Economics
Working Papers

2020-14

Anticipation of Deteriorating Health and Information Avoidance

Johannes Schünemann, Holger Strulik and Timo Trimborn

AARHUS BSS
DEPARTMENT OF ECONOMICS
AND BUSINESS ECONOMICS
AARHUS UNIVERSITY

Anticipation of Deteriorating Health and Information Avoidance*

Johannes Schünemann†, Holger Strulik‡ and Timo Trimborn§

September 2020.

Abstract. We integrate anticipatory utility and endogenous beliefs about future negative health shocks into a life-cycle model of physiological aging. Individuals care about their future utility derived from their health status and form endogenous beliefs about the probability of a negative health shock. We calibrate the model with data from gerontology and use the model to predict medical testing decisions of individuals. We find that anticipation in combination with endogenous beliefs provides a quantitatively strong motive to avoid medical testing for Huntington's disease which explains the low testing rates found empirically. We also study the case of breast and ovarian cancer and provide an explanation for why testing rates depend on the individual's income when treatment is available.

Keywords: Health, Anticipation, Longevity, Health Behavior, Beliefs, Information Avoidance

JEL: D11, D91, I12, J17

* We would like to thank Friedrich Breyer, Alexia Fürnkranz-Prskawetz, Volker Grossmann, Peter Haan, Holger Herz, Alexander Karmann, Mathias Kifmann, Michael Kuhn, Hendrik Schmitz, and Peter Zweifel for discussion and helpful comments. Financial support of the Swiss National Fund (SNF) and Deutsche Forschungsgemeinschaft (DFG) which co-finance the project "The Socioeconomic Health Gradient and Rising Old-Age Inequality" (grant no. 100018L_15009) is gratefully acknowledged.

† University of Fribourg, Department of Economics, Bd. de Perolles 90, 1700 Fribourg, Switzerland; email: johannes.schuenemann@unifr.ch

‡ University of Goettingen, Department of Economics, Platz der Goettinger Sieben 3, 37073 Goettingen, Germany; email: holger.strulik@wiwi.uni-goettingen.de.

§ Aarhus University, Department of Economics and Business Economics, Fuglesangs Allé 4, 8210 Aarhus V, Denmark; email: tt@econ.au.dk..

1. Introduction

Advances in the understanding of human genetics have considerably increased the possibilities of genetic testing for hereditary diseases. However, testing rates of high-risk individuals, i.e. individuals with a family history of a certain hereditary disease, remain far from universal even if testing costs are negligible and the predictive power of the test is high. Studies report low testing rates of high-risk individuals for Huntington's disease (HD) (5-10%; Shoulson and Young, 2011; Oster et al., 2013a), cancer (60%; Ropka et al., 2006; Lerman et al. 1996), and Alzheimer's disease (25%; Roberts et al., 2004). Standard health economic models cannot explain this observation. In these models, having more information on future health shocks is always beneficial since individuals are able to adjust and re-optimize their behavior.

Several papers suggest that the combination of low testing rates and low testing costs may be motivated by future beliefs affecting utility directly (e.g. Fels, 2015; Koszegi, 2003). A recent prominent study in this context was conducted by Oster et al. (2013a). The authors propose an optimal expectation model based on Brunnermeier and Parker (2005) with anticipatory utility in order to explain the low testing rates for HD found in their data. In their model, the anticipation of developing HD in the future reduces experienced utility already today which may compensate the utility loss from having less information, and thus encourage the individual to refuse the test. Benabou and Tirole (2016) define this type of information avoidance as strategic ignorance. While this strand of the literature carves out the mechanism of how endogenous beliefs in combination with anticipatory utility can lead to information avoidance for specific examples, there exists no general model yet which is able to provide quantitative predictions for a wide range of health-related cases of information avoidance.

We fill this gap by combining two strands of literature. We introduce anticipatory utility and endogenous beliefs about future negative health shocks into a quantitative stochastic life-cycle model of endogenous health and longevity[1]. In this model individuals choose endogenous beliefs about the probability at which negative health shocks arrive. The subjective probability may deviate from the true probability in order to balance the effect of avoiding the negative impact of

[1]Seminal studies in the literature on beliefs are given by Caplin and Leahy (2001, 2004) and Brunnermeier and Parker (2005). See Benabou and Tirole (2006) for a comprehensive survey. Our stochastic life-cycle model of endogenous health and longevity is based on the deterministic model of Dalgaard and Strulik (2014). See also Schünemann et al. (2017a, 2017b, 2020) for related applications.

anticipating the health shock with the detrimental effect of worse decision-making with respect to health-related behavior and consumption.

We propose a new method to solve these kind of stochastic problems. Our method relies nowhere on approximation of the policy function, but instead solves the non-linear problem up to a user-specified error. Because our model has a micro-foundation in gerontology, it can be applied to various health-related cases of information avoidance and predict quantitatively correctly the testing decisions of individuals.

The psychology of anticipatory feelings has been acknowledged and analyzed in various settings. Lazarus (1966) surveys the experimental literature showing that certain forms of physical pain, such as pin pricks, do not cause any distress beyond the mere anticipation of those events. By means of survey techniques, Loewenstein (1987) finds that the willingness to pay for avoiding an electric shock delayed by one up to ten days was substantially higher than for avoiding an immediate shock. These studies imply that people experience higher disutility from anticipating than from experiencing the negative shock. Most importantly for our study, the detrimental effect of anticipation has also been established in the medical literature. A natural way of identifying anticipatory effects in the context of future health deficits is to look at the effect of diagnoses on measures of happiness before symptoms of the disease have set in. Honiden et al. (2006), for example, find a negative impact of HIV diagnosis on health-related utility. Likewise, Cuypers et al. (2017) report a detrimental effect of prostate cancer diagnosis on health-related quality of life.

We set up a continuous-time stochastic life-cycle model of human aging in which not only the realization of actual health deficits but also the anticipation of future health deficits reduce instantaneous utility. At this point we should explain how we distinguish *anticipation* from *expectation* and how we extend the available health economic theory. In any dynamic economic model, individuals form expectations about the future and these expectations affect their current behavior. Conventional economic models, however, assume that the expectation of future events has no impact on *currently* experienced (i.e. instantaneous) utility. Anticipation, in contrast, means that the expectation of bad future events affects instantaneous utility already today. Through this channel, it may then elicit further behavioral changes that may amplify or dampen those evoked by the mere "cold" expectation of these events. We apply this notion of anticipation in a dynamic model of health and aging where individuals are assumed to experience a decline

in instantaneous utility today from health deficits developed in the future. We calibrate the model with gerontological data to fit observed health behavior and outcomes. To the best of our knowledge, we are the first to provide estimates for health-related anticipation parameters.

We then use our model of health deficit anticipation to contribute to the discussion on information avoidance. For this purpose we introduce a negative health shock that arrives with uncertainty and for which there is (potentially) specific treatment available to mitigate the shock. Individuals form endogenous beliefs about the probability with which the health shock may set in and face the decision whether they want to be tested and resolve (part) of the uncertainty associated with the shock. We then apply the general model to two cases to demonstrate the predictive power with respect to information avoidance.

We first design an experiment that mimics the case of genetic testing of high-risk individuals for HD. Although the individual is aware of the high risk of developing HD, the individual optimally chooses to believe that he or she is perfectly healthy if untested. This predicted behavior is in line with empirical evidence suggesting that high-risk untested individuals behave similarly to those who are certain not to have the disease (Oster et al., 2013a). The predictive power constitutes an important value added in comparison to the study by Oster et al. (2013a). In their model, the non-testing decision for HD is rationalized *ex post* by varying exogenous utility weights. If anticipatory utility is exogenously assigned a sufficiently high value in total utility, the model manages to motivate non-testing behavior found in the data. In our model, these utility weights are determined endogenously by calibrating the parameters with actual data. This allows us to provide predictions for testing behavior regarding different diseases. Our model then suggests that including anticipation of future health deficits into the utility function induces people to avoid being tested. Although individuals can re-optimize their decisions after being diagnosed, they experience an instantaneous utility loss from knowing that they will become ill. This loss is quantitatively substantial. We estimate the willingness to pay for avoiding a genetic test for HD at the age of 20 to be around $ 130,000 or five annual wages.

We also provide an explanation for why testing rates of high-risk individuals are far from universal even when effective treatment is available. To this end, we study the case of hereditary breast and ovarian cancer for which highly predictive tests exist. We show that a woman with average wage chooses to be tested and treated if tested positive. Below a certain threshold wage, however, treatment becomes too costly in terms of welfare. If treatment is not feasible,

the individual chooses to remain untested in order to avoid the disutility caused by anticipation following a potentially positive test result. Consistent with empirical evidence (e.g. Stenehjem et al., 2018; Lerman et al., 1996), our model thus suggests that testing behavior depends on the socioeconomic status of individuals even if the cost of the test is negligible.

The model that we develop below in order to discuss the effects of anticipation of deteriorating health is particularly suitable for this purpose since it conceptualizes aging as the progressive accumulation of health deficits. The alternative paradigm, the Grossman (1972) model, is less suitable since it is based on health capital accumulation. Besides structural shortcomings, health capital is a latent variable, unknown to doctors and medical scientists, which confounds any serious calibration of the model. The health deficit model, based on Dalgaard and Strulik (2014), in contrast, is founded in gerontological research, which enables us to calibrate it straightforwardly using the so-called frailty index (Mitnitski et al., 2002a,b). Other studies employing the health deficit model investigate the role of adaptation for health behavior (Schünemann et al., 2017a), the gender gap in mortality (Schünemann et al., 2017b), optimal aging in partnerships (Schünemann et al., 2020), the historical evolution of retirement (Dalgaard and Strulik, 2017), and the education gradient (Strulik, 2018).

The paper is organized as follows. Section 2 presents the basic model of health deficit anticipation which we calibrate to the health behavior and outcomes of a 20-year-old reference U.S. American in the year 2010. In Section 3, we contribute to the discussion on information avoidance. In Section 4 and 5, respectively, we apply our model to provide a quantitatively meaningful explanation for why people refuse to be tested for HD and we investigate why in the case of hereditary breast and ovarian cancer people refuse to be tested even if effective treatment is available. Section 6 concludes.

2. Anticipation in a Life-Cycle Model of Endogenous Health and Longevity

2.1. **The Basic Model.** Individuals maximize expected lifetime utility at time 0. In order to elaborate clearly the role of anticipation of severe health shocks we assume that aging, conceptualized as the accumulation of health deficits, is a deterministic process, which is interrupted at most once by a severe health shock. We also assume that individuals correctly anticipate the age at which the shock occurs, if it occurs, such that the only uncertainty in life is whether the shock occurs or not. As an example, consider an individual whose parent suffers from HD such

that the individual carries the genetic expansion with 50% probability. Therefore, the individual faces uncertainty whether the shock eventually occurs or not. Since the course of the disease is highly predictable, however, the individual can correctly anticipate the age and severity of the health shock in case the individual carries the genetic defect.

The individual derives utility from consumption and from being in good health. The (objective) state of health is measured by the accumulated health deficits D. We assume that health deteriorates through two different channels. First, the process of aging deteriorates health over time through the continuous accumulation of health deficits (see Dalgaard and Strulik; 2014). Individuals can slow down the accumulation of deficits caused by aging by deliberate (monetary) health investments h. Second, health might deteriorate as a result of a severe one-time health shock s that arrives with probability $0 < p < 1$ at time $\tilde{t} > 0$. Individuals can mitigate the health shock by targeted (monetary) health investments h_s, if treatment is available. The accumulation of health deficits is characterized by the following stochastic differential equation:

$$dD(t) = \mu(D(t) - Ah(t)^{\gamma} - a)dt + g(D(\tilde{t}^{-}), h_s)ds \tag{1}$$

where μ represents the "natural" rate of aging. The scale parameters A and the curvature parameter γ govern the health technology with $A > 0$ and $0 < \gamma < 1$, while a captures environmental influences beyond individual control. Note that at time $\tilde{t}$, s can take the states $s = 1$ with probability p and $s = 0$ with probability $1-p$. At all other points in time $s \equiv 0$ holds. The function g captures the properties of the specific health shock under consideration, i.e. if and at which efficacy treatment is available, and if the severity of the health shock depends on the already accumulated amount of deficits at time $\tilde{t}^{-}$, just before the shock arrives. The individual dies when it has accumulated a critical deficit level D_T.

