Abstract. In this paper I discuss a standard model of life cycle consumption behavior when the discount rate depends on the state of health and health deteriorates with increasing age. I show that this feature allows the introduction of time-consistent discounting at a non-constant rate and to model, in a convenient way, the notion that individuals discount future payoffs at higher rates when the risk of death increases. I show that the model generates an empirically plausible age-consumption pattern even when perfect annuity markets exist.

Keywords: discount rates, aging, risk of death, consumption behavior.

JEL: D11, D81 D91, I10, I12.
1. Introduction

Economists agree that the standard assumption of intertemporal choice theory that future gains and losses are discounted at a constant rate exists mainly for convenience and several proposals have been discussed to model more realistic discounting behavior (Frederick et al., 2002). In this paper, I focus on one aspect in this domain of research, namely the notion that individuals discount the future at higher rates when they grow older and, in particular, when death is near. I capture this phenomenon by introducing health-dependent discounting and physiological aging into a standard life cycle model. Conceptualizing the discount rate as a function of the state of health, time-consistent solutions of intertemporal choice are easily obtained. As the state of health deteriorates, death becomes more likely, and the pure rate of time preference increases. In order to evaluate their survival probability, individuals consider their physiological age (their state of health) instead of their chronological age. The feature that the state of health is time-variant but pre-determined at any age enables the unconventional result that decisions are time-consistent although the discount rate is not constant.

As a measure of health, I use the health deficit index developed by Mitnitski et al. (2001) "as an individual state variable, reflecting severity of illness and proximity to death." (ibid., p. 323). This measure, also known as frailty index, is an established methodology used by countless studies in gerontology. It has been introduced by Dalgaard and Strulik (2014) into economics (see also Hosseini et al., 2019). The health deficits index computes the number of health deficits present in a person relative to the number of potential health deficits. Health deficits are accumulated in an exponential way as individuals get older (Mitnitski et al., 2002a,b; Abelianzky and Strulik, 2018) and they are a precise predictor of mortality. The prediction of mortality can be so accurate that chronological age adds insignificant explanatory power when added to the regression (Rockwood and Mitnitski, 2007).

A limited number of studies have investigated how aging affects discounting. Huffman et al. (2019) find that, among the elderly, discount rates increase with age. Read and Read (2004) consider individuals from a larger range of ages between 19 and 89 and find the lowest discount rate for individuals of middle age, and thus, a u-shaped age-pattern of discounting. Sozou and Seymour (2002) show that such a u-shaped pattern can be motivated by an evolutionary theory of discounting. Chao et al. (2009) find evidence for a u-shaped association of the discount rate with health deficits and that age loses its predictive power for the discount rate when the state of health is taken into
account. Falk et al. (2019) confirm for a large cross-country data set, comprising 80,000 individuals in 76 countries, that increasing life expectancy as well as better individual perception of health status is associated with higher discount factors (i.e., lower discount rates). A recent study by Gassen et al. (2019) argues in favor of an evolutionary channel from the physical condition of the body to time preference and finds a negative association between inflammatory activity (as a measure of health deficits and cellular distress) and the ability to delay gratification.

I apply the new discounting method to motivate a hump-shaped age-consumption profile in a life cycle model.\textsuperscript{1} The literature has developed several explanations for such a non-monotonous consumption profile (see e.g., Gourinchas and Parker, 2002) and a particularly related proposal is built on age-dependent mortality (Büttler, 2001, Feigenbaum, 2008). This channel, however, breaks down when individuals are allowed to finance old age consumption with annuities. In order to establish health-dependent discounting as an independent pathway, I assume a perfect annuity market to shut off the imperfect-annuities channel. Strulik (2017) shows that the consideration of health in the utility function could also motivate a consumption hump. In contrast to the earlier studies, which were designed to motivate a consumption hump, health-dependent discounting is a more encompassing refinement of preferences that could potentially inspire a host of other applications for which proximity to death influences human behavior.

