Anticipation of Deteriorating Health and Information Avoidance*

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Revised Version. February 4, 2023

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, Peter Zweifel, and two anonymous reviewers 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.

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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)[1]. Standard economic reasoning cannot explain this observation since more information is valuable in providing better decision-making. A direct consequence is that information, in our case a medical diagnosis, should never be deliberately avoided (Golman et al., 2017).

Several papers suggest that the combination of low testing rates and low testing costs may be motivated by future beliefs affecting utility directly ("anticipatory utility"). Li et al. (2020) categorize three classes of models to explain test avoidance: the optimal expectations model (Brunnermeier and Parker, 2005; Oster et al., 2013a), the attention model (Karlsson et al., 2009; Golman and Loewenstein, 2018; Golman et al., 2019; Ganguly and Tasoff, 2017), and the curvature model (Caplin and Leahy, 2001; Caplin and Eliaz, 2003; Köszegi, 2003; Eliaz and Spiegler, 2006). Although all these models are built on the idea of anticipatory utility, one major difference is whether beliefs are allowed to be chosen irrationally. While in the attention model and curvature model beliefs are assumed to be rational, in the optimal expectations model individuals avoid the test in order to maintain overoptimistic beliefs about the future and thus a higher anticipatory utility. This idea is supported by recent causal evidence that individuals indeed engage in wishful thinking about potential negative shocks in the future (Engelmann et al., 2019).

A prominent study in this context was carried out 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

[1]The fact that individuals choose to avoid testing also shows for other diseases like diabetes (Li et al., 2020), HIV (Lyter et al., 1987; Thornton, 2008), and more recently for Covid 19 (Serra-Garcia and Szech, 2020; Thunström et al., 2020). Also, Ganguly and Tasoff (2017) present evidence from the laboratory that individuals are willing to pay to avoid testing for two different types of herpes and that people are more likely to avoid testing for the virus type that is perceived more threatening. For an excellent review on information avoidance (also in a non-health context), see Golman et al. (2017).

anticipation of developing HD in the future reduces experienced utility already today such that individuals may avoid the test (and thus a potential positive diagnosis) in order to maintain biased beliefs about the probability of the health shock. The test is avoided if the increase in anticipatory utility compensates the utility loss from having less information. The model presented in this paper follows the basic idea of an optimal expectations model as well. 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. In our 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 anticipating the health shock with the detrimental effect of worse decision-making with respect to health-related behavior and consumption (see Benabou and Tirole, 2016, for a comprehensive survey on beliefs).

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 to assess the quantitative impact of economic and health-related factors on the testing decision 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. In order to understand our extension of health economic theory, it is important to remember the distinction between *anticipation* and *expectation*. 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 may or may not be a specific treatment available. 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 check sensitivity of the results with respect to different income levels and find that the non-testing decision is not affected by income of the individual. However, the willingness to pay for test avoidance varies greatly with individual income.

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 income of individuals if treatment is available even if the cost of the test is negligible (in contrast to the no-treatment case).

Unlike previous optimal expectations models, our model allows for a (quantitative) analysis of the effects of anticipatory utility on life-cycle health behavior. We find that – regardless of whether a shock occurs or not – anticipatory utility leads to substantially lower health investments and lower life expectancy. We show that this result is independent of individual income and explain the mechanisms behind it.

In order to put our quantitative results into perspective, we would like to point out three different aspects which should be considered when interpreting the quantitative results. First, we acknowledge that low testing rates could be driven by alternative and potentially complementary

mechanisms outside of our model. These factors might be for example incomplete information (in particular missing health literacy), direct utility costs of treatment, aspects of stigma, and time inconsistent planning of tests (due to hyperbolic discounting).

Second, the willingness to pay can differ from our benchmark calibration when the specification of the model changes or when one of the key parameters changes. We address this problem by reporting the willingness to pay for testing in the case of Huntington disease for different specifications of the utility function and for adaptation to anticipation, i.e. the feature that people cope with negative health news as time proceeds. We also conduct an extensive sensitivity analysis of the key parameters of the model. Both types of analysis show that the quantitative results change somewhat, but the qualitative results remain the same.

Third, when interpreting test rates it is useful to remember that the model is calibrated for an average American. Deviations from the mean, captured by heterogeneity in model parameters, can result in differences in the willingness to pay for remaining untested. We explore this feature by providing comparative dynamic analyses with respect to the key parameters of the model.

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), the education gradient (Strulik, 2018a), addiction and pain (Strulik, 2018b, 2021), and the design of fair pension systems (Grossmann et al., 2021).

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 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 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. whether and with what effectiveness treatment is available and how the severity of the health shock depends on the state of health at the time when the shock arrives. The individual dies when a critical deficit level D_T has been reached.

Since the health shock by assumption occurs 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.

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. 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. 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.[2]

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[3]

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

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, future expectations affect current economic behavior. A particular form of expectations in our model refers to potential health shocks and the resolution of uncertainty through medical tests. If an

[2]The survey by Viscusi (2019) concludes that only severe health shocks alter the marginal utility of consumption while mild health losses affect only the level of utility. These findings are broadly consistent with our formulation of the utility function, which features a negative cross-derivative between consumption and health such that severe health shocks affect the marginal utility of consumption more than mild health shocks. We acknowledge, however, that our utility function does not capture the threshold effect found in Viscusi (2019) since, in our model, small health shocks influence the marginal utility of consumption a little (rather than not at all). A consideration of a strict threshold effect, however, would not change our main results since we consider mostly very large health shocks (HD, HIV, cancer). We provide a sensitivity analysis with respect to the shape of the utility function in Appendix C.

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

individual is tested and the test is positive, a health shock is said to be *expected* because the individual knows beforehand that the shock will arrive.

An individual who remains untested chooses beliefs about the likelihood of the potential health shock. If, for example, an individual chooses $b = 1$, he or she believes that the shock occurs with certainty. In case the shock eventually occurs, this setting would be identical to the one of a diagnosed and expected health shock. If the individual chooses $b = 0$, in contrast, he or she chooses to believe that the health shock will with certainty not materialize. Then, if a health shock occurs, it is said to be *unexpected*. Aside from these border cases, individuals can choose any interior value of beliefs b.