Because by assumption the health shock may only set in at time $\tilde{t}$, we simplify the differential equation for the intervals $[0, \tilde{t})$ and $(\tilde{t}, T]$ to

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

and treat the possible occurrence of a health shock at $\tilde{t}$ separately below.

Apart from the actual health state, utility is also affected by the anticipation of future health deficits. We model the "stock" of anticipation, denoted by R, as a weighted average of future

health deficits according to

$$R = D_T e^{-\theta(T-t)} + \theta \int_t^T e^{-\theta(\tau-t)} D(\tau)\ \mathrm{d}\tau, \tag{3}$$

where T is the time of death and θ captures the discounting of future health deficits. Therefore, a higher θ implies that deficits accumulated farther in the future receive a lower weight in the anticipation stock. The first term in equation (3) accounts for the anticipation of death, which will become more pronounced the closer the individual approaches the terminal health deficit level D_T. Note also that at the time of death T, the final level of the anticipation stock coincides with the final deficit level and thus $R(T) = D_T$. Our conceptualization of deficit anticipation is related to the modeling of consumption anticipation in Monteiro and Turnovsky (2016), which turns out to be analytically convenient. Differentiating equation (3) with respect to time provides the following simple differential equation:

$$\dot{R} = \theta\,(R - D)\,. \tag{4}$$

Following empirical evidence by Finkelstein et al. (2013), we assume that bad health affects both utility and marginal utility of consumption. Specifically, the instantaneous utility of the individual is given by

$$U(c, D, R) = \left(\frac{\bar{D}}{D}\right)^{\alpha} \left(\frac{\bar{D}}{R}\right)^{\beta} \cdot \tilde{u}(c), \qquad \text{with } \tilde{u}(c) = \begin{cases} \frac{c^{1-\sigma}-1}{1-\sigma} & \text{for } \sigma \neq 1 \\ \log(c) & \text{for } \sigma = 1 \end{cases}. \tag{5}$$

The actual health state as well as the anticipated future health state is evaluated relative to the state of best health $\bar{D}$. The parameter α captures by how much an additional health deficit affects utility, while β governs the impact of a unit increase in the anticipation stock.

The individual receives labor income w which can be spent on consumption, health services and savings. We suppose that the individual has access to financial markets and can save or borrow at net interest rate r. The budget constraint thus reads

$$\dot{k} = w + rk - c - p_h h, \tag{6}$$

where p_h denotes the relative price of health investments. If treatment is available and the individual chooses to be treated, there will be a one-time cost $p_s h_s$ at the time of the treatment

where p_s denotes the relative price of the treatment. Finally, individuals choose beliefs b about the probability at which a potential health shock will occur. These beliefs may differ from the true probability p. The individual chooses $c(t)$, $h(t)$, b, h_s, and whether to be tested or remain untested to maximize expected lifetime utility[2]

$$E_0[V_0] = E_0 \left[\int_0^T u(c, D, R) e^{-\rho t} \mathrm{d}t \right] \tag{7}$$

subject to (1), (4)–(6), as well as the initial conditions $D(0) = D_0$ and $k(0) = k_0$, and the terminal conditions $D(T) = D_T$, $k(T) = k_T$, and $R(T) = R_T = D_T$. The parameter ρ represents the time preference rate of the individual. The time of death T is endogenous. Through their health expenditure plan, individuals influence the accumulation of health deficits and thus the time of death, which occurs when D_T deficits have been accumulated.

At this point we should take up the discussion of the introduction again and elaborate on the distinction between *anticipation* and *expectation*. As in any other dynamic model, individuals form expectations about the future which affect current economic behavior. A particular form of expectations in our model refers to potential health shocks. If an individual is tested, the uncertainty about the health shock is resolved. In case the test is positive, this health shock is said to be *expected* because the individual knows before that the shock will eventually set in.

If the individual remains untested, the individual chooses beliefs about the probability at which the potential health shock occurs. If, for example, the individual chooses $b = 1$, the individual chooses to believe that the shock occurs with certainty. In case the shock eventually occurs, this setting is identical to the one for the diagnosed expected health shock. If the individual chooses $b = 0$, the individual chooses to believe that the health shock will with certainty not materialize and lives in denial. If a health shock occurs in this setting, this shock is said to be *unexpected*. Naturally, there are infinitely many scenarios depending on the beliefs the individual chooses.

In contrast to conventional models, currently experienced instantaneous utility is directly affected by these expectations (by including the anticipation stock R into $u(\cdot)$). Our benchmark individual is said to be *anticipating* because expectations about the future, like the expectation of future health shocks, reduce instantaneous utility already today. An individual is considered *non-anticipating*, on the other hand, if expectations have no direct impact on instantaneous utility today. According to our definition, an unexpected future health shock neither affects

[2]We elaborate on the testing decision in detail in Section 2.3.

today's instantaneous utility of the anticipating nor the non-anticipating type because people do not consider that the health shock will eventually arrive.

The implementation of unexpected health shocks will be our means to model an individual which carries a defected gene, is not diagnosed and lives in denial. With expected health shocks we will model carriers who are diagnosed before and thus know that they will develop a disease at some point in the future. In this case, anticipating individuals experience a reduction in instantaneous utility at the time of the diagnosis (and before the disease sets in), while instantaneous utility of non-anticipating types remains unaffected by the diagnosis. However, a positive diagnosis may still change health behavior of the non-anticipating type in order to prepare for the upcoming shock.

2.2. **Model Solution.** We decompose the stochastic optimization problem into two deterministic problems on the time intervals $[0, \tilde{t})$ and $(\tilde{t}, T]$, which are both independent from beliefs b. In the first step we derive interior optimality conditions which hold for both time intervals, and introduce the initial and final boundary conditions which have to hold at time 0 and T. In the second step, we connect both intervals by determining the interior boundary conditions for $\tilde{t}$.

The Hamiltonian associated with this life-cycle problem for time intervals $[0, \tilde{t})$ and $(\tilde{t}, T]$ is given by

$$\mathcal{H} = u(c, D, R) + \lambda_k \dot{k} + \lambda_D \dot{D}, \tag{8}$$

where λ_k and λ_D denote the shadow prices of capital and deficits. The individual takes the evolution of the anticipation stock as given. The transversality condition for this free-terminal-time problem is given by $H(T) = 0$. Note that the non-anticipating (i.e. the conventionally considered) individual can be easily deduced by setting $R = D_0 \, \forall \, t$. In that case, the anticipation stock equals initial health deficits over the whole life cycle so that utility is not affected by anticipation.

The following dynamic equations for consumption and health investment (9) and (10) are derived in the Appendix. From the first-order conditions for the maximization of $\mathcal{H}$, we obtain the Euler equation for consumption growth

$$\frac{\dot{c}}{c} = \frac{r - \rho - \alpha \frac{\dot{D}}{D} - \beta \frac{\dot{R}}{R}}{\sigma}. \tag{9}$$

In case $\alpha = \beta = 0$, consumption growth is equal to that in the standard life-cycle model. In case $\alpha > 0$, health matters for the individual in the utility function and deficit accumulation slows down consumption growth. The reason behind this result can be found in the health-consumption complementarity. Since a deteriorating state of health reduces the marginal utility of consumption, individuals substitute future for present consumption in order to consume when the marginal utility of consumption is still high. Since deficit anticipation enters utility qualitatively in the same way as actual health deficits, anticipation affects consumption growth symmetrically.

The Euler equation for health expenditure growth is obtained as

$$\frac{\dot{h}}{h} = \frac{r - \mu + \frac{\alpha U}{D\lambda_D}}{1 - \gamma}. \tag{10}$$

If $\alpha = 0$ and health does not affect utility, equation (10) equals the standard Euler equation for health investments developed in Dalgaard and Strulik (2014). In this case, health investments increase over the life cycle if the interest rate exceeds the rate of aging. If $\alpha > 0$, health expenditure growth declines as people substitute future for present health investments to enjoy a good state of health already at the beginning of their life. To see this, note that health deficits are a "bad" rather than a "good", implying that the shadow price λ_D and thus the last term in the numerator in equation (10) is negative.

While beliefs b and therefore the stochastic component of the maximization problem do not enter the intertemporal first order conditions, they have to be considered at the point of time $\tilde{t}$ when the health shock may set in. To this end, consider first a deterministic setting in which a health shock arrives with certainty such that the individual does not form any beliefs. In this case, optimality requires the forward looking variables λ_k, λ_D, and R to evolve continuously at $\tilde{t}$ (see Bryson and Ho, 1975, pp. 101-104).

If the health shock is stochastic, as in our case, individuals choose beliefs about the probability at which the shock will occur. In this case Bellman's principle requires that the forward looking variables are continuous in expected values. This implies that

$$\lambda_k(\tilde{t}^-) = b\lambda_{k,s=1}(\tilde{t}^+) + (1-b)\lambda_{k,s=0}(\tilde{t}^+) \tag{11a}$$

$$\lambda_D(\tilde{t}^-) = b\lambda_{D,s=1}(\tilde{t}^+) + (1-b)\lambda_{D,s=0}(\tilde{t}^+) \tag{11b}$$

$$R(\tilde{t}^-) = bR_{s=1}(\tilde{t}^+) + (1-b)R_{s=0}(\tilde{t}^+) \tag{11c}$$

holds where $\tilde{t}^-$ and $\tilde{t}^+$ relate to just before and after time $\tilde{t}$, respectively, and the indices $s = 0$ and $s = 1$ indicate the no-shock scenario and the shock scenario, respectively. These conditions capture that the individual considers the case of a shock ($s = 1$) and the case of no shock ($s = 0$) already for his health behavior before $\tilde{t}$ through the forward looking variables. The stronger the individual believes in the occurrence of the shock (the higher b), the larger is the impact of the shock scenario $s = 1$ for his planning and vice versa. Also note that if the individual chooses beliefs $b = 1$ ($b = 0$) and the shock in fact occurs (does not occur), the forward looking variables are continuous at time $\tilde{t}$ as in the deterministic case.

2.3. **The Testing Decision.** We assume that the decision whether the individual is tested or not has to be taken at time 0. If no treatment is available (as for the HD case), the individual chooses to be tested if $E_0[V_{tested}] > E_0[V_{untested}]$ where we omit the time index for V, with

$$E_0[V_{tested}] = pV_{s=1} + (1-p)V_{s=0} \tag{12}$$

$$E_0[V_{untested}] = pE[V(b)_{s=1}] + (1-p)E[V(b)_{s=0}]. \tag{13}$$

If the individual is tested, the expected lifetime utility is just a weighted average of the no-shock and the shock scenario where the weights are given by the objective shock probability p and the no-shock probability $(1-p)$ (Equation (12)). Since the true state about the genetic disorder is revealed, beliefs are no longer free to be chosen and thus do not affect decisions. Therefore, the value function V does not depend on b anymore. In this setting, the only uncertainty refers to whether the test is positive or negative, captured by the probability p. Once the individual receives the test result, the maximization problem becomes deterministic. Therefore, the forward looking variables behave continuously at the time of the shock, as for the deterministic case described above.

If the individual remains untested, individuals choose beliefs that affect the behavior already before the shock. When taking their optimal decisions in $t = 0$, individuals form subjective shock probabilities and these perceived probabilities determine the pre-shock behavior. At $\tilde{t}$, the individual either receives the health shock or not and the true state is revealed. This setting implies that before the shock the individual behaves the same no matter whether the shock will eventually set in or not. After $\tilde{t}$, the individual continues his life in the shock scenario $s = 1$ with probability p or in the no-shock scenario $s = 0$ with probability $1 - p$. This calculus is

formally summarized in Equation (13) where $E[V(b)_{s=1}]$ describes expected lifetime utility if untested in the shock scenario and $E[V(b)_{s=0}]$ describes expected lifetime utility if untested in the no-shock scenario.