2. The Basic Model

Consider an individual with uncertain lifetime. Following the biological foundation of aging, the probability to be alive at age $t$ does not directly depend on chronological age $t$ but on the accumulated health deficits at that age, $S(D(t))$, $S' < 0$, in which $D$ is the health deficit index.\textsuperscript{2} Accumulated health deficits are thus an informative indicator of the proximity of death. Individuals are aware of this fact and discount the future at a higher rate when they expect death to be near, i.e., when many health deficits have been accumulated. There exists a health deficit index $\bar{D}$ above which survival is impossible, $S(\bar{D}) = 0$.\textsuperscript{3} Let $u(c(t))$ be the utility experienced from consuming $c$ at age $t$. Expected

\textsuperscript{1}Increasing discount rates could also be an explanation for the low demand of long-term care insurance (Brown and Finkelstein, 2011) because the costs arise in the near future while the benefits are expected to occur far in the future in old age.

\textsuperscript{2}In gerontology, aging is defined as the intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death (Arking, 2006, p. 11) and in a successful theory of aging, there should be no role for chronological age in explaining death (ibid., p. 10).

\textsuperscript{3}All equations of motion and statements about functional forms hold only for $D < \bar{D}$, i.e., until death has occurred with certainty.
lifetime utility is then given by

\[
V = \int_0^\infty S(D(t))u(c(t))e^{-\int_0^t \rho(D(v))dv}dt.
\] (1)

The feature that the discount rate \(\rho(D)\) depends on the state of health, which is – although time-variant – a predetermined state variable at any age, implies time consistency of decisions and avoids any complications that may arise from the recursiveness of the utility functional.\(^4\) Intuitively, the discount rate is stationary over time and over age, \textit{conditional} on the state of health of the individual.

The state of health, however, is not a choice variable. This is true even when health investment is a choice variable since the current state of health is predetermined by health investments earlier in life (see Appendix). By assuming that \(\rho' > 0\) we capture the idea that individuals discount utility more heavily when death is near. The mortality rate \(m\) is defined as the rate of change of the survival rate, \(m \equiv -\dot{S}/S = -S\dot{D}/S\).

We measure health deficits by the health deficits index, also called frailty index (Mitnitski et al, 2001). Mitnitski et al. (2002a) show that the relative number of health deficits \(D(t)\) increases with age \(t\) in an exponential way such that \(D(t) = a + be^{\mu t}\). This “law of deficit accumulation” explains around 95 percent of the variation in the data and its parameters are estimated with great precision. For most nations, the force of aging \(\mu\) is found to be around 3 to 4 percent (Mitnitski et al., 2002a, Harttgen et al., 2013, Abeliansky and Strulik, 2018). Differentiating \(D(t)\) with respect to age, we obtain the law of motion for health deficits:

\[
\dot{D} = \mu(D - a).
\] (2)

From a gerontological perspective, biological aging (i.e. the accumulation of health deficits and the deterioration of biomarkers) is not \textit{explained} by chronological age. As argued above, a successful theory of aging eliminates chronological age as an explanation of biological aging (Arking, 2006). In this sense, (2) is the key equation. It shows that the presence of many health deficits is conducive to a faster development of new health deficits (for evidence, see e.g., Mitnitski et. et al., 2006). This self-productivity of health deficit accumulation explains the progressive nature of biological aging. The “residual” \(a\) stands in for determinants that reduce or increase the speed of health deficit

\(^4\)See, for example, Obstfeld (1990) for recursive utility when the discount rate depends on consumption. Strulik (2012) explores the idea that the discount rate (of an infinitely long living individual) depends on wealth.
accumulation, such as health investments (see Appendix). In the benchmark model we treat these factors as exogenous.

Individuals can freely save and borrow and face the budget constraint

\[ \dot{k} = w + (r + m)k - c, \]  

(3)

in which \( k \) is financial wealth and \( w \) is a flow of non-financial income. We consider perfect annuity markets such that the interest rate is a compound of the return on capital \( r \), which is assumed to be constant, and the mortality rate \( m \) and individuals inherit no wealth and leave no bequests. As explained in the Introduction, this is an interesting benchmark since it has been shown that the feature of mortality as such is capable to generate a hump-shaped age-consumption pattern only when a (perfect) market for annuities is absent.