In contrast to conventional models, currently experienced instantaneous utility is directly affected by these beliefs (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 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 who carries a defected gene, is not diagnosed and lives in denial. By expected health shocks, we model carriers of the gene defect who have been previously diagnosed and therefore know they will develop the 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

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

For $\alpha = 0$, health does not affect utility and 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 of 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 and Treatment 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 willingness to pay for the test derives from $E_0[V_{tested}] - E_0[V_{untested}]$ where we omit the time index for V. Accordingly, the individual chooses to be tested if $E_0[V_{tested}] > E_0[V_{untested}]$ 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 or her 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 (as in the case of cancer), the treatment and testing decision are interlinked (Felder and Mayrhofer, 2017; Chapter 5). Four cases must be considered: (i) the individual is tested and treated if tested positive (tested-treated), (ii) the individual is tested but abstains from treatment even if tested positive (tested-untreated; note that there could still be utility gains associated with the test since the individual can adjust non-treatment related behavior), (iii) the individual is not tested but treated (untested-treated), (iv) the individual remains untested and untreated (untested-untreated). The individual considers the associated expected lifetime utilities: (i) $E_0[V_{tested,treated}]$, (ii) $E_0[V_{tested,untreated}]$, (iii) $E_0[V_{untested,treated}]$, and (iv) $E_0[V_{untested,untreated}]$ with

$$E_0[V_{tested,treated}] = pV_{s=0,tr} + (1-p)V_{s=0} \tag{14}$$

$$E_0[V_{tested,untreated}] = pV_{s=1} + (1-p)V_{s=0}. \tag{15}$$

$$E_0[V_{untested,treated}] = V_{s=0,tr} \tag{16}$$

$$E_0[V_{untested,untreated}] = pE[V(b)_{s=1}] + (1-p)E[V(b)_{s=0}]. \tag{17}$$

and chooses the option that provides the highest expected lifetime utility. 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. As for testing without treatment, however, the only uncertainty is whether the test is positive (with probability p) or negative (with probability $1-p$). Note that the model could easily be extended to study only partially effective treatment or an uncertain treatment outcome. In these cases, individuals would choose their beliefs based on the effectiveness of the treatment.

The individual chooses to be tested if

$$max\{E_0[V_{tested,treated}], E_0[V_{tested,untreated}]\} > max\{E_0[V_{untested,treated}], E_0[V_{untested,untreated}]\}$$

such that the value of the test derives as

$$max\{E_0[V_{tested,treated}], E_0[V_{tested,untreated}]\} - max\{E_0[V_{untested,treated}], E_0[V_{untested,untreated}]\}. \tag{18}$$

2.4. **Model Calibration.** We solve the model numerically using a shooting procedure[4]. 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

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

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 $D(T) = 0.106$ 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 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 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.

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

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

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%.[5] 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 the 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. 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 how the calibrated model fits to the targeted data, we focus in this subsection on the age trajectories when no health shock is

[5]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 $\alpha = 0.01$.

considered. Therefore, the anticipation stock only refers to the anticipation of aging-related deficits.[6] 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 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.

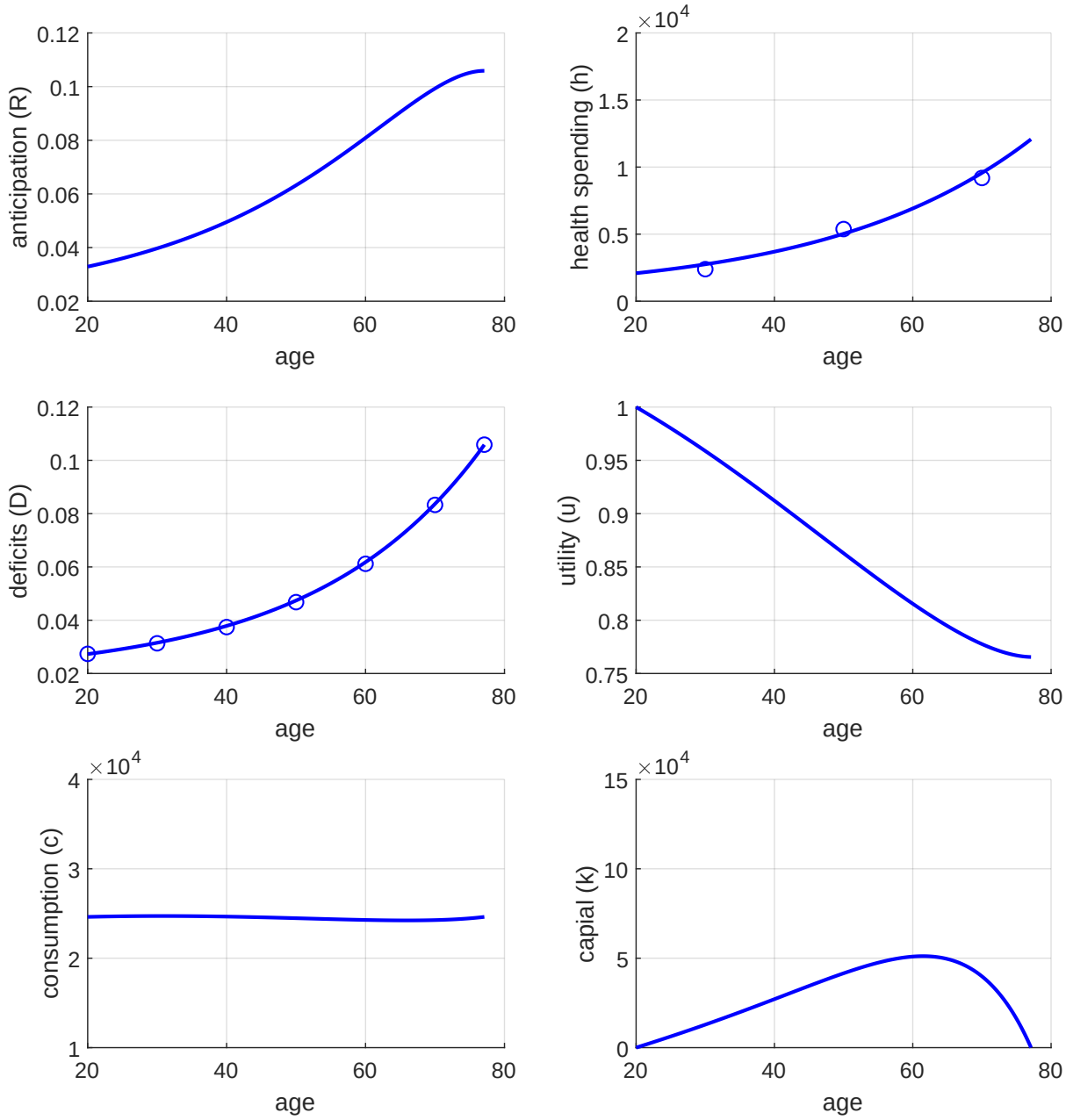
As can be seen from the upper-right panel in Figure 1, health expenditure increases over the life cycle as observed empirically (MEPS, 2010). The panel also shows that the calibrated model fits the health expenditure data reasonably well. The center left panel shows the exponential evolution of health 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 very low rates of testing in high-risk individuals, even though the cost of testing is very low. Low testing rates were reported 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).