If treatment is available (like in the cancer case), the individual is tested and treated if tested positive if $E[V_{treated}] > max\{E[V_{tested}], E[V_{untested}]\}$ with

$$E[V_{treated}] = pV_{s=0,tr} + (1-p)V_{s=0}. \tag{14}$$

We consider here the special case that treatment is effective and removes the risk of developing the disease completely. The associated value function is denoted by $V_{s=0,tr}$. This scenario differs from the no-shock scenario in the tested case without treatment $V_{s=0}$ since treatment is costly and thus affects the budget constraint. Like in the tested case without treatment, however, the only uncertainty is whether the test is positive (with probability p) or negative (with probability $1-p$). Note however that our model can be easily extended to the case where treatment is only partially effective or where the outcome of the treatment is uncertain. In this case, individuals would choose their beliefs based on the effectiveness of the treatment.

2.4. **Model Calibration.** We solve the model numerically using a shooting procedure[3]. For this purpose, we calibrate the baseline model to a 20-year-old male U.S. American in the year 2010. As far as the biological parameters are concerned, the regression analysis by Mitnitski et al. (2002a) employing the frailty index provides us with most of the parameter values. The frailty index has been established in gerontology as a straightforward metric to measure the state of health. It includes various health deficits ranging from mild nuisances (e.g. reduced vision) to fatal disorders (e.g. cancer). The frailty index is then constructed as the proportion of deficits that an individual has from a set of potential deficits. Naturally, the frailty index increases as a function of age. In order to quantify the state of best health $\bar{D}$, we set it equal to the initial state of health when the individual is born in our model, i.e. $\bar{D} = D(0)$. We back out $D(0) = 0.0274$ as the relevant initial deficit level associated with a man of age 20 which is the starting age in our model. The terminal state is given by $D(T) = 0.1059$ which is associated with a man 57.1 years later; the life expectancy at age 20 for males was 57.1 years in 2010 (i.e. death at 77.1 years; NVSR, 2017). Since $R(T) = D(T)$, the same value applies for the final level of the anticipation stock. From the same study, we take $\mu = 0.043$ as the value for the natural

[3]Details on the solution procedure are provided in the Appendix.

rate of aging. Moreover, we take the estimates for the environmental constant $a = 0.013$ and the curvature parameter of the health technology $\gamma = 0.19$ directly from Dalgaard and Strulik (2014). We further set $r = 0.07$ according to the long-run interest rate from Jorda et al. (2019), normalize the relative price of health services to one ($p_h = p_s = 1$) and set $w = 27,928$ according to data on wages and salaries for U.S. American single men in 2010 (BLS, 2012).

We simultaneously estimate the remaining six parameters ρ, σ, α, β, A, and θ by fitting the following six data points: (i) a stable consumption path over the life cycle (see e.g. Browning and Ejrnæs, 2009), (ii) the average life expectancy of 20-year-old men, (iii) health expenditures at age 30, 50, and 70 (MEPS, 2010), and (iv) a reduction of health-related utility of 8% following an HIV diagnosis (Honiden et al., 2006). The study of Honiden et al. (2006) measures how health-related utility is affected by an HIV diagnosis, before symptoms and treatment have set in. The survey is carried out for HIV diagnoses in the 1980s and 1990s, a time where there was basically no effective treatment for HIV. Without treatment, the median time from seroconversion to AIDS has been found to be 9 years, while death occurs one year later (Nakagawa et al., 2013). In order to determine the anticipation parameter, we therefore model a health shock which is diagnosed at the age of 35 (the mean age at diagnosis in Honiden et al.'s study), sets in 9 years later and leads to death after another year. We then adjust parameters such that the shock implies a loss in health related utility, captured in our model by $\left(\frac{\bar{D}}{D}\right)^{\alpha}\left(\frac{\bar{D}}{R}\right)^{\beta}$, of 8% at the time of diagnosis. The calibration results and the externally set biological parameters are shown in Table 1.

Table 1a: Calibration Results

ρ	σ	α	β	A	θ
0.065	1.16	0.01	0.22	0.00146	0.10

Table 1b: Externally Set Parameters

D_0	D_T	μ	a	γ	w	r
0.0274	0.0159	0.043	0.013	0.19	27,928	0.07

While some of these parameters are latent and thus cannot be compared to empirical estimates in the literature, our estimate for σ fits well the findings in previous studies that the 'true" value of σ is probably close to unity (e.g. Chetty et al., 2006) or slightly above unity, around

1.2 (Layard et al., 2008). Our calibrated value for α implies that a one-standard-deviation increase in deficits is associated with a reduction in the marginal utility of consumption of 0.4%.[4] Finkelstein et al. (2013) report a point estimate of this effect of 11%, with a confidence interval ranging from 2.7%–16.8%. Therefore, our parameter value is below the lower bound of Finkelstein et al.'s (2013) confidence interval. When estimating the effect of health on the marginal utility of consumption, however, Finkelstein et al. (2013) do not take into account the effect of anticipation which makes our results not directly comparable to those obtained in their study. Another reason for the discrepancy between the estimates may lie in the fact that the Finkelstein et al. (2013) study only uses a narrow set of very severe health deficits while the frailty index includes a wide range of health deficits of varying severity.

Comparing the parameter values of α and β reveals that the individual experiences much more disutility from anticipating future deficits than from developing actual health deficits. This observation is consistent with the empirical observations reported in the introduction that the anticipation of electric shocks or pin pricks is more harmful for the individual than the negative shock itself. Calibrating a theory of health anticipation to actual data, we confirm this notion also in the context of human aging and the associated accumulation of health deficits.

As far as the anticipation parameter θ is concerned, Monteiro and Turnovsky (2016) who model anticipation of consumption in a structurally similar way draw on habit formation studies and set $\theta = 0.2$ in their numerical analysis. Besides the fact that consumption anticipation cannot be directly compared to the anticipation of future health deficits, in the habit formation literature the reference stock is a weighted average of past consumption levels, while in an anticipation framework we consider a future stock of consumption streams. To the best of our knowledge, we are the first to calibrate the anticipation parameter θ to actual data.

2.5. **Results.** In order to show the baseline results and our model fit to the data, we focus in this subsection on the age trajectories when no health shock is considered[5]. Therefore, the anticipation stock only refers to the anticipation of aging-related deficits. Figure 1 shows the results for the age trajectories of the anticipation stock, health investments, health deficits, utility, consumption and capital. The dots indicate data points. The upper-left panel of Figure

[4]Given the average life expectancy applied in our calibration of 77.1 years, the frailty index in Mitnitski et al. (2002) implies a deficit mean of 0.0521 and a standard deviation of 0.0221. Starting from the mean, a one-standard-deviation increase in deficits reduces the marginal utility of consumption by 0.04% for $\alpha = 0.01$.

[5]The HIV shock considered in the calibration section only served for pinning down the anticipation parameters. In the benchmark run carried out in this section, there will be no such shock.

1 shows the evolution of the anticipation stock which increases in the course of aging. Inspecting equation (3), there are two mechanisms how the stock of anticipation is affected as people age. First, the lower bound of the integral increases, which, taken for itself, reduces the anticipation stock since the time span at which future deficits are accounted for decreases. Second, the number of health deficits increases with age, implying that, in the course of aging, health states with a high number of deficits come closer and are discounted less heavily. This increases the anticipation stock and it is the dominating effect as shown in Figure 1.

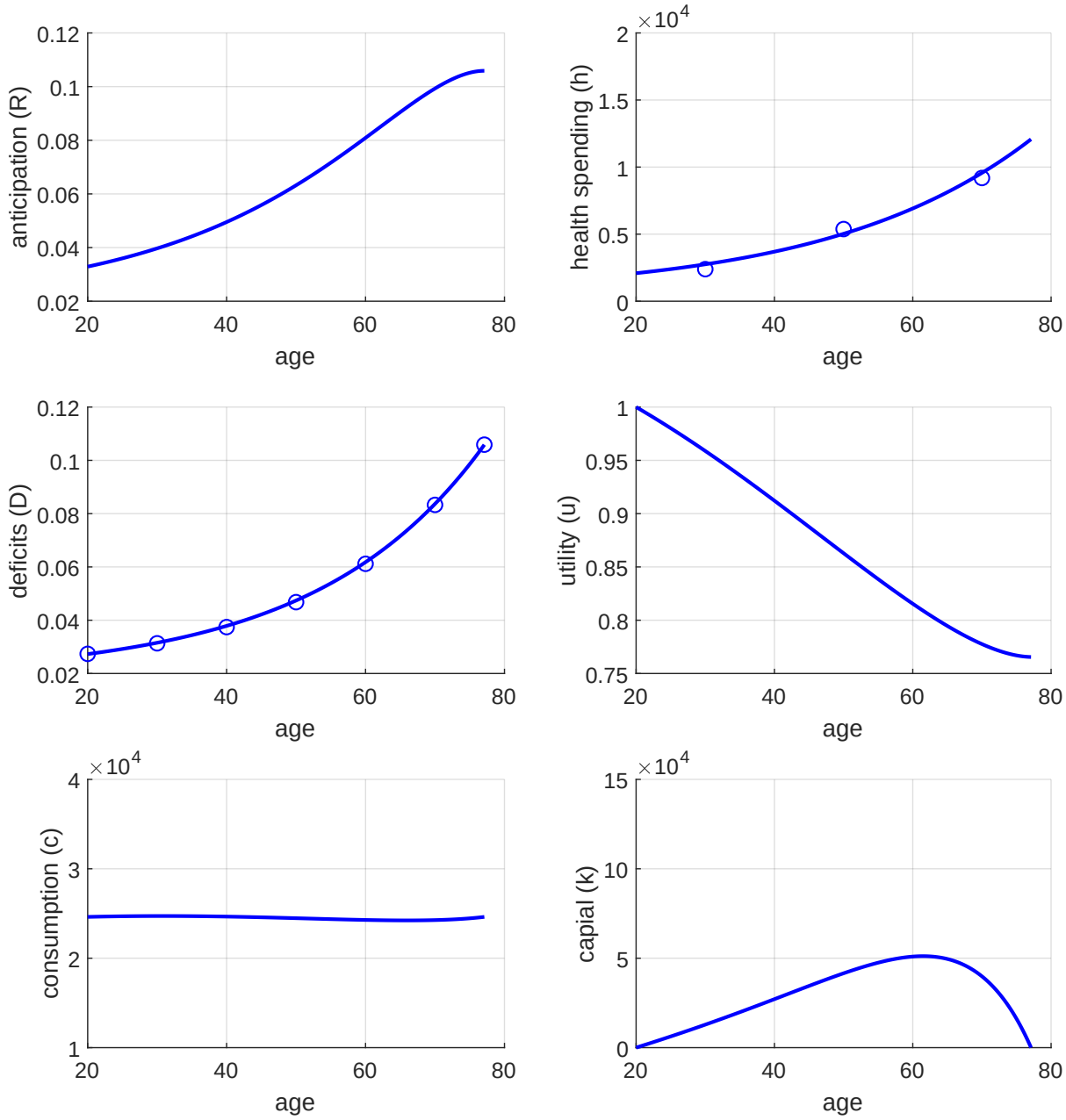
As can be seen from the upper-right panel in Figure 1, health expenditure increases over the life cycle as observed empirically (MEPS, 2010). The panel also shows that the model manages to fit actual expenditure data reasonably well. The center left panel shows the exponential evolution of health deficits over the life course. Although our calibration strategy targets only the first and final value of D, the model predicts the whole age trajectory of deficit accumulation in a satisfying manner. The center right panel illustrates instantaneous utility, which decreases in the course of aging. Since consumption is calibrated to be constant over the life cycle (see lower left panel), the utility decline is a direct result of aging (the accumulation of D) and anticipation (the accumulation of R). The last panel shows the typical evolution of capital in a life-cycle model.

