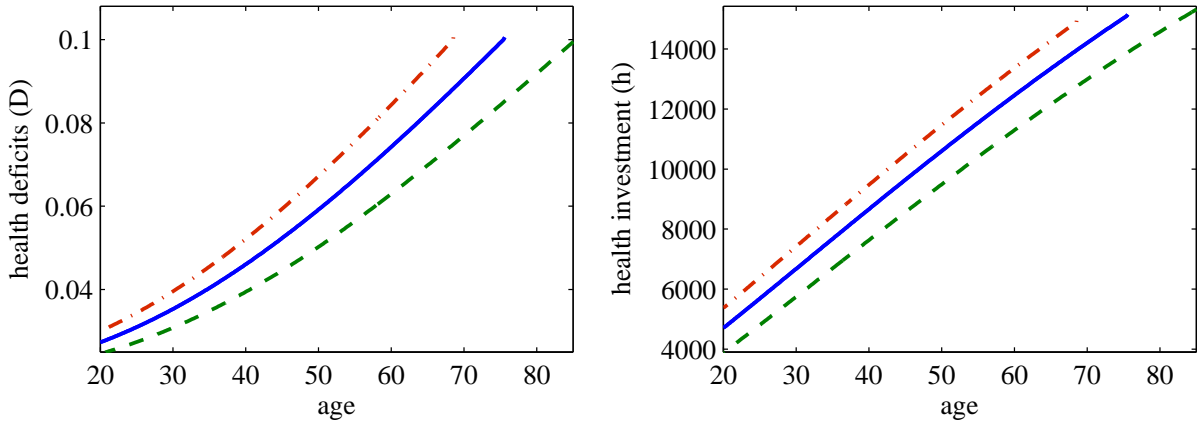
Blue (solid) lines replicate results for the Reference American in Dalgaard and Strulik (2014). Green (dashed) lines: individual with 10 percent less initial health deficits at age 20. Red (dash-dotted) lines: individual with 10 percent more initial health deficits at age 20.

time, i.e. as individuals age, as seen by the fact that the vertical distance between the individuals' deficit trajectories gets larger as they age, see the panel on the left-hand side of Figure 2. The underlying reason for this pattern is that initial deficits influences the effectiveness of health investments: the greater the health deficits the smaller the impact of a given amount of health investments in prolonging life. In this sense the model involves dynamic complementarities akin to those found in the human capability theory (Heckman, 2007).

3.1. **Adding a Childhood Period.** As is evident the above model tracks the evolution of deficits, health and consumption over the life cycle, starting from about the age of 20. Hence, in a strict sense, the analysis does not fully capture fetal origins. While initial shocks *in utero* may create differences in deficits at age 20 it is clearly more satisfactory for the process to start at birth.

Hence we next augment the health deficit model by a childhood period. For that purpose we assume (as e.g., Heckman, 2007) that health investment during childhood is provided by parents and abstain from modeling a parental calculus of child health spending. Instead we introduce health spending of parents in an exogenous, piecewise continuous way. Assuming that the law of motion for health deficit accumulation (3) holds during childhood as well, we obtain health deficits from age s to age τ as:

$$D(\tau) = D(s)\exp(\mu(\tau - s)) - [Ah_s^\gamma + a]\left[\exp(\mu(\tau - s) - 1\right], \tag{6}$$

in which h_s is assumed to be a piecewise continuous function of health expenditure for the age interval $[s, \ \tau]$. From HCCI (2012) we obtain the estimates of average health expenditure per child of \$ 3,426 for ages 0-3, of \$ 1,219 for ages 4-8, of \$ 1,245 for ages 9-13, and of \$ 1,858 for ages 14-18. We assume that the last figure holds for ages 19-20 as well. Given these data points we estimate initial health deficits at birth such that initial health deficits at age 20 are 0.0274, as estimated by Mitnitski et al. (2002a) and as assumed in Dalgaard and Strulik (2014). This leads to the estimate of $D = 0.0224$ at birth.

FIGURE 3: INITIAL HEALTH AND HEALTH DEFICIT ACCUMULATION: INCLUDING CHILDHOOD
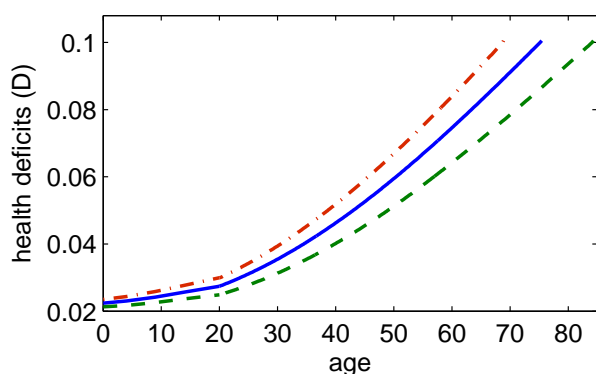
Blue (solid) lines replicate results for the Reference American in Dalgaard and Strulik (2014). Green (dashed) lines: individual with 5 percent less initial health deficits at birth. Red (dash-dotted) lines: individual with 5 percent more initial health deficits at birth.