[6]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.

Figure 1: Health Anticipation: Benchmark Run



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).

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 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 income 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.

We start by analyzing the case in which no treatment is available. 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 and by considering 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 take decisions to 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 is $E[V(b)_{s=0}]$ 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 negatively affects instantaneous utility already 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 (note that the same argument can also be made for a belief $b > 0$). 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 may add 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 (for $b > 0$) 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).

If treatment is available (and affordable), both the anticipating and non-anticipating type choose to be tested if treated ($E_0[V_{tested,treated}] > E_0[V_{untested,treated}]$ always holds for both

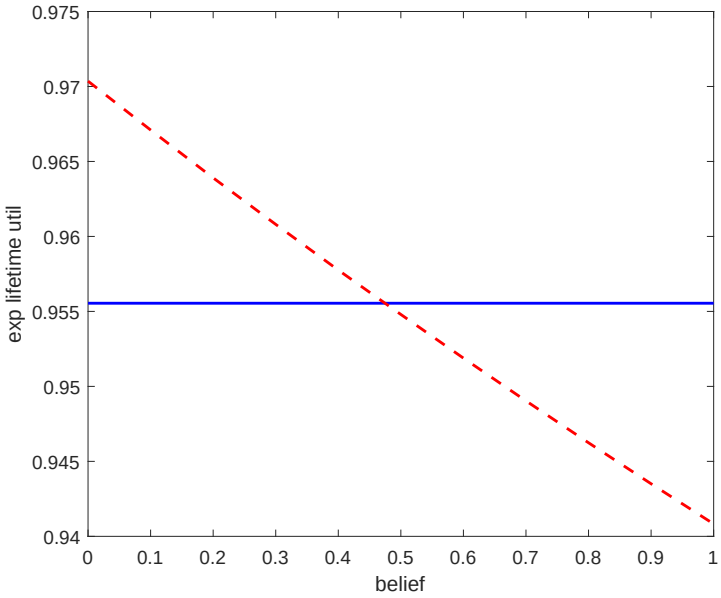
types). This follows directly from Equations (14) and (16). The intuition is that by taking the test, the individual can learn his or her health status and save on treatment cost in case he or she is not sick. In this way, the availability of treatment does not add an additional channel through which anticipating individuals might remain untested. Note, however, that anticipating individuals still prefer to remain untested if $E_0[V_{untested,untreated}] > max\{E_0[V_{tested,untreated}], E_0[V_{tested,treated}]\}$. This case occurs, for example, if the costs of treatment are too high in comparison to the gains and treatment is avoided. A test would then reveal only the health status and it is also avoided if the utility loss from the anticipation of bad health is sufficiently large. In Section 5, we discuss how this case emerges when poor women avoid costly breast cancer treatment.

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 by $0.70 \cdot D_0$, i.e. by 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, relative to the lifetime utility of the 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

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 $V_{s=0}$.

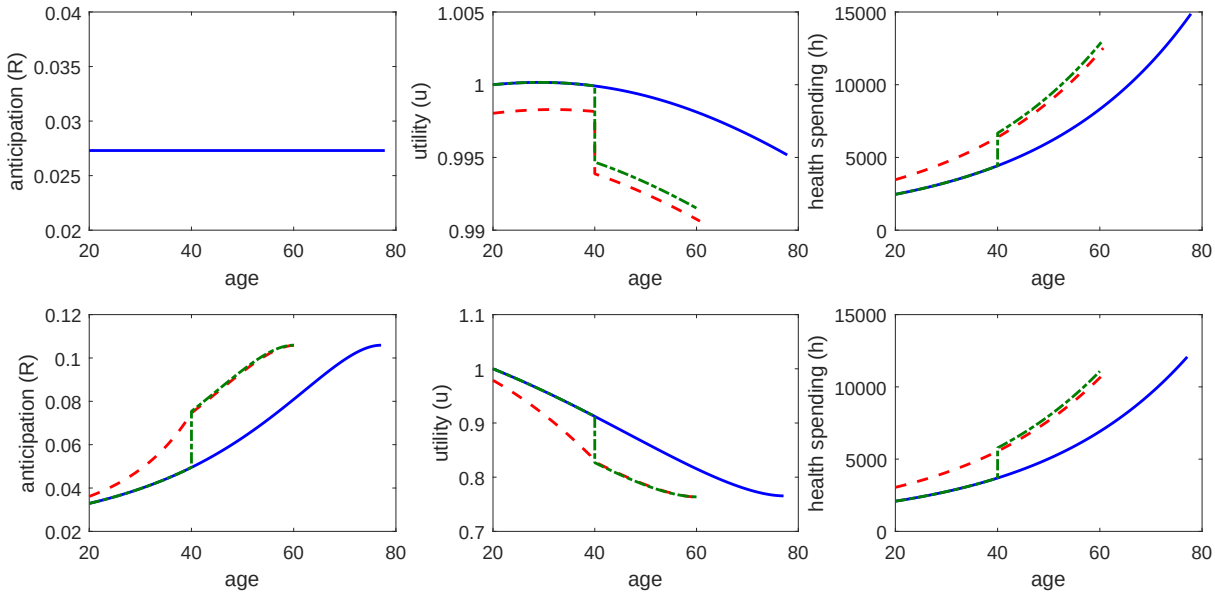
health shock more likely, the negative effect of anticipation on instantaneous utility increases and the relative gain from remaining untested shrinks. As can be seen in the figure, the threshold belief at which individuals start to prefer being tested lies slightly below 0.5. Furthermore, expected lifetime 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%). Individuals facing an expected health shock adjust by increasing their health expenditure already beforehand such that they are in better health when the shock hits. Being in generally better health allows them to live longer and to experience instantaneous utility over a longer lifespan, 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.