3. Testing vs. Non-Testing and Information Avoidance

In this section, we use our model of health anticipation to investigate information avoidance in the context of health. Several studies have reported considerably low testing rates among high-risk individuals although the cost for testing is very low. These studies report low testing rates for e.g. Huntington's disease (HD) (Oster et al., 2013a; Shoulson and Young, 2011), cancer (Lerman et al. 1996; Ropka et al., 2006), and Alzheimer's disease (Roberts et al., 2004). This pattern is hard to reconcile with standard economic theory (Oster et. al, 2013a).

The idea that anticipation may lead to information avoidance is not an entirely new one. Several previous studies have acknowledged that the combination of low testing rates and low testing costs may be motivated by future beliefs affecting utility directly (e.g. Koszegi, 2003). In particular, Oster et al. (2013a) propose an optimal expectation model based on Brunnermeier and Parker (2005) with anticipatory utility in order to explain the low testing rates for HD found in their data. We introduce anticipation into a life-cycle model of endogenous health

Figure 1: Health Anticipation: Benchmark Run



Blue (solid) lines: anticipating type. Red (dashed) lines: non-anticipating type. Utility is instantaneous utility relative to initial utility of an anticipating individual. Dots indicate data points. Data for health spending are from MEPS (2010) and data on deficits are from the frailty index in Mitnitski et al. (2002).

and longevity in which the actual health state has a micro-foundation in gerontology. Since we include anticipation as a state variable dependent on the actual evolution of future health, we are able to quantify the detrimental effect that the diagnosis of a future disease has on life-cycle behavior and thus longevity and welfare of the individual. While the study of Oster et al. (2013a) rationalizes non-testing behavior ex post, we employ our model to predict testing rates

and show that testing rates may depend on the socioeconomic status of the individual. For this purpose we do not only apply our model to HD for which no effective treatment is available, but also to the case of hereditary breast and ovarian cancer to study the role of anticipation when effective treatment exists. In the latter case testing rates depend on the individual's income.

The decision whether an individual prefers to be tested or to remain untested depends on the comparison of expected lifetime utilities for both scenarios (Equations (12) and (13)). We show that a non-anticipating individual will always prefer to be tested if testing costs are negligible. Including anticipation in the utility function along with endogenous beliefs, on the other hand, makes it possible that individuals prefer to remain untested. We start by focusing on a non-anticipating individual. We first focus on the scenario in which the health shock arrives. In this case, the tested individual realizes lifetime utility $V_{s=1}$ and the untested individual realizes expected lifetime utility $E[V(b)_{s=1}]$ with $V_{s=1} \geq E[V(b)_{s=1}]$. This can be seen by inspecting the maximization problem. At the beginning of their lifetime individuals maximize expected utility over their life-cycle. Since in both the testing case and the non-testing case a negative health shock arrives, maximization is in both cases conditional on the same constraints. In other words when individuals are aware of the true information (i.e. health shock arrives), they already take decisions which maximize lifetime utility. Being exposed to incomplete information (i.e. individuals form beliefs about the probability of the health shock although it does not arrive) can only lead to lower or equal lifetime utility, because the incomplete information does not restrict the choice set of individuals and they could still take the same, utility maximizing decisions as when they are tested. This implies that $V_{s=1} \geq E[V(b)_{s=1}]$ holds and therefore the first component of $E[V_{tested}]$ (Equation (12)) is greater or equal compared to the first component of $E[V_{untested}]$ (Equation (13)), i.e. $pV_{s=1} \geq pE[V(b)_{s=1}]$ holds.

The same argument applies when we consider the no-shock scenario. Individuals realize lifetime utility $V_{s=0}$ if they are tested and expected lifetime utility $E[V(b)_{s=0}]$ if they remain untested. Tested individuals have a superior information set but the choice set is exactly the same as for untested individuals. Hence, $V_{s=0} \geq E[V(b)_{s=0}]$ holds and thus $pV_{s=0} \geq pE[V(b)_{s=0}]$. Combining both parts implies that $E[V_{tested}] \geq E[V_{untested}]$ always holds. In other words, non-anticipating individuals always prefer to be tested when testing costs are negligible.

For both comparisons it has been essential that individuals have exactly the same choice set and that the same choices would result in the same lifetime utility. This, however, does no

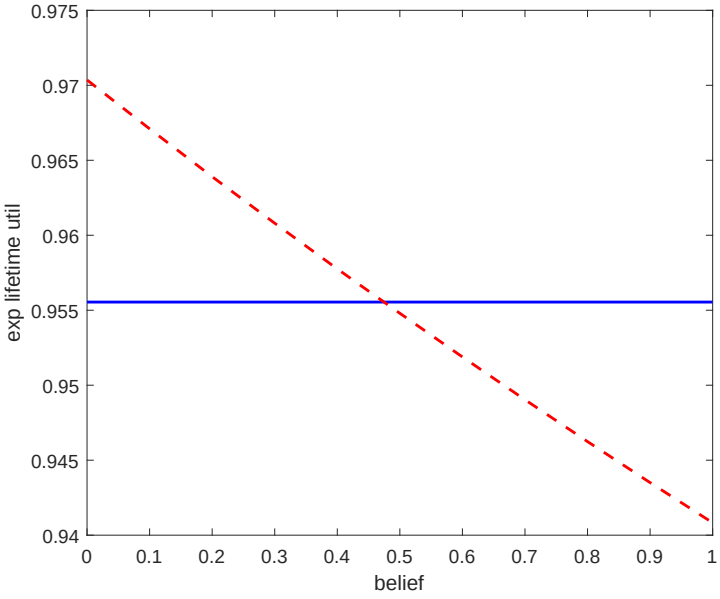
longer hold when anticipation of future health deficits is introduced into the model. In this case, anticipating or believing in future negative events already negatively affects instantaneous utility before the event sets in. We now redo the comparison from above for anticipating individuals. If the health shock arrives, anticipation adds a channel through which $E[V(b)_{s=1}]$ may be higher compared to $V_{s=1}$. If the individual is tested positive, the anticipation of the health shock reduces instantaneous utility already today. If the individual remains untested, the individual forms beliefs about the probability of the health shock. Consider, for example, an untested individual chooses to believe that the probability of a health shock to arrive is zero. In this case, the individual lives in denial of the negative shock and thus avoids the negative utility from anticipation. If this channel is quantitatively large enough to compensate the detrimental effect caused by having a smaller information set, $V_{s=1} \leq E[V(b)_{s=1}]$ holds. If the health shock does not arrive, anticipation adds an additional channel through which $E[V(b)_{s=0}]$ is lower compared to $V_{s=0}$. If untested, individuals may falsely believe that a negative health shock will occur which negatively affects their instantaneous utility before they realize that the health shock will in fact not materialize. Hence, $V_{s=0} \geq E[V(b)_{s=0}]$ still holds. Taken together this implies that there exists a b^* such that for all $b < b^*$, $E[V_{tested}] < E[V_{untested}]$ holds. In other words, anticipating individuals may prefer to remain untested, even when testing costs are negligible. This belief-induced behavioral phenomenon has been introduced as strategic ignorance in the behavioral literature (Benabou and Tirole, 2016).

4. Huntington's Disease

In this section, we first investigate anticipation and information avoidance in the context of HD. HD is a neurological disorder that is genetically inherited. Individuals with one parent carrying the expansion in the Huntington gene will eventually develop the disease with 50% probability. Although the cost for genetic testing is fairly small, the literature finds considerably low testing rates (below 10%) among individuals at risk, i.e. among individuals with a family background of HD. The onset of HD is around the age of 40 while death occurs on average 20 years later at the age of 60 (Oster et al. 2013a; Shoulson and Young, 2011). Therefore, we model a health shock at 40 such that the age at death of the anticipating type declines from 77.1 years to 60 years. In terms of our model, this amounts to an increase in the deficit level at 40 of $0.70 \cdot D_0$, i.e. 70% of the initial deficit level. Since there is a 50% chance of inheriting the

genetic expansion, the probability of the shock is $p = 0.5$. Figure 2 shows the expected lifetime utilities of the anticipating type associated with this health shock if being tested ($E[V_{tested}]$) and being untested ($E[V_{untested}]$) for different levels of beliefs, both relative to the lifetime utility of the no-shock scenario $V_{s=0}$.

Figure 2: HD and Beliefs



Blue (solid) line: tested. Red (dashed) line: untested. Exp lifetime util is expected lifetime utility relative to lifetime utility in the benchmark no-shock scenario $V_{s=0}$.

The blue (solid) line shows the expected lifetime utility in case the individual receives the test result while the red (dashed) line shows the expected lifetime utility in case the individual remains untested. If tested, the expected lifetime utility does not depend on beliefs since the test reveals the true state about the genetic disorder. If untested, the expected lifetime utility declines in the individual's beliefs about the future health shock. As the individual perceives the health shock more likely, the negative effect of anticipation on instantaneous utility increases and the relative gain from remaining untested shrinks. As can be seen in the figure, the threshold belief at which individuals start to prefer being tested lies slightly below 0.5. Furthermore, expected lifetime utility is maximized for $b = 0$, i.e. the individual chooses to believe that the probability for a health shock to arrive is zero. This is consistent with Oster et al. (2013a) who find in their study that individuals at risk for HD who refuse testing behave similarly to those who are certain not to carry the genetic expansion. In other words, individuals tend to live in denial although they are aware of their high risk of developing HD.

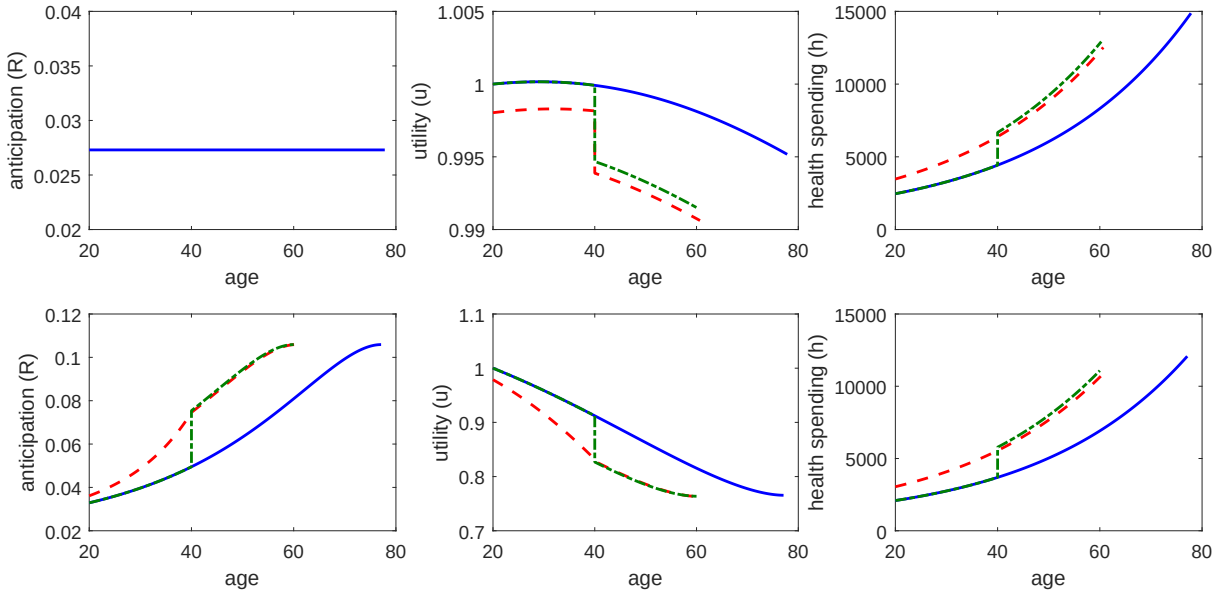
Figure 3 shows the results for $b = 0$ with respect to the anticipation stock, instantaneous utility, and health expenditure. Blue (solid) lines represent the benchmark run (i.e. when no health shock is considered), red (dashed) lines show the model response to the expected health shock (i.e. when being diagnosed), and green (dash-dotted) lines the results for the unexpected health shock (i.e. when remaining untested and choosing $b = 0$). The upper panels of Figure 3 show the non-anticipating individual and the lower panels show the anticipating individual.