The easiest way to solve (1) subject to (2) and (3) is to apply a transformation of variables. Define \( q \equiv \int_0^k \rho(D(v))dv \) such that \( dq/dt = \rho(D) \) and \( dt = dq/\rho(D) \). This implies \( \dot{k} \equiv dk/dt = (dk/dq)(dq/dt) \) such that \( dk/dq = \dot{k}/\rho(D) \). The transformed problem is thus given by

\[ \text{max} \int_0^\infty S(D)u(c)e^{-q/\rho(D)}dq \text{ subject to } \dot{k} = \dot{k}/\rho(D). \]

The associated Hamiltonian reads:

\[ H = \frac{S(D)u(c)}{\rho(D)} + \frac{\lambda_k}{\rho(D)} [w + (r + m)k - c], \]  

(4)

with costate variable \( \lambda_k \). The first order condition and costate equation are:

\[ \frac{\partial H}{\partial c} = S(D)u'(c) - \lambda_k = 0 \]  

\[ \frac{\partial H}{\partial k} = \frac{\lambda_k(r + m)}{\rho(D)} = \lambda_k - \frac{d\lambda_k}{dq}. \]  

(5)  

(6)

We next reintroduce age by substituting \( dq = \rho(D)dt \). Thus (6) becomes \( \lambda_k(r + m) = \lambda_k\rho(D) - \dot{\lambda}_k \).

Differentiating (5) with respect to age provides \( \dot{S}/S + (u''/u')\dot{c} = \dot{\lambda}_k/\lambda_k \). Substituting \( \dot{\lambda}_k \), \( \lambda_k \), and the mortality rate \( m \equiv -\dot{S}/S \), provides the Euler equation:

\[ \frac{\dot{c}}{c} = \frac{r - \rho(D)}{\sigma}, \]  

(7)

in which \( \sigma \equiv -(u''/u')c \) denotes the inverse of the intertemporal elasticity of substitution, which is assumed to be constant for the benchmark model. For constant \( \rho(D) \) the solution collapses to the standard Ramsey rule and consumption evolves monotonously with age. Equation (7) also reflects the well-known result that the survival probability plays no role for the age-profile of consumption...
when individuals have access to annuities (Butler, 2001; Feigenbaum, 2008). In contrast, non-monotuous age-profiles of consumption can be motivated by a health-dependent discount rate. For example, if $\rho' > 0$, $\rho(0) < r$, and $\rho(\bar{D}) > r$, consumption exhibits a hump-shaped age-profile.

In order to explore the quantitative features of the result, we begin by considering the following parsimonious specification of the discount rate:

$$\rho = \bar{\rho}e^{\phi(D-D_0)}, \quad (8)$$

such that the discount rate equals $\bar{\rho}$ at the initial age and is exponentially increasing with the accumulation of health deficits. As a benchmark, I set $r = 0.07$ according to the long-run interest rate estimated in Jorda et al. (2019). The model is calibrated for a 20 years old male U.S. American in 2010. I set $w = 27,928$ when the individual is between 20 and 65 years old (i.e. the average labor income for single men in the year 2010; BLS, 2012) and $w = 0.45 \cdot 27,928$ above age 65, according to an average replacement rate of 0.45 (from the OECD, 2016). For health deficit accumulation, I take from the estimates of Mitnitski et al. (2002a), $\mu = 0.043$, $a = 0.02$, and $D_0 = 0.027$ at the initial age of 20 years. I approximate the empirical survival curve by the simple concave function $S(D) = \psi - \frac{\nu}{\nu + \chi D}$ and estimate $\psi = 1.75$, $\nu = 0.7$, and $\chi = 3.1$, see Appendix for details. I then calibrate $\sigma$, $\bar{\rho}$, and $\phi$ to approximate three points of age-specific consumption as estimated by Fernandez-Villaverde and Krueger (2007), namely at age 25, 50 (peak consumption), and 80. This leads to the estimates $\sigma = 0.99$, $\phi = 8.1$, and $\bar{\rho} = 0.056$. The estimated elasticity of intertemporal substitution is close to unity (log-utility), in line with studies suggesting that the “true” value of $\sigma$ is probably close to unity (Chetty et al., 2006; Layard et al., 2008).