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).

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 is also reflected in the age trajectories for instantaneous utility shown 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. 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 also lower compared to the case of an unexpected shock. The drop in instantaneous utility cannot be compensated for by the 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 2 shows the quantitative results for the two outcome variables life expectancy and expected lifetime utility or welfare for the tested (as defined in (12)) and non-tested case (as defined in (13)). 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. The upper part of the table considers the benchmark individual with average labor income. 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 percentage point 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 2: HD at age 40: Impact on Life Expectancy and Welfare

	no anticipation		anticipation	
outcome	untested	tested	untested	tested
average wage w				
1) LE	-15.17	-14.82	-14.97	-14.60
2) Welfare	-2.56	-2.55	-2.96	-4.45
$0.5 * w$				
3) LE	-14.42	-14.15	-14.28	-14.00
4) Welfare	-2.65	-2.64	-2.95	-4.38
$2 * w$				
5) LE	-16.21	-15.76	-15.94	-15.45
6) Welfare	-2.45	-2.43	-2.99	-4.53

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.

We also analyze the influence of income in this context. To do this, we reduce and increase the labor income w by a factor of two. The results for these experiments are shown in the second and third part of Table 2. As the table shows, the results are affected only mildly by different levels of income. This implies that the testing decision does not depend on income when no treatment is available.

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 the reference individual is obtained as 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.

Naturally, the willingness to pay increases in the income of the individual. We find that an anticipating individual endowed with twice (half) the benchmark income is willing to pay $ 299,583 ($ 55,660) for avoiding the test, while a non-anticipating individual is willing to pay $ 3,845 ($ 393) to be tested. The testing decision, however, remains unaffected by different income levels of the individual.

When interpreting the quantitative results, it is important to understand that we aim to isolate the impact that anticipatory utility has on the testing decision of the individual. We acknowledge that there are other factors outside the model which affect the testing decision like, for example, incomplete information (in particular missing health literacy) or stigma. Also, note that we report the willingness to pay for the average American to provide a quantitative rationalization for why so many individuals remain untested. Therefore, heterogeneity in the model parameters may lead to different measures in the willingness to pay. In Appendix D, we explore this argument by providing a comparative dynamic analysis with respect to the key parameters of the model.

In Appendix C, we demonstrate the robustness of results with a comprehensive sensitivity analysis in terms of functional forms and parameter choices. Here, we briefly sketch some particularly interesting robustness checks. For example, we abandoned multiplicative separability of the utility function and replaced Cobb-Douglas utility by CES utility of the form $U(c, D, R) = \vartheta[\kappa u(c)^\epsilon + \alpha u(D)^\epsilon + \beta u(R)^\epsilon]^{\frac{1}{\epsilon}}$. The elasticity of substitution between $u(c)$, $u(D)$, and $u(R)$ is given by $\delta \equiv 1/(1-\epsilon)$. The benchmark model is included as special case for $\delta \to 1$. For $\delta < 1$, the utility components become more complementary while for $\delta > 1$, they become more substitutable. In the limiting case $\delta \to \infty$ ($\epsilon \to 1$), the utility function becomes additively separable.

When the elasticity of substitution declines by one third (from 1 to 0.67), the re-calibrated model predicts that the welfare reduction due to the HD shock increases from 2.96% to 3.37% in the untested case and from 4.45% to 4.76% in the tested case. The willingness to pay for avoiding the test decreases from $ 129,000 to $ 101,000 (Table A.1). Analogously, when the elasticity of substitution increases by one third, the welfare reduction due to the HD shock declines to 2.68%

if untested and 4.28% if tested and the willingness to pay increases from $ 129,000 to $ 156,000. In the limit, when utility becomes additively separable, the model predicts a welfare decline of 2.45% if untested and 4.18% if tested and the willingness to pay for test avoidance increases to $ 185,000.

The channels through which the changing elasticity of substitution affects results are hard to disentangle. First, a lower elasticity of substitution implies a smaller impact of anticipation on utility from consumption, which requires a larger stand-alone effect of anticipation on utility, i.e. a larger value of β, in order to match the calibration targets. Second, since the elasticity of substitution enters the Euler equation, a larger elasticity implies a larger value of the calibrated time preference rate and therewith smaller welfare effects from (the anticipation of) future health shocks due to greater discounting. Third, a larger β reduces the intertemporal elasticity of substitution and requires a higher value of the calibrated σ, which in turn entails a higher value of (the anticipation of) future health shocks in terms of income units. Overall, however, these changes are quantitatively not substantial and the qualitative results are robust to the assumed form of the utility function. In particular, the robustness checks eliminate the concern that the obtained large impact of anticipation on welfare and willingness to pay could be an artefact of the impact of anticipation on the (marginal) utility from consumption implied by the Cobb-Douglas form of the utility function.