Starting with the non-anticipating individual, we see that, by construction, the anticipation stock is constant at $R = D_0$. Since future health deficits do not enter instantaneous utility directly, the results for instantaneous utility look quite similar for the expected and unexpected health shock (the difference is around 0.1-0.2%). Note however, that individuals with an expected health shock experience instantaneous utility over a longer lifespan resulting in higher lifetime utility compared to individuals with an unexpected health shock. At age 40, in both cases utility declines on impact when HD sets in. The age trajectories for health expenditure, however, depend on whether the individual knows that it will become ill or not. If the disease is diagnosed at the age of 20, the individual immediately responds by increasing health investments at any age in order to counteract the upcoming health shock. If the individual does not know that it carries the genetic expansion, it behaves as in the benchmark case until age 40. When the disease sets in, non-diagnosed individuals increase health investments on impact above the level of the expected health shock scenario to compensate for the time in which they did not adjust their behavior.

Turning to the anticipating individual, the blue (solid) line in the first lower panel reiterates the benchmark run for the anticipation stock of the anticipating type which we already analyzed in Figure 1. Looking at the unexpected health shock, the anticipation stock coincides with the benchmark run until the age of 40 and then increases on impact when HD sets in. If the individual is diagnosed with HD at the age of 20, however, the anticipation stock increases gradually already from the beginning as the knowledge of the future evolution of HD-related health deficits enters the anticipation variable. Since anticipation affects utility, this also translates into the age trajectories for instantaneous utility illustrated in the second lower panel.

Again, for the unexpected shock utility decreases on impact when the disease sets in at the age of 40. If the individual learns about the HD diagnosis at age 20, utility declines already right from the beginning due to the disutility caused by anticipating the future health shock.

Figure 3: Expected vs. Unexpected Health Shocks



Blue (solid) lines: benchmark (no shock). Red (dashed) lines: expected shock. Green (dash-dotted) lines: unexpected shock. The upper panels show results for the non-anticipating type and the lower panels for the anticipating type. Utility is instantaneous utility relative to initial utility of the benchmark run (no shock).

At age 40, utility additionally jumps down on impact following the sudden increase in actual health deficits. Since instantaneous utility is much lower before the disease sets in for individuals experiencing the expected shock, lifetime utility is lower compared to those who do not expect the shock. In contrast to non-anticipating individuals, this cannot be compensated for by a longer lifespan of individuals expecting the shock compared to those not expecting the shock. The last panel shows the effects of the health shock on health investments which are qualitatively similar to those of the non-anticipating type.

Table 3 shows the quantitative results for the two outcome variables life expectancy and expected lifetime utility or welfare for the tested and non-tested case. The first pair of columns includes the shocks for the non-anticipating type while the second pair looks at the anticipating individual. The numbers are provided as percentage deviation from the benchmark run without any health shock. In all cases, the reduction in life expectancy amounts to around 15%, which in absolute terms implies a decrease of around 8.6 years. This is the weighted average between the HD scenario as calibrated above (the difference between longevity of 77.1 years and longevity of an individual with HD of 60 years) and the no-shock scenario in which longevity remains unchanged.

Interestingly, in case of the non-anticipating type, there is only a small difference between the testing and non-testing scenario in terms of welfare. Although in the non-testing case the individual can only start re-optimizing at the age of 40, he experiences virtually the same lifetime utility as when the shock is known at the age of 20. By construction, welfare is still higher for tested individuals. This picture changes, however, when we look at the anticipating type. Opting for the genetic test at the age of 20 results in a 1.5 percent lower expected lifetime utility than if the individual remains untested. Consequently, the individual would choose to avoid being tested and rather live an additional 20 years without the potential burden of knowing that HD will eventually set in.

Table 3: HD at age 40: Impact on Life Expectancy and Welfare

	no anticipation		anticipation	
outcome	untested	tested	untested	tested
1) LE	-15.17	-14.82	-14.97	-14.60
2) Welfare	-2.56	-2.55	-2.96	-4.45

The values are deviations in percent from the benchmark run without health shock; LE denotes life expectancy at age 20 and Welfare denotes expected lifetime utility.

With our model at hand, we can also determine the willingness to pay to avoid testing or, in other words, the monetary compensation value for undergoing the test. For this purpose, we calculate the difference in the Value of Life (VoL) between choosing and refusing the test. The VoL converts lifetime utility measured in "utils" into monetary equivalents and is given by the expression $VOL = \int_0^T \mathrm{e}^{-\rho\tau} u[c(\tau), D(\tau), R(\tau)]\mathrm{d}\tau)/u_c[c(0), D(0), R(0)]$ where u_c denotes the marginal utility of consumption. The VoL of our reference anticipating individual is 8.73 million dollars. This value is in line with empirical estimates which find the VoL to range from $ 7 million (Murphy and Topel, 2006, Fig. 3) to $ 10 million (Moran and Monje, 2016). Calculating the welfare difference between the testing and non-testing scenario, we find that the individual needs a compensation of $ 129,266 in order to be indifferent between testing and non-testing. In other words, the individual would forgo around five annual wages to lead an unburdened life until age 40. We also calculate the willingness to pay of the non-anticipating individual for being tested which amounts to 1,321 dollars.

5. Breast and Ovarian Cancer

Our second experiment refers to genetic testing for breast and ovarian cancer. 5-10% of all breast cancer cases and 10-15% of all ovarian cancer cases are hereditary, meaning that they are caused by a gene defect inherited by a parent. One of the most researched genetic disorders is given by the defect of BRCA (Breast Cancer) gene 1 and 2 which is inherited with a probability of 50% if one parent carries the genetic defect. Although the detection of the genetic expansion is not a perfect predictor whether the disease will be eventually developed, recent evidence suggests that women with BRCA1 (BRCA2) mutation face a risk of developing breast or ovarian cancer before age 80 of 72% (69%) and 44% (17%), respectively (Kuchenbaecker et al., 2017).

Despite these high cancer risks, the testing rate of high-risk members of families with hereditary breast or ovarian cancer only lies at around 60% (Lerman et al., 1996; Ropka et al., 2006). This observation is even more surprising when considering the treatment options available for high-risk individuals. Prophylactic bilateral mastectomy (the removal of both breasts) and prophylactic bilateral salpingo-oophorectomy (the removal of both ovaries along with fallopian tubes) decrease the risk of breast and ovarian cancer almost entirely by 90-95% (Metcalfe et al., 2004; Rebbeck et al., 2004) and 80-90% (Finch et al., 2006; Kauff et al., 2002; Rebbeck et al., 2002). Also, pre-menopausal salpingo-oophorectomy is found to further reduce the risk of breast cancer (Eisen et al., 2005; Metcalfe et al., 2004, Rebbeck et al., 2004).

There is empirical evidence that household income is negatively correlated with testing for the BRCA gene (e.g. Stenehjem et al., 2018; Lerman et al., 1996). In the study by Lerman et al. (1996), the test was offered for free so that testing costs cannot explain this observation. As we will illustrate, our model provides an argument for non-testing by showing that testing behavior depends on the socioeconomic status of the individual. If individuals are endowed with sufficiently low income, they refuse to be tested even if effective treatment is available and the genetic test is free of cost.

Since the baseline calibration of the model refers to men, we start by recalibrating the model for women. We assume that women have the same anticipation parameters as men and estimate the remaining preference parameters to fit life-cycle health investments (MEPS, 2010) and an average life expectancy of women at 20 of 61.9 years (i.e. death at 81.9 years; NVSS, 2014). The parameter estimates are summarized in Table 4.

Table 4a: Calibration Results

ρ	σ	α	A
0.063	1.38	0.13	0.00139

Table 4b: Externally Set Parameters

D_0	D_T	μ	a	γ	θ	β	w	r
0.0381	0.1428	0.031	0.013	0.19	0.10	0.22	17303	0.07

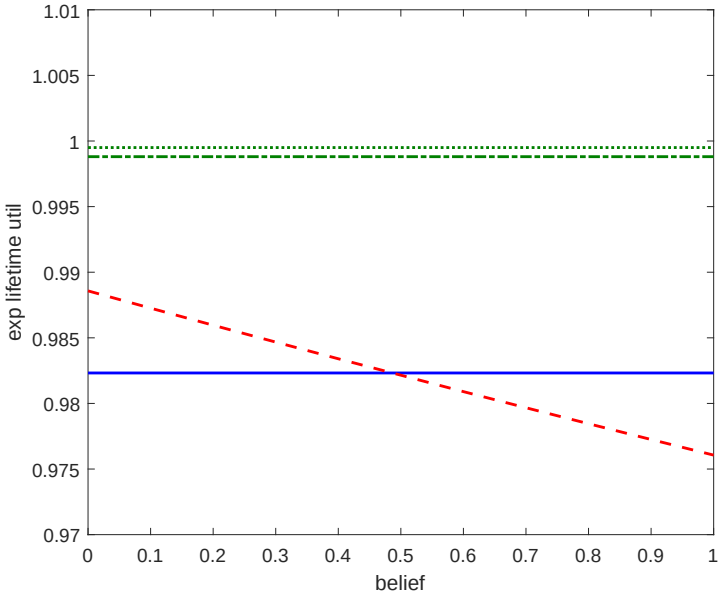
Comparing the parameter values to those of men, we find that women are more patient (smaller ρ), more risk-averse (higher σ), and value health to a higher degree (higher α) which is in line with vast empirical evidence (Cohen and Einav, 2007; Croson and Gneezy, 2009; Read and Read, 2004; Sunden and Surette (1998); Waldron, 1985; Wardle et al., 2004; see Schünemann et al. (2017b) for a detailed discussion on gender-specific parameter estimates).

According to a simulation study by Sigal et al. (2012), average life expectancy at 30 of women who carry the BRCA 1 mutation is 41.5 years (i.e. death at 71.5 years) when no treatment is applied. When carrying the BRCA 1 mutation, breast and ovarian cancer set in 20 and 10 years earlier than in the average population, i.e. at 42 and 52, respectively (Brose et al., 2002). Therefore, we design an experiment in which the health shock sets in at the age of 47 (the average age of onset of breast and ovarian cancer) and which leads to death of the individual at the age of 71.5 years. In model terms, this shock amounts to an increase in health deficits at age 47 of $0.41 * D_0$, i.e. 41% of the initial deficit level.

In order to be consistent with the stylized facts on the age at treatment, we assume that the treatment sets in at age 30. At this age, we can observe an uptake in the prophylactic measures discussed above (Metcalfe et al., 2019). Depending on the residential location and type of reconstructive measures after the surgery, the combined cost of prophylactic bilateral mastectomy and salpingo-oophorectomy ranges from $ 20,000–50,000 (Grann et al., 2011; Mattos et al. (2015); MD, 2020). We will report results for both the lower and upper bound of this range. Given the high efficacy of the prophylactic measures in reducing the risk of breast and ovarian cancer, we assume that treatment prevents the genetically-induced health shock completely. Therefore, the only utility loss that the treated individual experiences compared

to the benchmark no-shock scenario relates to the cost of the treatment[6]. Figure 4 shows the expected lifetime utilities for the tested (blue solid line), untested (red dashed line), and treated scenario (green dotted (dash-dotted) line for low-cost treatment of $p_s h_s = 20,000$ (high-cost treatment of $p_s h_s = 50,000$)) for various beliefs of the representative individual.