Figure 3 shows the implied health deficit accumulation for three individuals. The blue (solid) line represents again the Reference American, now extended by a childhood period. The red (dash-dotted) line shows results for an individual with initially (*in utero*) 5% more health deficits and the green (dashed) line represent the outcome for an initially (*in utero*) 5% percent healthier individual. Everything else is kept from the benchmark model. Initial health differences at age 20 are now endogenous and already a bit larger than those at birth. As individuals age, initial differences get more and more amplified (under endogenous optimal health expenditure) and the initially slightly less healthy individual dies 6.7 years earlier than the Reference American. Notice that the model also implies that health shocks (positive or negative) matter more for longevity when they are experienced early in life such that any attempt to repair the initial damage of the disadvantaged child is more powerful (efficient) when it happens earlier in life.

4. Physiological Foundation of Health Deficit Accumulation: Reliability Theory

In this section we adapt Gavrilov and Gavrilova's (1991) micro-foundation of the Gompertz law of mortality to explain the exponential nature of health deficit accumulation. For that purpose we are neglecting environmental factors and health expenditure ($E = 0$), and reformulate health deficits accumulation (2) in continuous time as $D(t) = R\exp(\mu t)$, in which μ is the rate of health deficit accumulation (the force of aging). This means that new health deficits arrive as

$$\dot{D}(t) = \mu R\exp(\mu t). \tag{7}$$

The challenge here is to explain (i) why health deficits arrive at a higher rate as people get older, i.e., why they are aging, and (ii) why health deficits (when unremedied with health expenditure) accumulate in this specific exponential fashion, akin to the Gompertz law of mortality.

A micro-foundation of aging is challenging because the problem cannot simply be delegated to a lower physiological level. Trying to explain aging following a line of reasoning by stating that humans age because their organs (e.g. the cardiovascular system) age, and that organs age because the tissue they are made of ages etc. is not sufficient. At some point a micro-level will be reached that consists of non-aging entities, for example atoms. Eventually, we want to explain why systems age that consists of non-aging elements.[8]

In explaining aging systems biologists built upon a subdiscipline in engineering, reliability theory, which is concerned just with this particular problem. That is, the problem how complicated mechanical systems consisting of non-aging elements (like cars) are increasingly losing function over time so that the failure rate – the probability of the expiry of the system – increases with age (Barlow and Proschan, 1975). Here, the task is to apply the available theory such that it is capable of motivating the exponential nature of health deficits accumulation.

In order to introduce the idea of reliability theory we first consider a simplistic model, according to Gavrilov and Gavrilova (1991). Suppose a human body part (e.g. an organ) is constructed of n non-aging blocks. Non-aging means that the failure rate λ is constant over time. Given age t the probability of a block to fail is $1 - \exp(-\lambda t)$. Blocks are connected in parallel and the organ is functioning as long as at least one block is in order. The probability that the body part becomes defect (loses functionality) before age t is given by $F(t) = [1 - \exp(-\lambda t)]^n$ and the probability

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[8]See Lopez-Otin et al., 2013 and Rockwood et al., 2015 for aging at the cellular level.

of a fully functioning body part at time t is $1 - F(t)$. The unconditional probability of a health deficit at age t is thus given by $f(t) = \mathrm{d}F/\mathrm{d}t = -\lambda n \exp(-\lambda t)[1 - \exp(-\lambda t)]^{n-1}$. Defining a deficit as a defect body part, the expected number of new deficits equal the probability of a body part becoming defect at age t:

$$\dot{D}(t) = -\frac{f(t)}{1 - F(t)} = \frac{\lambda n \exp(-\lambda t)[1 - \exp(-\lambda t]^{n-1}}{1 - [1 - \exp(-\lambda t)]^n}.$$

By the law of large numbers, the probability of a specific body part becoming defect at age t is equal to the share of additional health deficits that occur at age t, which is the increase in the health deficit index $\dot{D}(t)$. For $\lambda t << 1$, we approximate $1 - \exp(-\lambda t) \approx \lambda t$, and the expression simplifies to

$$\dot{D}(t) \approx n\lambda^n t^{n-1}. \tag{8}$$

The simple model is thus capable of explaining aging: the number of additional health deficits acquired at any age t is an increasing function of age.

Reliability theory explains aging as a *loss of redundancy* over time. 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). The problem with the simple model is that health deficits are not accumulated exponentially as in (7). Health deficit follows – similar to the failure rate of mechanical systems – a Weibull law (Gavrilov and Gavrilova, 1991). In order to describe the aging process of humans, the model has to be made "more human" by introducing the probability of initial (fetal) health deficits.