In the Appendix C, we also provide a sensitivity with respect to the time preference rate ρ and therewith the implied age-consumption profile. Independently from the assumed level of the time preference rate, we found a 1.5 percentage points welfare difference between the untested and tested case of the health shock. A change in the assumed time preference rate entails a change in the calibrated curvature of the utility function (σ) in order to meet the calibration targets. In sum of these changes, the discounted value of lifetime income in the re-calibrated models remains essentially unchanged. The well-known property that life cycle behavior depends on permanent income then explains the independence of the results in relative terms. In absolute terms, however, results change substantially because the implied re-calibration of σ affects the level of welfare and the willingness to pay for avoiding the test. When ρ changes from 0.59 to 0.72, the willingness to pay changes from $ 97,000 to $ 171,000.

Finally, we discuss the role of adaptation. The medical and economics literature has provided ample evidence that individuals learn to cope with bad states of affairs and, in particular, with

health-related issues. The seminal study by Brickman et al. (1978), comparing paraplegics and lottery winners, was the starting point for a series of studies (e.g. Wu, 2001; Albrecht and Devlieger, 1999; Riis et al., 2005) showing that people adapt to health problems over time. Adaptation can also play a role in the testing decision as individual learn to cope with the bad news of a positive test. We introduce adaptation to anticipation following the modeling of adaptation to deteriorating health in Schünemann et al. (2017a). Details of the extended model are provided in Appendix E.

In the calibration of adaptation, we focus on two cases: (i) the speed of adaptation equals the above calibrated speed of anticipation (low speed scenario), (ii) the speed of adaptation equals the speed of adaptation to poor health as calibrated in Schünemann et al. (2017a) (high speed scenario). Redoing the computational experiments from above, we find that, for the low speed scenario, the willingness to pay for avoiding the test decreases from 129,000 in the benchmark scenario to 45,000 dollars. In the high speed scenario, the willingness to pay for avoiding the test drops further to 21,000 dollars. Since individuals learn to cope with the bad news of the future health shock, the effect of anticipation on welfare declines and the decline is greater for faster adaptation. However, all qualitative results of the benchmark model are preserved under adaptation.[7]

A distinct feature of our model in comparison to previous optimal expectation models is that we can analyze and quantify the effect of anticipatory utility on health behavior as shown in the right panels in Figure 3. To this end, we compare the present discounted value (PDV) of health expenditures for the different scenarios of the non-anticipating and anticipating type. The first row of Table 3 shows the percentage deviation of the PDV of health expenditures of the anticipating type from the non-anticipating type for our considered scenarios. In all three scenarios (no shock, unexpected shock, expected shock), anticipation leads to substantially lower health investments. This implies that the tested scenario (as a weighted average of the no-shock and the expected scenario) and untested scenario (as a weighted average of the no-shock and unexpected scenario) of the anticipating type features lower health investments as well. The difference in health investments in all cases considered lies between 13.1 % and 16.6 %. As

[7]This conclusion may change for milder diseases with less substantial impact on disutility from anticipation. Then, if individuals underestimate their ability to adapt (here the speed of adaptation), informing about adaptation could lead to taking a test.

shown in the second row of Table 3, lower health investments directly translate into lower life expectancy which reduces by 0.61 % to 1.25 %.

Table 3: Impact of Anticipation on Health Expenditure

outcome	no shock	unexpected	expected	untested	tested
average wage w					
1) PDV h	-16.6	-15.2	-13.1	-15.9	-14.6
2) LE	-1.25	-0.70	-0.61	-1.02	-0.98
$0.5 * w$					
3) PDV h	-16.8	-15.7	-13.6	-16.3	-15.0
4) LE	-0.95	-0.57	-0.51	-0.79	-0.77
$2 * w$					
5) PDV h	-16.2	-14.6	-12.6	-15.4	-14.2
6) LE	-1.65	-0.86	-0.75	-1.33	-1.29

The values are deviations of the results for the anticipating type in percent from the non-anticipating type; PDV h refers to the present discounted value of lifetime health expenditures, LE denotes life expectancy at age 20.

The driving force behind these results is an interesting feedback effect of anticipation. The anticipation of future health events makes anticipating types less happy as it reduces instantaneous utility. Since life is less worthwhile at any age, anticipating types invest less in prolonging life compared to non-anticipating types. This mechanism is illustrated in the center panels of Figure 2 that show instantaneous utility in the course of aging. Since anticipating types draw negative utility from anticipating future health deficits, their utility declines more rapidly over life compared to non-anticipating types. These observations qualitatively hold regardless of whether a shock occurs and whether the potential shock is expected or unexpected.

In the second (rows 3 and 4) and third part (rows 5 and 6) of Table 3, we report results for a comparative dynamic analysis with respect to labor income. As shown, the results change only slightly when income increases or decreases by a factor of two.

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 income 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	17,303	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).

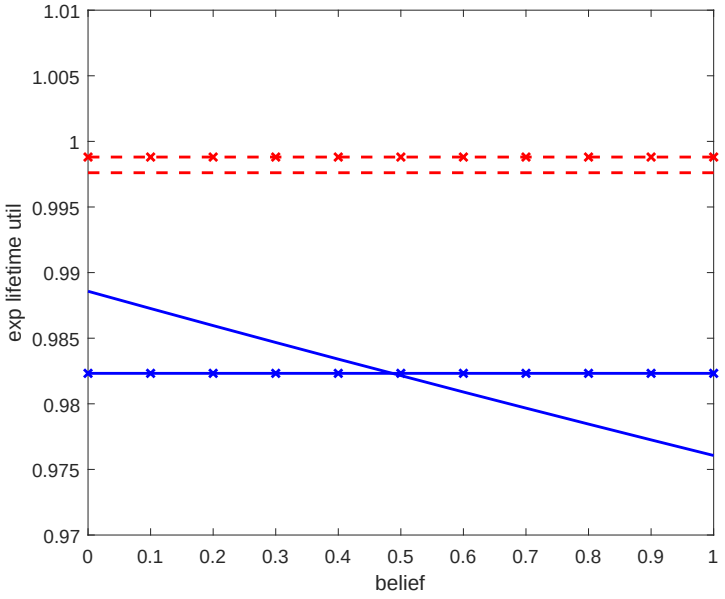
To investigate testing and treatment decisions, we consider a woman whose parent carries the BRCA genetic defect and thus has a 50% chance of inheriting the defect. We assume that presence of the BRCA gene without treatment causes cancer with certainty. A free test could provide information whether the woman carries the genetic defect. Alternatively, the woman could avoid the test and form an optimal belief of whether the genetic defect is present. 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 age 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.