Figure 4: Breast and Ovarian Cancer and Beliefs



Blue (solid) line: tested. Red (dashed) line: untested. Green (dotted) line: treated low cost. Green (dash-dotted) line: treated high cost. Exp lifetime util is expected lifetime utility relative to lifetime utility in the benchmark no-shock scenario $V_{s=0}$.

If no treatment was available, the figure qualitatively mimics the HD experiment. The individual would choose belief $b = 0$ and decide to not undergo the test. If effective treatment for the individual is available, however, the individual chooses to be tested and treated if the test result is positive. The upper part of Table 5 summarizes the quantitative results for the untested, tested, and treated case (for $b = 0$). Rows 1 and 2 refer to the low-cost treatment while rows 3 and 4 refer to the high-cost treatment.

The genetic defect reduces life expectancy by around 8% if untreated which implies an absolute reduction of around 5 years as calibrated above (recall that the defect of the BRCA 1 gene decreases life expectancy by around 10 years and the probability of inheriting the gene is 50%). Undergoing treatment almost re-establishes benchmark life expectancy. The 0.10% (0.26%) loss

[6]We acknowledge other costs related to these highly invasive surgeries with regard to body image, reproductive or other personal preferences, or the quality of life. Further, Fang and Wang (2015) show that in the presence of hyperbolic discounting, present bias and naivety may induce individuals to not undergo mammography. In our experiment, we focus on the monetary cost of the treatment.

Table 5: Breast and Ovarian Cancer: Impact on Life Expectancy and Welfare

	no anticipation			anticipation		
outcome	untested	tested	treated	untested	tested	treated
Average wage w						
1) LE	-8.30	-8.23	-0.10	-8.27	-8.19	-0.11
2) Welfare	-1.04	-1.04	-0.03	-1.14	-1.77	-0.05
3) LE	-8.30	-8.23	-0.26	-8.27	-8.19	-0.26
4) Welfare	-1.04	-1.04	-0.09	-1.14	-1.77	-0.12
Threshold wage w^*						
5) LE	-7.68	-7.64	-0.71	-7.67	-7.63	-0.67
6) Welfare	-1.11	-1.11	-1.11	-1.15	-1.72	-1.15
7) LE	-7.82	-7.77	-1.09	-7.80	-7.76	-1.01
8) Welfare	-1.09	-1.09	-1.09	-1.15	-1.73	-1.15

The values are deviations in percent from the benchmark run without health shock; LE denotes life expectancy at age 20 and Welfare denotes expected lifetime utility. The upper part of the table shows results for the average wage while the lower part shows results for the threshold wage. Rows 1 and 2 as well as 5 and 6 refer to the low-cost treatment while rows 3 and 4 as well as 7 and 8 refer to the high-cost treatment.

shown in the table stems from the fact that the treatment cost of $ 20,000 ($ 50,000) crowds out aging-related health investments and thus accelerates the accumulation of aging-related deficits. Both the non-anticipating and the anticipating type choose the treatment option since the welfare loss is comparably small (0.03% (0.05%) and 0.05% (0.12%), respectively). If treatment was not available, the same argument as in the HD case applies. While the non-anticipating type would undergo testing to re-optimize behavior unrelated to the cancer treatment (welfare difference not visible in the table due to rounding), the anticipating type would choose to remain untested to minimize the welfare reduction from the potential health shock ($-1.14\% > -1.77\%$).

The upper part of Table 6 shows the associated differences in the VoL (the willingness to pay) relative to the untested case for the low-cost treatment (row 1) and the high-cost treatment (row 2). Depending on the treatment cost, the non-anticipating type would pay $ 160,320 or $ 152,034 (nine annual wages) for receiving a test result if treatment is available and $ 195 if treatment is not available. The anticipating type would pay $ 182,308 or $ 170,158 (ten annual wages) to be tested if treatment was available, while the individual would pay $ 104,083 (six annual wages) to avoid testing if treatment was not available.

Table 6: Δ Value of Life (in $)

	no anticipation		anticipation	
outcome	tested-untested	treated-untested	tested-untested	treated-untested
Average wage w				
1) Δ VoL	195	160,320	-104,083	182,308
2) Δ VoL	195	152,034	-104,083	170,158
*Threshold wage w**				
3) Δ VoL	7	0	-4,212	0
4) Δ VoL	17	0	-12,240	0

Δ VoL refers to the difference in expected lifetime utility expressed in dollars. The upper part of the table shows results for the average wage while the lower part shows results for the threshold wage. Rows 1 and 3 refer to the low-cost treatment while rows 2 and 4 refer to the high-cost treatment.

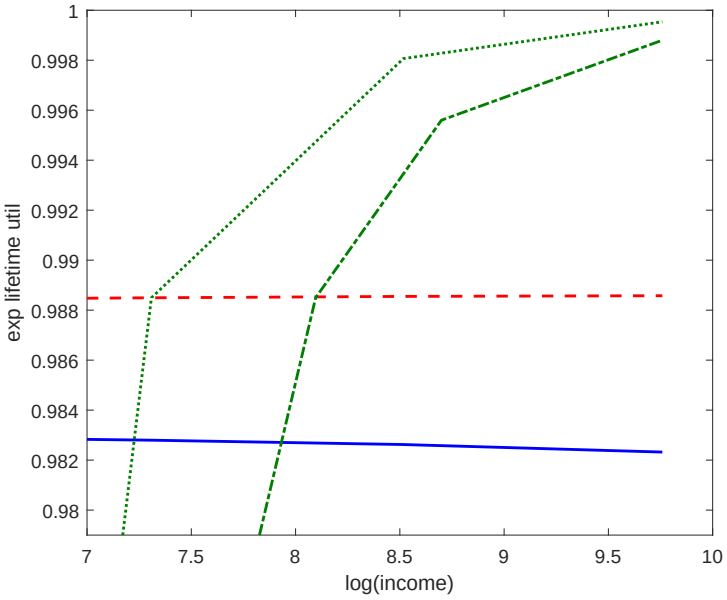
According to our model, a U.S. American single female endowed with the average wage would always choose to be tested if treatment was available. As discussed above, however, only 60% of the high-risk individuals opt for a genetic test. Possible explanations for this observation may be the highly invasive nature of the treatment procedure or insufficient information on risk and treatment benefits. Our model suggests that part of the non-testing can be explained by income. The channel works as follows. As wages of the individual decrease, the cost of the surgery becomes a more and more unbearable burden. In addition to the pure income effect, the concavity of the utility function aggravates this mechanism. The poorer the individual gets, the more hurtful it becomes to forgo instantaneous consumption in order to finance the treatment. At some point, treatment becomes too costly in terms of welfare. While the non-anticipating type would still undergo testing to potentially re-optimize behavior other than the treatment, the non-anticipating type refuses the test due to the potential cost of anticipation and chooses to live in denial ($b = 0$).

This mechanism is shown in Figure 5 which depicts expected lifetime utility for various wages (up to the average wage) for $b = 0$. Again, the blue (solid) line shows expected lifetime utility for the tested scenario, the red (dashed) line for the untested scenario, and green (dotted (low cost), dash-dotted (high cost)) lines for the treated scenario, all relative to the respective benchmark no-shock scenario. As can be seen in the figure, income has little effect on the relative welfare reduction in the tested and untested case. For all incomes, the non-testing scenario leaves the

anticipating type with a higher expected lifetime utility than the testing scenario due to the anticipation effect. The treatment case, however, becomes less and less attractive in terms of welfare when wages decrease. The threshold wage at which the individual would rather remain untested than paying for the treatment if tested positive amounts to approximately $ 1,500 in the low cost case and $ 3.300 in the high cost case. At this wage, expected lifetime utility in the treatment case becomes lower than the expected lifetime utility if untested. The individual then chooses to remain untested to avoid disutility from anticipation.

We calculate the effects on welfare and life expectancy when the woman is endowed with the threshold wage. Results are shown in the lower part of Table 5 in which rows 5 and 6 refer to the low cost case and rows 7 and 8 refer to the high cost case. The non-anticipating woman

Figure 5: Breast and Ovarian Cancer: Income



Blue (solid) line: tested. Red (dashed) line: untested. Green (dotted) line: treated low cost. Green (dashed-dotted) line: treated high cost. Exp lifetime util is expected lifetime utility relative to lifetime utility in the respective benchmark no-shock scenario $V_{s=0}$.

would still choose to be tested to adjust her health and consumption behavior other than the treatment (not visible in the table due to rounding). The anticipating woman, on the other hand, would remain untested since the potential utility loss from anticipating the disease would leave her with lower expected lifetime utility if tested ($-1.72 < -1.15$). We also calculate the willingness to pay regarding the testing decision in this case (lower part of Table 6). While the non-anticipating would (slightly) prefer to receive the test results, the anticipating type would

pay $ 4,212 or $ 12,240 (three or four annual wages) to avoid the test depending on the treatment cost.

The calculated threshold wages appear low at first glance. Note however that this wage represents an average wage paid throughout the entire lifetime and not only during working age.[7] Furthermore, the wage is only part of total income since in our model the individual also receives capital income. According to the consumer expenditure survey, in 2010 the average income in the lowest decile of the single female income distribution amounts to $ 2136 (BLS, 2012). Acknowledging the potentially strong selection effects in the data, our model implies that single females at this lower end of the income distribution would prefer to remain untested. Another way to interpret our results is to look at the share of females that depend on some sort of means-tested social welfare programs. By definition of means-tested program eligibility, the recipients of those programs would find it hard to pay for the treatment cost, implying that they would remain untested to avoid the potential burden of anticipation. According to Irving and Loveless (2015), in 2010, the average monthly participation rate in these programs amounted to around 20 percent of the non-institutionalized population in the U.S. For female households (i.e. households with no husband present), the participation rate was even higher at almost 50%. These numbers suggest that there exist a considerable number of households for which treatment costs constitute an unbearable welfare burden, implying that these individuals avoid to be tested in the first place.

6. Conclusion

In this paper we set up a gerontologically founded stochastic life-cycle model of human aging in which individuals form beliefs about future health shocks that directly affect currently experienced instantaneous utility. We calibrated the model to a 20-year-old U.S. American and applied our model to contribute to the literature on information avoidance in the context of health. We illustrated that anticipatory feelings are a quantitatively powerful explanation for why people refuse to be tested even if testing costs are low. For our benchmark case, we estimated that individuals at the age of 20 would forgo around five annual wages to avoid a Huntington's disease diagnosis. With the example of breast and ovarian cancer, we also provided an explanation for why testing rates are far from universal when effective treatment is available. If income of women

[7]This is no drawback in our model since through the capital market only the present value of lifetime income matters.

falls below a certain threshold, the cost of treatment becomes too hurtful in terms of expected lifetime utility. If treatment is not feasible, women decide to remain untested since they want to avoid the disutility from anticipation following a potentially positive test result. Our model thus suggests that testing behavior depends on the socioeconomic status of the individual.

Our framework of anticipation also has implications for policy. Although information avoidance may be privately optimal, i.e. from an individual perspective, higher testing rates are likely to be socially optimal when there are externalitites. External effects become most obvious when we think about contagious diseases. Like Oster et al. (2013b) report for the case of HD, Thornton (2008) and Weinhardt et al. (1999) find that a positive HIV diagnosis changes behavior (higher condom use), but that a negative test result leaves behavior unchanged. Combining this feature with low testing rates for HIV (Thornton et al, 2008), the transmission risk of HIV generates a huge potential for social cost due to test avoidance. But also other diseases bear the risk of social costs that may not be internalized in individual calculus. In the case of HD, for example, the disease can be further inherited to children of carriers. Low testing rates for hereditary cancer screening imply that treatment costs are expected to soar when the detection of the disease is delayed.

Our model could be applied to analyze other life-cycle decisions. A natural model extension would be to investigate unhealthy consumption patterns in the light of anticipation. As Oster et al. (2013b) point out, being diagnosed with Huntington's disease reduces the probability to quit smoking. Moreover, anticipation of future health deficits may also play a role for the retirement decision since anticipating individuals are expected to have a shorter working life.