**Figure 1: Health-Dependent Discounting and Age-Consumption Profile**

Blue (solid) lines: calibrated model. Dots indicate targeted data points, see text for details. Red (dashed lines): empirical estimates from Fernandez-Villaverde and Krueger (2007). Consumption is measured relative to peak consumption.
Results are shown in Figure 1. The panel on the left-hand side shows the imputed law of health deficit accumulation (2). The center panel shows the “inverse hyperbolic” age-profile of the implied discount rate $\rho(D)$. The panel on the right-hand side shows life cycle consumption, measured relative to peak consumption. Dots display the targeted data points. The full age-consumption profile from Fernandez-Villaverde and Krueger (2007) is shown by red (dashed) lines. The model traces the actual consumption profile quite closely from young to middle age and reasonably well from middle to old age.

The goodness of fit does not depend on the prevailing interest rate since there are three degrees of freedom to adjust the calibrated discount rate. For example, for $r = 0.05$, a match of the consumption hump with the targeted data points is obtained for $\phi = 10.1$, $\bar{\rho} = 0.038$, and $\sigma = 0.89$.

Non-constant discount rates, i.e. non-exponential discounting methods, are usually thought of as implying time-inconsistency (see e.g. Strotz, 1956; Angeletos et al., 2001) although there are exceptions when discounting is multiplicatively separable in planning time and decision time (see Burness, 1976; Strulik and Trimborn, 2018). Multiplicative separability, however, implies that preferences are no longer stationary and time-invariant (Drouhin, 2020), features that are regarded as desirable by many scholars. Moreover, multiplicative separability severely limits the potential functional forms that discount rates could assume.

Conceptualizing the discount rate as health-dependent provides an alternative way to implement non-constant discount rates that lead to time-consistent decisions. Tying the discount rate to the state of health allows to capture any possible association between the discount rate and biological age, measured by the accumulated health deficits in a person. These features make the approach of more general use than just an amendment to generate a plausible consumption path. For example, it could also be used to implement a declining (hyperbolic) discount rate at young age, capturing the idea that young persons (with almost perfect health) tend to discount the future at a high rate because death is far away. Combined with increasing discount rates at old age this behavior would then imply a u-shaped life-cycle pattern of the discount rate, as motivated in the Introduction.

These ideas can be integrated into the model by the health-dependent discount rate:

$$
\rho = \bar{\rho} + \phi_1 e^{-\phi_2 D} + \phi_3 e^{\phi_4 D},
$$

which replaces (8). The second term in (9) captures the hyperbolic decline of the time preference rate in young age (when health is very good). The third term captures the increase in time preference
in old age (and bad health). Figure 2 shows the implied discounting and consumption over the life cycle for an example where $\bar{\rho} = -0.03$, $\phi_1 = 30$, $\phi_2 = 250$, $\phi_3 = 0.05$, and $\phi_4 = 12$. In young adulthood, when the hyperbolic part of the discount rate is dominating, consumption is convex in age, it turns into a concave shape in middle age and reaches a maximum, after which it falls in old age.