We assume that treatment sets in at age 30 since this is the age at which the uptake of prophylactic measures is observed (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.[8]

Figure 4 shows the expected lifetime utilities for four different scenarios relative to the no-shock scenario for various beliefs. A testing decision is indicated by crosses, while a treatment decision is indicated by red lines. Therefore, the tested-treated scenario is represented by red crossed lines, the untested-treated scenario by red non-crossed lines, the tested-untreated scenario by blue crossed lines, and the untested-untreated scenario by blue non-crossed lines. We illustrate the high-cost treatment case with $p_s h_s = 50,000$. According to expression (18), the value of the test is determined by the difference in expected lifetime utility associated with the optimal (treatment) behavior when tested vs. non-tested. Hence, the value of the test can be easily read off the figure by the difference between the highest crossed line and the highest non-crossed line.

Figure 4: Breast and Ovarian Cancer: Beliefs and Lifetime Utility



Crosses indicate testing, red lines indicate treatment. Specifically: Blue (solid) non-crossed line: untested-untreated. Blue (solid) crossed line: tested-untreated. Red (dashed) non-crossed line: untested-treated. Red (dashed) crossed line: tested-treated. Exp lifetime util is expected lifetime utility relative to lifetime utility in the benchmark no-shock scenario $V_{s=0}$.

[8]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. Also, incomplete information or stigma may play a role in the testing decision. 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.

If no treatment were available, Figure 4 would be qualitatively equivalent to Figure 2 from the HD analysis. The woman would choose belief $b = 0$ and not take the test. When effective treatment is available, however, the woman chooses to be tested and and, if the test result is positive, to have treatment. Since breast and ovarian cancer have huge effects on lifetime utility, the woman would also prefer to be treated than go untreated if a test were not available. The positive value of the test results from the fact that the test provides information about whether treatment is necessary or not. The test avoids the cost of treatment in 50% of cases where the woman is healthy.

The effects of the testing and treatment decision on life expectancy and welfare are summarized in Table 5. Rows 1 and 2 refer to the low-cost treatment and rows 3 and 4 refer to the high-cost treatment. The left-hand side shows outcomes without anticipation and the right-hand side shows outcomes with anticipation (and $b = 0$). Without treatment, the genetic defect reduces life expectancy by around 8%, which equates to a reduction in life expectancy of about 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%). Treatment almost restores benchmark life expectancy. The small loss in life expectancy 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 to be tested and treated because then the welfare loss caused by the potential health shock is smallest (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 (the difference in welfare is not visible in the table due to rounding), the anticipating type would choose to remain untested to minimize the welfare loss from the potential health shock ($-1.14\% > -1.77\%$).

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

		No Anticipation				Anticipation			
		untested	tested	untested	tested	untested	tested	untested	tested
outcome	costs	untreated	untreated	treated	treated	untreated	untreated	treated	treated
1) LE	low	-8.30	-8.23	-0.21	-0.10	-8.27	-8.19	-0.21	-0.11
2) Welfare	low	-1.04	-1.04	-0.07	-0.03	-1.14	-1.77	-0.09	-0.05
3) LE	high	-8.30	-8.23	-0.52	-0.26	-8.27	-8.19	-0.52	-0.26
4) Welfare	high	-1.04	-1.04	-0.17	-0.09	-1.14	-1.77	-0.24	-0.12

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.

Table 6 shows the associated willingness to pay for being tested if no treatment is available (as a hypothetical counterfactual) and if treatment is available for the low-cost treatment (row 1) and the high-cost treatment (row 2). If no treatment is available, the value of the test is determined by simply comparing the tested-untreated and the untested-untreated scenario ($E_0[V_{tested,untreated}] - E_0[V_{untested,untreated}]$). If treatment is available, the value of the test derives from expression (18). From Figure 4 and Table 5 it is immediately apparent that $E_0[V_{tested,treated}] > E_0[V_{tested,untreated}]$ and $E_0[V_{untested,treated}] > E_0[V_{untested,untreated}]$. Therefore, expression (18) implies that the willingness to pay for the test is given by the difference in lifetime utility between the tested-treated and untested-treated scenario (the distance between the two red lines in Figure 4) converted into monetary equivalents.

If treatment is not available, a non-anticipating woman would pay $ 195 for receiving a test result. This amount reflects the value of life gained by adjusting life cycle behavior to the test result. If treatment is available, a non-anticipating woman would pay $ 5,004 or $ 11,918 for the test. This amount reflects the value of life gained by avoiding treatment when the test result is negative. An anticipating woman would pay $ 104,083 (six annual wages) to avoid testing if treatment is not available, while the woman would pay $ 7,339 or $ 17,458 to be tested if treatment was available. Anticipation thus increases the value of a test when treatment is available. The mechanism works as follows. The value of the test expresses in monetary terms the gain in lifetime utility that results from saving the treatment costs in case the test is negative (the woman does not have the genetic marker). Both types of women use the saved treatment costs to increase consumption and health spending along the life-cycle. This increases lifetime utility for both types of women. For anticipating women, however, an additional mechanism is at work: higher health spending along the life-cycle leads to a healthier and prolonged life. This positively affects remaining lifetime utility through anticipation. Since only anticipating women benefit from this channel, they value the test higher.