Appendix A: Derivation of the Euler Equations

The first-order conditions associated with the optimal control problem read:

$$\frac{\partial \mathcal{H}}{\partial c} = 0 \Leftrightarrow \lambda_k = \left(\frac{D_0}{D}\right)^{\alpha} \left(\frac{D_0}{R}\right)^{\beta} c^{-\sigma} \tag{A.1}$$

$$\frac{\partial \mathcal{H}}{\partial h} = 0 \Leftrightarrow p\lambda_k = -\lambda_D \mu A \gamma h^{\gamma-1} \tag{A.2}$$

$$\begin{aligned} \frac{\partial \mathcal{H}}{\partial D} &= -\dot{\lambda}_D + \lambda_D \rho \\ \Leftrightarrow \frac{\dot{\lambda}_D}{\lambda_D} &= \rho - \mu + \frac{\alpha}{D\lambda_D} u(c, D, R) \end{aligned} \tag{A.3}$$

$$\begin{aligned} \frac{\partial \mathcal{H}}{\partial k} &= -\dot{\lambda}_k + \lambda_k \rho \\ \Leftrightarrow \frac{\dot{\lambda}_k}{\lambda_k} &= \rho - r. \end{aligned} \tag{A.4}$$

Log-differentiating (A.1) w.r.t. time and using (A.4) provides:

$$\begin{aligned} \frac{\dot{\lambda}_k}{\lambda_k} &= -\alpha \frac{\dot{D}}{D} - \beta \frac{\dot{R}}{R} - \sigma \frac{\dot{c}}{c} \\ \Leftrightarrow \rho - r &= -\alpha \frac{\dot{D}}{D} - \beta \frac{\dot{R}}{R} - \sigma \frac{\dot{c}}{c}. \end{aligned} \tag{A.5}$$

Solving (A.5) for consumption growth provides equation (9) in the main text.

Log-differentiating (A.2) w.r.t. time and using (A.3) and (A.4) we obtain:

$$\begin{aligned} \frac{\dot{\lambda}_k}{\lambda_k} &= \frac{\dot{\lambda}_D}{\lambda_D} + (\gamma - 1)\frac{\dot{h}}{h} \\ \Leftrightarrow \rho - r &= \rho - \mu + \frac{\alpha}{D\lambda_D} u(c, D, R) + (\gamma - 1)\frac{\dot{h}}{h}. \end{aligned} \tag{A.6}$$

Using (A.1) and (A.2) and solving (A.6) for health expenditure growth provides equation (10) in the main text.

7. Appendix B: Solution Method

We describe the solution method for solving the model in case the individual does not get tested and for given believes $0 \leq b \leq 1$. In a second step we then optimize over b to find the beliefs which maximize lifetime-utility. For the case that the individual gets tested, we apply the same solution procedure and set $b = 0$ or $b = 1$, depending on the test result.

We start by deriving the following dynamic system

$$\dot{D} = \mu(D - Ah^{\gamma} - a) \tag{15a}$$

$$\dot{R} = \theta(R - D) \tag{15b}$$

$$\dot{k} = w + rk - c - ph \tag{15c}$$

$$\dot{\lambda}_D = -\lambda_D(\rho - \mu) + \frac{\alpha}{D}u(c, D, R) \tag{15d}$$

$$\dot{\lambda}_k = \lambda_k(\rho - r) \tag{15e}$$

which holds for both intervals $[0, \tilde{t})$ and $[\tilde{t}, T]$. While for the first interval prior to the realization of the shock the variables follow a unique path, for the second interval we have to distinguish the cases of $s = 0$ and $s = 1$. Therefore, variables relating to the second interval are indexed accordingly.

The system is complemented by initial, interior, and final boundary conditions. Initial boundary conditions for $t = 0$ are given by $D(0) = D_0$ and $k(0) = 0$. In case the individual faces treatment costs we reduce $k(0)$ by an amount equivalent in net present value terms to the costs at the point of time when they accrue.

Interior boundary conditions are given by

$$\lambda_k(\tilde{t}^-) = b\lambda_{k,s=1}(\tilde{t}^+) + (1-b)\lambda_{k,s=0}(\tilde{t}^+) \tag{16a}$$

$$\lambda_D(\tilde{t}^-) = b\lambda_{D,s=1}(\tilde{t}^+) + (1-b)\lambda_{D,s=0}(\tilde{t}^+) \tag{16b}$$

$$R(\tilde{t}^-) = bR_{s=1}(\tilde{t}^+) + (1-b)R_{s=0}(\tilde{t}^+) \tag{16c}$$

$$k(\tilde{t}^-) = k_{s=0}(\tilde{t}^+) \tag{16d}$$

$$k(\tilde{t}^-) = k_{s=1}(\tilde{t}^+) \tag{16e}$$

$$D(\tilde{t}^-) = D_{s=0}(\tilde{t}^+) \tag{16f}$$

$$D(\tilde{t}^-) = D_{s=1}(\tilde{t}^+) + \tilde{D}\,. \tag{16g}$$

Equations (16a)-(16c) are the interior boundary conditions for the forward looking variables described in section 2.2. Equations (16d) and (16e) state that the capital stock evolves continuously, independent of the realization of the shock. Equation (16f) states that health deficits

evolve continuously in case no shock sets in, and Equation (16g) states that health deficits D are reduced by $\tilde{D}$ in response to the health shock.

Finally, the final boundary conditions require that $\mathcal{H}(T) = 0$, $R(T) = D_T$, and $k(T) = 0$ hold, both for $s = 0$ and $s = 1$.

To solve for the optimal life-cycle trajectories we apply a shooting algorithm.[8] This type of algorithm is frequently used to solve differential equations for which only some of the initial conditions are given and additionally a set of interior and final boundary conditions has to be satisfied, i.e. the problem is a multi-point boundary value problem. The general idea of shooting is to guess the unknown initial values of the variables and calculate a trial solution by integrating the dynamic system for a given time span. Then, the initial values are updated in an iteration process until the remaining boundary conditions are met as well. We have to adapt the standard shooting procedure to our setting, because we have two time intervals connected by interior boundary conditions and the length of the total time span T is endogenous.

The initial guess for the iteration process consists of those variables at $t = 0$, which are not given by initial conditions and those variables at $t = \tilde{t}$, which are not explicitly given by interior boundary conditions. This means that we provide an initial guess for $R(0)$, $\lambda_D(0)$, $\lambda_k(0)$, $\lambda_{k,s=0}(\tilde{t}^+)$, $\lambda_{k,s=1}(\tilde{t}^+)$, $\lambda_{D,s=0}(\tilde{t}^+)$, $\lambda_{D,s=1}(\tilde{t}^+)$, $R_{s=0}(\tilde{t}^+)$, and $R_{s=1}(\tilde{t}^+)$. We then solve system (15) with the standard Matlab routine for initial value problems (`ode45.m`) until $\tilde{t}$ and using part of the initial guess also for the interval $(\tilde{t}, T]$ until the individual dies at D_T. This implies that the lifetime T is determined by $D(T) = D_T$ separately for $s = 0$ and $s = 1$, respectively. There are, however, a number of conditions that the trial solution does not satisfy: The transversality condition $\mathcal{H}(T) = 0$ and the final boundary conditions $R(T) = D_T$ and $k(T) = 0$ (both for $s = 0$ and $s = 1$), and the interior boundary conditions (16a)-(16c). Because the dimension of the initial guess and the number of conditions to be met both sum up to nine, the problem is well-defined. We then adjust the initial conditions until these nine conditions are met by using a Newton-Raphson algorithm.

[8] This part of the method builds on the numerical method used in Schünemann et al. (2020).

References

Alvarez-Cuadrado, F., Monteiro, G., and Turnovsky, S. J. (2004). Habit formation, catching up with the Joneses, and non-scale growth. *Journal of Economic Growth* 9(1), 47-80.

Arking, R. (2006). *The Biology of Aging: Observations and Principles*, Oxford University Press, Oxford.

Bénabou, R. and Tirole, J. (2016). Mindful Economics: The Production, Consumption, and Value of Beliefs. *Journal of Economic Perspectives* 30(3), 141–164.

BLS (2012). Consumer Expenditure Survey. U.S. Bureau of Labor Statistics, September 2012 (`http://www.bls.gov/cex/csxcross.htm#y1011`).

Brose M. S. ,Rebbeck T. R., Calzone K. A. , Stopfer J. E., Nathanson K. L., and Weber B. L. (2002). Cancer Risk Estimates for BRCA1 Mutation Carriers Identified in a Risk Evaluation Program. *Journal of the National Cancer Institute* 94(18), 1365-1372

Browning, M., and Ejrnaes, M. (2009). Consumption and children. *The Review of Economics and Statistics* 91(1), 93-111.

Bryson, A. E., and Ho, Y. C. (1975). Applied optimal control: optimization, estimation, and control [M]. *Washington, DC: Hemisphere.* Brunnermeier, Markus, K., and Jonathan A. Parker. 2005. Optimal expectations. *American Economic Review*, 95 (4): 1092-1118.

Caplin, Andrew, and Leahy, John (2001). Psychological expected utility theory and anticipatory feelings. *Quarterly Journal of Economics* 116(1), 55-79.

Caplin, Andrew, and Leahy, John (2001). The supply of information by a concerned expert. *Economic Journal* 114(497), 487-505.

Carroll, C. D., Overland, J.R., and Weil, D. N. (1997). Comparison utility in a growth model. *Journal of Economic Growth* 2, 339-67.

Case, A., and Deaton, A.S. (2005). Broken down by work and sex: How our health declines. In: *Analyses in the Economics of Aging*, University of Chicago Press, 185-212.

CES (2017). Consumer Expenditure Survey. U.S. Bureau of Labor Statistics. https://www.bls.gov/cex/2016/CrossTabs/singlesbyinc/malesinc.PDF

Chetty, R. (2006). A new method of estimating risk aversion. *American Economic Review* 96, 1821-1834.

Cohen, A. and Einav, L. (2007). Estimating risk preferences from deductible choice. *American Economic Review* 97(3), 745-788.

Croson, R. and Gneezy, U. (2009). Gender differences in preferences. *Journal of Economic Literature* 47(2), 448-74.

Cuypers, M., Lamers, R.E.D., Cornel, E.B., van de Poll-Franse, L.V., de Vries, M., and Kil, P.J.M. (2017). The impact of prostate cancer diagnosis and treatment decision-making on

health-related quality of life before treatment onset. *Support Care Cancer*, University of Chicago Press, 185-212.

Dalgaard, C-J., and Strulik, H. (2014). Optimal aging and death: Understanding the Preston Curve. *Journal of the European Economic Association* 12, 672-701.

Dalgaard, C-J., and Strulik, H. (2017). The genesis of the golden age – accounting for the rise in health and leisure. *Review of Economic Dynamics* 24, 132-151.

Eisen A., Lubinski J., Klijn J., Moller P., Lynch H. T., Offit K., et al. (2005). Breast cancer risk following bilateral oophorectomy in BRCA1 and BRCA2 mutation carriers: an international case-control study. *Journal of Clinical Oncology* 23, 7491–7496

Fang, H. and Wang, Y. (2015). Estimating dynamic discrete choice models with hyperbolic discounting, with an application to mammography decisions. *International Economic Review* 56, 565-596

Fels (2015). On the value of information: Why people reject medical tests. *Journal of Behavioral and Experimental Economics 56*, 1-12.

Finch A., Beiner M., Lubinski J., Lynch H. T., Moller P., Rosen B., et al. (2006) Salpingo-oophorectomy and the risk of ovarian, fallopian tube, and peritoneal cancers in women with a BRCA1 or BRCA2 Mutation. *Journal of the American Medical Association* 296, 185-192.