Following again Gavrilov and Gavrilova (1991), we consider next a body part (or organ) consisting of m *irreplaceable* blocks, i.e. blocks connected in series such that the body part becomes defect if one block fails. Each block is further partitioned into n elements, connected in parallel with age-independent failure rate λ. Following the computations from (8) we know that the failure rate of a block is $n\lambda^n t^{n-1}$. Because blocks are connected in series (each of them being essential), the failure rate of the body part equals the sum of failure rates of blocks i.e. $m \cdot n\lambda^n t^{n-1}$.

Next suppose that some elements of the block are initially defect. The probability of an initially functioning element is given by q. The failure rate of a block with i initially functioning elements is thus $\pi = i\lambda^i t^{i-1}$. Blocks, ordered according to their number of initially functioning

elements, are binomially distributed. We approximate the binomial distribution with a Poisson distribution,

$$P(i) = c \cdot \exp(-k) \frac{k^i}{i!},$$

where $k \equiv nq$ is the mean number of initially functioning elements and c is a normalizing constant ensuring that the sum of probabilities equals one. The probability of a specific body part to become defect is computed as the sum of the failure rate of blocks and obtained as

$$\dot{D}(t) = \sum_{i=1}^{n} m P(i) \pi(i) = mc \exp(-k) P(i) = mc \cdot \exp(-k) \sum_{i=1}^{n} \frac{k^i}{i!} \pi(i). \tag{9}$$

Again, by the law of large numbers, the probability of a specific body part becoming defect at age t coincides with the increase in the share of defect body parts at age t, i.e. the increase of the health deficit index.

Inserting $\pi(i)$ in (9) we obtain that the change in the health deficit index as

$$\dot{D} \approx R \sum_{i=1}^{n} \frac{(k\lambda t)^{i-1} \cdot i}{i!} = R \cdot \left[\sum_{i=1}^{\infty} \frac{(k\lambda t)^{i-1}}{(i-1)!} - \sum_{i=n+1}^{\infty} \frac{(k\lambda t)^{i-1}}{(i-1)!} \right].$$

with $R \equiv mc\lambda k \exp(-k)$. Now consider a complex, redundant organ with a large number of elements. In the limit, for $n \to \infty$, the last term in brackets converges to zero. The first term in brackets simplifies to $\exp(k\lambda t)$. We thus obtain $\dot{D} \approx Re^{\mu t}$ with $\mu = k\lambda$. The organism ages according to the exponential law of health deficit accumulation (7).

Empirically there exists a very strong linear association between the frailty index (measuring the relative number of health deficits that a person has) and the force of mortality, with an R^2 above 0.99 (Mitnitski et al., 2002b). The above model explains why. More damages accumulated at the sub-cellular level make health deficits more likely (Mitnitski et al., 2013; Rockwood et al., 2015). Deficits accumulate from the sub-cellular level to the level of organs and death occurs once sufficiently many (severe) deficits have been accumulated (Rockwood et al., 2015). Accordingly, the analysis in the present section suggests that (exponentially) rising health deficits and the (exponentially rising) mortality rate from Gompertz law are mirror images of one another.

5. Conclusion

An influential strand of literature within health economics has over the last decade provided convincing evidence in favor of the fetal origins hypothesis: *in utero* shocks have the ability to

influence late-in-life outcomes. Relevant outcomes involve both health issues that have remained latent through life, as well as a range of socio-economic outcomes.

In this study we have argued that the current workhorse model of health economics, the Grossman (1972) model, is incapable of accounting for such effects. Indeed, since the notion of health – health capital– is analogous to physical capital, the model posits that health status depreciates more when the health status of individuals is high (usually in youth) and less when the health status is low (usually in old age). These features imply, as demonstrated above, that the Grossman model generates the prediction that individuals with different initial conditions, prompted by *in utero* shocks, converge in health status during life holding investments fixed. "Convergence" in health status in the aftermath of early-in-life shocks occur both in a relative and in an absolute sense. This prediction is strengthened if one allows the health depreciation rate to grow over time, as is required for the Grossman model to be reconcilable with the fact of mortality. It is possible to generate important persistence in health outcomes through investments; for example, by introducing the human capability theory of Heckman (2007). But since the fetal origins hypothesis asserts an impact from *in utero* influences *conditional* on investments the Grossman model remains irreconcilable with the hypothesis.

In contrast, the framework developed in Dalgaard and Strulik (2014) offers radically different predictions. At its core the model conceptualizes aging as a continual process of loss of function – increasing frailty – that culminates in death. The notion of frailty is captured by way of the deficit index: as humans age (health declines) the relative fraction of potential age-related health conditions climb steadily upward. This underlying process, which can be slowed down by health investments, is exponential in nature. By implication, small differences in initial conditions are amplified during life. The exponential process can be given micro-foundations, by employing a variant of the biological foundations of mortality due to Gavrilov and Gavrilova (1991), as demonstrated above. Moreover, the exponential nature of increasing deficits during life has been confirmed repeatedly by empirical work within gerontology. Overall, the deficit model seems well positioned to account for the type of dynamics implied by the fetal origins hypothesis.

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