The simple $\rho(D)$ functions cannot explain the convex part of the age-consumption profile in old age. One possibility would be to introduce more complex $\rho(D)$-functions. Indeed, it can be shown that a double-logistic function would generate the concave-convex pattern visible in the data. An alternative and intuitively more appealing way to explain the convexification in old age is to replace the iso-elastic utility function by a Stone-Geary utility function, $u(c) = [(c - \bar{c})^{1-\sigma} - 1]/(1 - \sigma)$ for $c > \bar{c}$ (and $-\infty$ otherwise). It provides the feature that the elasticity of intertemporal substitution declines as $c$ comes closer to the constant $\bar{c}$ and a greater share of consumption consists of “essentials” such as nutrition and shelter. Redoing the analysis as for the benchmark model, we arrive at the Euler equation

$$\dot{c} = \frac{r - \rho(D)}{\sigma} \cdot (c - \bar{c}),$$

which replaces (7). The rest of the model is as before. In particular, we can keep the simple monotonously increasing $\rho(D)$-function from the benchmark model. The first term on the right hand side of (10), taken in isolation produces a concave consumption hump. The second term reduces the slope as $c$ gets closer to $\bar{c}$ and generates a concave-convex consumption profile. Results for the calibrated model are shown in Figure 3 for $\bar{c} = 18,500$ and a re-calibration of the parameter values of the simple $\rho(D)$-function, $\phi = 31$, $\bar{\rho} = 0.034$. 

Age-discounting pattern according to (9) with $\bar{\rho} = -0.03$, $\phi_1 = 30$, $\phi_2 = 250$, $\phi_3 = 0.05$, and $\phi_4 = 12$. Rest of the model specified as for Figure 1.
Figure 3: Health-Dependent Discounting and Age-Consumption Profile with Subsistence Needs


3. Conclusion

Modeling discounting as health-dependent provides a straightforward and empirically plausible way to introduce a non-constant discount rate without time-inconsistency problems. This note focused on the age profile of consumption as an application. The Appendix shows an extension towards endogenous health behavior. Interesting further applications include problems where preferences depend on past consumption like habit formation or addiction. Here, we focused on an exponential discount factor. Whether similar generalizations are possible for other functional forms of the discount factor could be an interesting question for future research.


In this Appendix, I consider a more complex model in which the accumulation of health deficits can be slowed down by deliberate health investments such that equation (2) from the main text is replaced by

\[ \dot{D} = \mu (D - Ah^\gamma - a), \]  

(A.1)

where \( h \) is health investment and \( A \) and \( \gamma \) are parameters describing the available health technology. This functional form has been introduced and extensively discussed in Dalgaard and Strulik (2014).

The budget constraint (3) from the main text is replaced by

\[ \dot{k} = w + (r + m)k - c - ph, \]  

(A.2)

in which \( k \) is financial wealth, \( w \) is labor income, and \( p \) is the price of health care. The objective function remains the same as in the basic model. For convenience, it is here restated as (A.3)

\[ V = \int_0^\infty S(D(t))u(c(t))e^{-\int_0^t \rho(D(v))dv}dt. \]  

(A.3)

Individuals maximize (A.3) subject to (A.1) and (A.2) using the controls \( c \) and \( h \). Again, the easiest way to solve this problem is to apply a transformation of variables. Define \( q \equiv \int_0^t \rho(D(v))dv \) such that \( dq/dt = \rho(D) \) and \( dt = dq/\rho(D) \). This implies \( \dot{k} \equiv dk/dt = (dk/dq)(dq/dt) \) such that \( dk/dq = \dot{k}/\rho(D) \) and likewise for \( \dot{D} \equiv dD/dt \). The time-transformed problem (A.1)–(A.3) thus reads

\[ \max \int_0^\infty S(D)u(c)e^{-q} \frac{d}{\rho(D)} dq \quad \text{s.t.} \quad \frac{dk}{dq} = \frac{\dot{k}}{\rho(D)}, \quad \frac{dD}{dq} = \frac{\dot{D}}{\rho(D)}. \]  

(A.4)

The associated Hamiltonian reads

\[ H = \frac{S(D)u(c)}{\rho(D)} + \frac{\lambda_k}{\rho(D)} [w + (r + m)k - c - ph] + \frac{\lambda_D \mu}{\rho(D)} [D - Ah^\gamma - a], \]  

(A.5)

with costate variables \( \lambda_k \) and \( \lambda_D \). The first order conditions and costate equations are:

\[ \frac{\partial H}{\partial c} = S \frac{u'}{\rho} - \frac{\lambda_k}{\rho} = 0 \]  