Finkelstein, A., Luttmer, E.F., and Notowidigdo, M.J. (2013). What good is wealth without health? The effect of health on the marginal utility of consumption. *Journal of the European Economic Association* 11(1), 221-258.

Grann, V. R., Patel P. R., Jacobson, J. S. et al. (2011). What good is wealth without health? The effect of health on the marginal utility of consumption. *Breast Cancer Research and Treatment* 125(3), 837–847.

Grossman, M. (1972). On the concept of health capital and the demand for health. *Journal of Political Economy* 80, 223-255.

Honiden, S., Sundaram, V., Nease, R.F., Holodniy, M., Lazzeroni, L.C., Zolopa, A., and Owens, D.K. (2006). The effect of diagnosis with HIV infection on health-related quality of life. *Quality of Life Research* 15, 69-82.

Jorda, O., Knoll, K., Kuvshinov, D., Schularick, M., and Taylor, A.M. (2019). The Rate of Return on Everything, 1870-2015. The Quarterly Journal of Economics 134 (3), 1225-1298.

Kauff N. D., Satagopan J. M., Robson M. E., Scheuer L., Hensley M., Hudis C. A., et al. (2002) Risk-reducing salpingo-oophorectomy in women with a BRCA1 or BRCA2 mutation. *The New England Journal of Medicine* 346, 1609–1615

Koszegi, B. (2003). Health anxiety and patient behavior. *Journal of Health Economics* 22, 1073-1084.

Koszegi, B. (2006). Emotional agency. *Quarterly Journal of Economics* 121(1), 121-155.

Kuchenbaecker K. B., Hopper J. L., Barnes D. R., et al. (2017). Risks of Breast, Ovarian, and Contralateral Breast Cancer for BRCA1 and BRCA2 Mutation Carriers. *Journal of the American Medical Association* 317(23), 2402–2416.

Layard, R., Mayraz, G., and Nickell, S. (2008). The marginal utility of income. *Journal of Public Economics* 92, 1846-1857.

Lazarus, R. (1966). Psychological Stress and the Coping Process. *New York: McGraw Hill.*

Lerman, C., Narod, S., Schulman, K., Hughes C., Gomez-Caminero A., Bonney G., Gold K., et al. (1996). BRCA1 Testing in Families with Hereditary Breast-Ovarian Cancer. A Prospective Study of Patient Decision Making and Outcomes. *Journal of the American Medical Association* 275(24), 1885-1892.

Loewenstein, G. (1987). Anticipation and the valuation of delayed consumption. *Economic Journal* 97, 666-684.

Irving, S. K., and Loveless, T. A. (2015). Dynamics of Economic Well- Being: Participation in Government Programs, 2009–2012, *Household Economic Studies*, Report no. P70–141.

Mattos, D., Gfrerer, L., Reish, R., Hughes, K., Cetrulo, C., Colwell, A., Yaremchuk, M., Austen, W., and Liao, E.. (2015). Lifetime Costs of Prophylactic Mastectomies and Reconstruction versus Surveillance. *Plastic and Reconstructive Surgery*136(6), 730e-740e.

MD (2020). MDsave. https://www.mdsave.com/procedures/double-mastectomy/d78af4cb and https://www.mdsave.com/procedures/ovary-removal-salpingoectomy-oophorectomy/d78bfacb (retrieved on March 24, 2020)

Metcalfe, K., Eisen, A., Senter, L. et al. (2019) International trends in the uptake of cancer risk reduction strategies in women with a BRCA1 or BRCA2 mutation. *British Jornal of Cancer* 121, 15–21.

Metcalfe K., Lynch H. T., Ghadirian P., Tung N., Olivotto I., Warner E., et al. (2004) Contralateral breast cancer in BRCA1 and BRCA2 mutation carriers. *Journal of clinical oncology* 22, 2328–2335.

MEPS (2010), U.S. Department of Health & Human Services. Center for Financing, Access and Cost Trends, Agency for Healthcare Research and Quality: Medical Expenditure Panel Survey, 2010 (`https://meps.ahrq.gov/mepsweb/data_stats/quick_tables_results.jsp?component=1&subcomponent=0&year=-1&tableSeries=1&searchText=&searchMethod=1&Action=Search`)

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002). The accumulation of deficits with age and possible invariants of aging. *Scientific World* 2, 1816-1822.

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002b). The mortality rate as a function of accumulated deficits in a frailty index. *Mechanisms of ageing and development* 123, 1457-1460.

Monteiro, G. and Turnovsky S.J. (2016). Anticipated consumption and its impact on capital accumulation and growth: "Forward-looking" versus "backward-looking" consumption reference. *International Journal of Economic Theory* 12, 203-232.

Moran, Molly J. and Monje, Carlos (2016). Guidance on Treatment of the Economic Value of Statistical Life (VSL) in U.S. Department of Transportation Analyses - 2016 Adjustment. U.S. Department of Transportation.

Murphy, K.M., and Topel, R.H. (2006). The value of health and longevity. *Journal of Political Economy* 114, 871-904.

Nair, K.S. (2005). Aging muscle, *American Journal of Clinical Nutrition* 81, 953-963.

Nakagawa, F., May, M., and Phillips, A. (2013). Life expectancy living with HIV: recent estimates and future implications. *Current Opinion in Infectious Diseases* 26 (1), 17-25.

NVSS (2014). US department of health and human services, national vital statistics system. National Vital Statistics Report 63(7).

OECD (2018). Health spending (indicator). OECD (2018), Health spending (indicator). doi: 10.1787/8643de7e-en (Accessed on 26 March 2018).

Oster, E., Shoulson, I., and Dorsey, R. (2013a). Optimal expectations and limited medical testing: Evidence from Huntington Disease. *American Economic Review* 103(2), 804-830.

Oster, E., Shoulson, I., and Dorsey, R. (2013b). Limited life expectancy, human capital and health investments. *American Economic Review* 103(5), 1977-2002.

Read, D., and Read, N.L. (2004). Time discounting over the lifespan. *Organizational Behavior and Human Decision Processes* 94(1), 22-23.

Rebbeck T. R., Friebel T., Lynch HT, Neuhausen S. L., van't Veer L., Garber J. E., et al. (2004). Bilateral prophylactic mastectomy reduces breast cancer risk in BRCA1 and BRCA2 mutation carriers. *Journal of clinical oncology* 22, 1055–1062.

Rebbeck T. R., Lynch H. T., Neuhausen S. L., Narod S. A., Van't Veer L., Garber J. E., et al. (2002). Prophylactic oophorectomy in carriers of BRCA1 or BRCA2 mutations. *The New England Journal of Medicine* 346, 1616–1622.

Roberts, J. Scott, Barber, M., Brown, T.M., Cupples, L.A., Farrer, L.A., LaRusse, S.A., Post, S.G., et al. (2004). Who seeks genetic susceptibility testing for Alzheimer's Disease? Findings from a multisite randomized clinical trial. *Genetics in Medicine* 6(4), 197-203.

Ropka, Mary, Jennifer Wenzel, Elayne Phillips, Mir Siadaty, and John Philbrick (2006). Uptake rates for breast cancer genetic testing: A systematic review. *Cancer Epidemiology, Biomarkers and Prevention*, 15(5) 840-844.

Rockwood, K., and Mitnitski, A. (2007). Frailty in relation to the accumulation of deficits. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences* 62(7), 722-727.

Schünemann, J., Strulik, H., Trimborn, T. (2017a). Going from bad to worse: Adaptation to poor health, health spending, longevity, and the value of life. *Journal of Economic Behavior and Organization*, 140, 130-146.

Schünemann, J., Strulik, H., Trimborn, T. (2017b). The gender gap in mortality: How much Is explained by behavior? *Journal of Health Economics*, 54, 79-90.

Schünemann, J., Strulik, H., Trimborn, T. (2020). The marriage gap: Optimal aging and death in partnerships. *Review of Economic Dynamics* 36, 158-176.

Shoulson, I., and Young, A. (2011). Milestones in Huntington Disease. *Movement Disorders* 26(6), 1127-1133.

Sigal, B. M., Munoz D. F. , Kurian A. W., and Plevritis Sylvia K. (2012). A simulation model to predict the impact of prophylactic surgery and screening on the life expectancy of BRCA1 and BRCA2 mutation carriers. *Cancer Epidemiology, Biomarkers & Prevention* 21(7), 1066-1077.

Skirbekk, V. (2004), Age and individual productivity: A literature survey, *Vienna Yearbook of Population Research*, 133-153.

Stenehjem, D.D., Au, T., Sainski, A.M. et al. (2018). Impact of a genetic counseling requirement prior to genetic testing. BMC Health Services Research 18, 165.

Strulik, H. (2015). Frailty, mortality, and the demand for medical care, *Journal of the Economics of Ageing* 6, 5-12.

Strulik, H. (2018a). The Return to education in terms of wealth and health. *Journal of the Economics of Ageing* 12, 1-14.

Sunden, A. E. and Surette, B.J. (1998). Gender differences in the allocation of assets in retirement savings plans. *The American Economic Review* 88(2), 207–211.

Thornton, R. L. (2008). The demand for, and impact of, learning HIV status. *American Economic Review* 98(5), 1829-1863.

Waldron, I. (1993). Recent trends in sex mortality ratios for adults in developed countries. *Social Science & Medicine* 36(4), 451-462.

Wardle, J., Haase, A.M., Steptoe, A., Nillapun, M., Jonwutiwes, K., and Bellisie, F. (2004). Gender differences in food choice: the contribution of health beliefs and dieting. *Annals of Behavioral Medicine* 27(2), 107-116.

Weinhardt, Lance, Michael Carey, Blair Johnson, and Nicole Bickham (1999). Effects of HIV Counseling and Testing on Sexual Risk Behavior: A Meta-Analytic Review of Published Research, 1985-1997. *American Journal of Public Health* 89(9), 1397-1405.

Economics Working Papers

2019-11: Erik Strøjer Madsen: From Local to Global Competitors on the Beer Market

2020-01: Nikolaj Kirkeby Niebuhr: Managerial Overconfidence and Self-Reported Success

2020-02: Tine L. Mundbjerg Eriksen, Amanda Gaulke, Niels Skipper and Jannet Svensson: The Impact of Childhood Health Shocks on Parental Labor Supply

2020-03: Anna Piil Damm, Helena Skyt Nielsen, Elena Mattana and Benedicte Rouland: Effects of Busing on Test Scores and the Wellbeing of Bilingual Pupils: Resources Matter

2020-04: Jesper Bagger, Francois Fontaine, Manolis Galenianos and Ija Trapeznikova: Vacancies, Employment Outcomes and Firm Growth: Evidence from Denmark

2020-05: Giovanni Pellegrino: Uncertainty and Monetary Policy in the US: A Journey into Non-Linear Territory

2020-06: Francesco Fallucchi and Daniele Nosenzo: The Coordinating Power of Social Norms

2020-07: Mette T. Damgaard: A decade of nudging: What have we learned?

2020-08: Erland Hejn Nielsen and Steen Nielsen: Preparing students for careers using business analytics and data-driven decision making

2020-09 Steen Nielsen: Management accounting and the idea of machine learning

2020-10 Qazi Haque, Nicolas Groshenny and Mark Weder: Do We Really Know that U.S. Monetary Policy was Destabilizing in the 1970s?

2020-11 Giovanni Pellegrino, Efrem Castelnuovo and Giovanni Caggiano: Uncertainty and Monetary Policy during Extreme Events

2020-12 Giovanni Pellegrino, Federico Ravenna and Gabriel Züllig: The Impact of Pessimistic Expectations on the Effects of COVID-19-Induced Uncertainty in the Euro Area

2020-13 Anna Folke Larsen, Afonso Saraiva Câmara Leme and Marianne Simonsen: Pupil Well-being in Danish Primary and Lower Secondary Schools

2020-14 Johannes Schünemann, Holger Strulik and Timo Trimborn: Anticipation of Deteriorating Health and Information Avoidance