According to our model, an American single woman endowed with the average wage would always choose to be tested and treated 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 non-testing can also be explained by poverty. As income decreases, marginal utility of instantaneous consumption increases, and

Table 6: Breast and Ovarian Cancer: Δ Value of Life (Value of Testing in $)

		No Anticipation		Anticipation	
		tested-untreated vs.	tested-treated vs.	tested-untreated vs.	tested-treated vs.
outcome	costs	untested-untreated	untested-treated	untested-untreated	untested-treated
1) Δ VoL	low	195	5,004	-104,083	7,339
2) Δ VoL	high	195	11,918	-104,083	17,458

Δ VoL refers to the difference in expected lifetime utility expressed in dollars.

with it the opportunity cost of investing in life extension through expensive treatment. At some point, treatment becomes too costly in terms of utility. This means that treatment becomes de facto unavailable. Without treatment, the anticipating woman refuses the test because of the potential utility cost from anticipation and chooses to live in denial ($b = 0$).

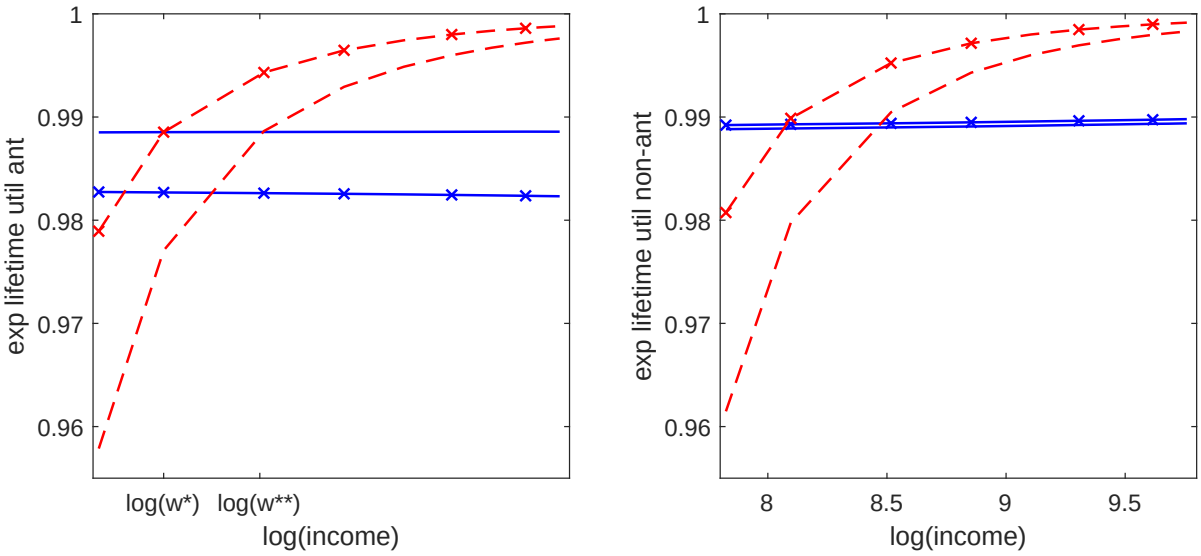
The association between income and the testing decision is illustrated in Figure 5, which shows the expected lifetime utility for various wages (up to the average wage) for $b = 0$. All other parameters are kept as calibrated. Again, crosses indicate a testing decision, while red (dashed) lines indicate a treatment decision. Expected lifetime utility is expressed relative to the respective no-shock scenario. Results are shown on the left for the anticipating woman and on the right for the non-anticipating woman.

We first focus on the anticipating woman. As can be seen in Figure 5, without treatment (blue lines) income has little effect on the welfare loss from a potential health shock. With treatment (red lines), however, the welfare loss due to a potential health shock increases sharply with income because the treatment costs burden the woman all the more, the poorer she gets. We can calculate a threshold wage w^* below which women prefer to remain untested. At income levels below w^*, treatment becomes too costly in terms of welfare such that the woman remains untreated and – due to the negative effects of anticipation – she also remains untested. For the calibrated benchmark woman, the threshold wage is $ 1,500 in the low-cost case and $ 3,300 in the high-cost case.

The value of the test, according to expression (18), is shown in the Figure as the difference between the highest crossed and the highest non-crossed line. For $w < w^*$, the value of the test is negative. At w^* the value of the test is zero while for $w > w^*$ the value of the test is positive. We can calculate another threshold w^{**} as the wage at which treatment becomes optimal regardless of the testing decision. For $w \leq w^{**}$, the value of the test is given

by $E_0[V_{tested,treated}] - E_0[V_{untested,untreated}]$. For $w > w^{**}$, the value of the test is given by $E_0[V_{tested,treated}] - E_0[V_{untested,treated}]$. In Appendix D, we provide a comparative dynamic analysis with respect to the threshold wage in order to account for heterogeneity in the model parameters.

Figure 5: Breast and Ovarian Cancer: Income and Lifetime Utility



Left: anticipating woman. Right: non-anticipating woman. Crosses indicate testing, red lines indicate treatment. Specifically: Blue (solid) non-crossed line: untested-untreated. Blue (solid) crossed line: tested-untreated. Red (dashed) non-crossed line: untested-treated. Red (dashed) crossed line: tested-treated. Exp lifetime util is expected lifetime utility relative to lifetime utility in the respective benchmark no-shock scenario $V_{s=0}$. w^* refers to the threshold wage at which the anticipating individual is indifferent between testing and non-testing. w^{**} indicates the threshold wage at which treatment becomes optimal for the anticipating individual independent of the testing decision.

As a counterfactual experiment, we show the results for an otherwise identical non-anticipating woman on the right-hand side of Figure 5. Now, the value of the test (the difference between the highest crossed line and the highest non-crossed line) is always positive, at all levels of income. Therefore, the non-anticipating woman always chooses to be tested regardless of the income level. When treatment becomes too costly in terms of welfare, the non-anticipating woman still decides to have a test, in order to re-optimize behavior unrelated to treatment. In summary, poverty is a potential explanation for non-testing behavior only if women anticipate future health shocks.

The calculated threshold wage w^* appears low at first glance. Note however that this wage represents an average wage paid throughout the entire lifetime and not only during working

age.[9] In 2010, the average income in the bottom decile of the income distribution of single woman was $ 2136 according to the Consumption Expenditure Survey (BLS, 2012). Our model predicts that single woman at this lower end of the income distribution would prefer to remain untested in the high-cost case but would get tested in the low-cost case. Therefore, subsidizing treatment could lead to testing (when women anticipate future shocks).