(A.6)

\[ \frac{\partial H}{\partial h} = -\frac{\lambda_k p}{\rho} - \frac{\lambda_D \mu}{\rho} A \gamma h^{\gamma-1} = 0 \]  

(A.7)

\[ \frac{\partial H}{\partial k} = \frac{\lambda_k (r + m)}{\rho} = \lambda_k - \frac{d\lambda_k}{dq} \]  

(A.8)
\[
\frac{\partial H}{\partial D} = \frac{S' - \rho' S}{\rho^2} u + \frac{\lambda D \mu}{\rho^2} [\rho - \rho' (D - Ah^\gamma)] - \frac{\lambda k f}{\rho^2} [[w + (r + m)k - c - ph]] = \lambda_D - \frac{d \lambda_D}{dq}.
\]

We next reintroduce age by substituting \( dq = \rho(D)dt \). Thus (A.8) and (A.9) become

\[
\lambda_k (r + m) = \lambda_k \rho - \dot{\lambda}_k
\]

\[
\left[ S' - \frac{\rho' S}{\rho} \right] u + \frac{\lambda k f}{\rho} [w + (r + m)k - c - ph] + \lambda D \mu \left[ 1 - \frac{\rho'}{\rho} (D - Ah^\gamma - a) \right] = \lambda_D \rho - \dot{\lambda}_D
\]

(A.11)

Substituting \( \lambda_k \) from (A.6) and \( \lambda_D \) from (A.7), (A.11) becomes:

\[
- \left\{ \left( \frac{S'}{S} - \frac{\rho'}{\rho} \right) \frac{u}{w} - \frac{\rho'}{\rho} [w + (r + m)k - c - ph] \right\} \frac{\mu A h^\gamma - 1}{p} + \mu \left[ 1 - \frac{\rho'}{\rho} (D - Ah^\gamma - a) \right] - \rho = - \frac{\dot{\lambda}_D}{\lambda_D}.
\]

(A.12)

Differentiating (A.7) with respect to age and inserting (A.10) and (A.12) provides:

\[
\frac{\dot{h}}{h} = \frac{1}{1 - \gamma} \left\{ r + m - \mu \left[ 1 - \frac{\rho'}{\rho} (D - Ah^\gamma - a) \right] + \frac{\mu A h^\gamma - 1}{p} \left[ \left( \frac{S'}{S} - \frac{\rho'}{\rho} \right) \frac{u}{w} - \frac{\rho'}{\rho} (w + (r + m)k - c - ph) \right] \right\}
\]

(A.13)

Differentiating (A.6) with respect to age and inserting (A.10) provides the same Euler equation as for the basic model:

\[
\frac{\dot{c}}{c} = \frac{r - \rho(D)}{\sigma},
\]

(A.14)

in which \( \sigma \) denotes the inverse of the elasticity of intertemporal substitution. All increasing complexity thus arises from (A.13), which collapses to the simple health Euler equation in Dalgaard and Strulik (2014) for \( S' = \rho' = 0 \), i.e. when neither survival nor discounting depends on health.

In order to explore how the presence of health expenditure changes consumption behavior, I assume that health-dependent survival is given by the function:

\[
S(D) = \psi - \frac{\nu}{1 - \chi D}.
\]

(A.15)

I estimate the three parameters such that the model predicts a reasonable approximation of the empirical survival function \( S(t) \) when I feed in the predicted health deficits \( D(t) \), \( S(t) = S(D(t)) \).

The panel on the left-hand side of Figure A.1 shows the association between \( D(t) \) and \( S(t) \) implied by (A.15) for \( \psi = 1.75 \), \( \nu = 0.7 \), and \( \chi = 3.1 \). The survival probability declines at an increasing rate as more health deficits are accumulated and nobody survives \((1 - \nu/\psi)/\chi \) health deficits. The middle panel shows the association between age and accumulated deficits estimated by Mitnitski et
Figure A.1: Health-Dependent Survival and Survival by Age

\[ S(t) \] is the unconditional probability of surviving until age \( t \). Left: Assumed function \( S(D) \). Middle: Estimated association \( D(t) \) (Mitnitski et al., 2002). Right: Predicted (line) and estimated (stars) association between age and survival probability (estimates from Strulik and Vollmer, 2013). Implied life expectancy at 20: 55.5 years.

al. (2002) for 19-75 years old Canadian men. When I feed these data into the \( S(D(t)) \) function, I get the “reduced form”, \( S(t) \), which shows survival as a function of age. The implied functional relationship is shown on the right-hand side of Figure A.1. Stars in the panel on the right-hand side indicate the survival probability estimated from life tables for U.S. American men in 1975-1999, taken from Strulik and Vollmer (2013). The approximation somewhat overestimates the survival of the elderly and underestimates the survival of the oldest old but, altogether, it fits the data reasonably well.

The model is calibrated for a 20 years old male U.S. American in 2010. As for the simple model, I set \( r = 0.07 \), and \( \gamma = 0.2 \) as well as \( \mu = 0.043 \), and \( D_0 = 0.027 \) from Mitnitski et al. (2002a). I assume that the discount rate increases exponentially with deteriorating health, according to (8) from the main text. I normalize \( p = 1 \) and set \( w = 27,928 \) when the individual is between 20 and 65 years old (i.e. the average labor income for single men in the year 2010; BLS, 2012) and \( w = 0.45 \cdot 27,928 \) above age 65, according to the an average replacement rate of 0.45 (from the OECD, 2016). I then calibrate the remaining parameters to fit three points, at age 25, 50, and 80, from the empirical age-consumption curve (as in the main text) and two points at age 30 and 80 from health expenditure of American men in the year 2010, as well as a life expectancy at age 20 of 57.1 years (expected age at death at 77.1; NVSS, 2014). This provides the estimates \( \phi = 6.55 \), \( \bar{p} = 0.056 \), \( \sigma = 1.11 \), \( a = 0.0158 \), \( A = 0.0005 \). The predicted age-trajectories are shown in Figure A.2. Targeted data points are indicated by circles. The model predicts the age-profiles for health expenditure and consumption reasonably well.

We next explore the interaction between aging, discounting, and health expenditure with two numerical experiments. The first experiment considers an individual starting out at 20 with 10
percent more health deficits. The comparative dynamics are shown by red (dashed) lines in Figure A.2. Due to the self-productive nature of health deficit accumulation ($\dot{D}$ being positively affected by $D$), the initially less healthy individual develops new health deficits more quickly and ages faster, in a biologically sense (upper left panel in Figure A.2). This effect, taken for itself, induces more health expenditure in order to slow down aging. Faster aging also implies a faster increase of the discount rate (upper right panel), which taken for itself induces the individual to care less about a long life and to save less for health expenditure in old age. In summary, the second effect is dominated by the first effect, and health expenditure at any age is higher than in the benchmark case (lower right panel). Higher health expenditure, however, is not able to completely cure faster aging. The implied life expectancy at 20 is 51.3 years (compared to 57.1 years in the benchmark case).

The second experiment considers an individual facing a by 50 percent reduced level of medical efficacy $A$. Assuming an increase of medical efficacy of 1.4 percent per year (Abeliansky and Strulik, 2019), the experiment could capture the same individual born 50 years earlier. All other parameters,
including initial deficits, are set to benchmark level. Comparative dynamics are shown by green (dash-dotted) lines in Figure A.2. Facing a lower return on health expenditure, the individual spends less on health (lower right panel). Low efficacy and low health care spending interact such that the individual develops new health deficits more quickly (upper left panel). Included in the effects on faster biological aging is a feedback effect from health deficits to discounting (upper right panel). The implied life expectancy at 20 is 54.0 years.

In both experiments, faster aging induces more consumption in young age and less consumption in old age (lower left panel). The hump-shaped age-consumption profile is preserved.

**Additional References**