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 figures suggest that there is a significant number of women for whom the cost of treatment places an unbearable burden on welfare, implying that they choose not to get tested at all.

The affordability of treatment cost calls for a discussion on health insurance. Although not legally required, the majority of private health insurers cover preventive surgery to reduce risk for breast and ovarian cancer in the U.S. However, individuals may face high out-of-pocket payments due to deductibles or co-payments. Also, some health plans do not consider prophylactic surgery medically necessary and thus do not cover prophylactic mastectomy. As far as public health insurance is concerned, Medicare typically does not cover preventive surgeries, while Medicaid coverage varies by state. This being said, we briefly discuss health insurance in our model framework also to extend our findings to other countries where health insurance coverage is more generous than in the U.S. (FORCE, 2021).

For our calibrated average woman, health insurance does not change the test and treatment decision since she is already willing to pay for the treatment out of pocket. This, however, does not mean that any woman in the population is willing to do so even when she is endowed with the average wage. In a comparative dynamic analysis (Appendix D) we show that a woman with a 50 % lower coefficient of relative risk aversion (σ) than the benchmark woman would choose to remain untested. The threshold wage for this woman is $ 26,755 while the average

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

wage is $ 17,303. For this example, we can calculate the degree of insurance coverage for which the woman would choose to be tested and treated. We find that if health insurance covers at least 36 % of the treatment cost, the woman would opt for a test and potential treatment.

To assess the impact of health insurance coverage on the threshold wage, we calculate for different degrees of insurance coverage the wage at which women are indifferent between remaining untested and taking the test and potential treatment. We find that for every percentage increase in coverage for medical expenses, the threshold wage decreases by about one percent.

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 it to contribute to the literature on information avoidance in the context of health. We illustrated that the anticipation of bad health shocks is 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 falls below a certain threshold, the cost of treatment becomes too hurtful in terms of expected lifetime utility. If treatment is not affordable, 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 income of the individual.

Our study 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.

In addition to the factors mentioned throughout the paper, we acknowledge that there are further determinants which affect the testing decision and its outcomes. Oster et al. (2013a; 2013b) report that a positive test result triggers behavioral changes related to education, fertility, retirement, or unhealthy consumption which are not present in our model. Neglecting the reoptimization of these factors after a positive diagnosis is likely to increase the cost of information avoidance which would result in a lower willingness to pay for avoiding the test. Therefore, 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. Finally, we would like to point out that an alternative explanation for non-testing behavior may be found in hyperbolic discounting such that individuals (infinitely) postpone a planned medical test.

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 beliefs $0 \leq b \leq 1$. 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

$$\dot{D} = \mu(D - Ah^{\gamma} - a) \tag{19a}$$

$$\dot{R} = \theta(R - D) \tag{19b}$$

$$\dot{k} = w + rk - c - ph \tag{19c}$$

$$\dot{\lambda}_D = -\lambda_D(\rho - \mu) + \frac{\alpha}{D}u(c, D, R) \tag{19d}$$

$$\dot{\lambda}_k = \lambda_k(\rho - r) \tag{19e}$$

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

$$\lambda_k(\tilde{t}^-) = b\lambda_{k,s=1}(\tilde{t}^+) + (1-b)\lambda_{k,s=0}(\tilde{t}^+) \tag{20a}$$

$$\lambda_D(\tilde{t}^-) = b\lambda_{D,s=1}(\tilde{t}^+) + (1-b)\lambda_{D,s=0}(\tilde{t}^+) \tag{20b}$$

$$R(\tilde{t}^-) = bR_{s=1}(\tilde{t}^+) + (1-b)R_{s=0}(\tilde{t}^+) \tag{20c}$$

$$k(\tilde{t}^-) = k_{s=0}(\tilde{t}^+) \tag{20d}$$

$$k(\tilde{t}^-) = k_{s=1}(\tilde{t}^+) \tag{20e}$$

$$D(\tilde{t}^-) = D_{s=0}(\tilde{t}^+) \tag{20f}$$

$$D(\tilde{t}^-) = D_{s=1}(\tilde{t}^+) + \tilde{D}\,. \tag{20g}$$

Equations (20a)-(20c) are the interior boundary conditions for the forward looking variables described in section 2.2. Equations (20d) and (20e) state that the capital stock evolves continuously, independent of the realization of the shock. Equation (20f) states that health deficits

evolve continuously in case no shock sets in, and Equation (20g) states that health deficits D are increased 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.[10] 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 (19) 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 (20a)-(20c). 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.

Appendix C: Sensitivity Analysis

Utility Function. In this section, we check sensitivity with respect to the substitution elasticity between the components in the utility function. In our benchmark specification, we implicitly

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

assumed a substitution elasticity of 1 by relying on a Cobb-Douglas utility function. We now generalize the utility function to a CES specification that allows for the analysis of anticipation for different substitution elasticities. In particular, we assume a utility function of the form

$$U(c, D, R) = \vartheta[\kappa u(c)^{\epsilon} + \alpha u(D)^{\epsilon} + \beta u(R)^{\epsilon}]^{\frac{1}{\epsilon}} \tag{21}$$

where $u(c) = \frac{c^{1-\sigma}-1}{1-\sigma}$, $u(D) = \left(\frac{\bar{D}}{D}\right)$, $u(R) = \left(\frac{\bar{D}}{R}\right)$, $\kappa = 1 - \alpha - \beta$, $\epsilon \neq 0$, and $\vartheta > 0$. The parameter ϵ determines the elasticity of substitution between $u(c)$, $u(D)$, and $u(R)$, defined as $\delta \equiv \frac{1}{1-\epsilon}$. For $\delta < 1$ ($\epsilon < 0$), the utility components become more complementary while for $\delta > 1$ ($\epsilon > 0$), they become more substitutable. In the limiting case $\delta \to \infty$ ($\epsilon \to 1$), the utility function becomes additively separable. For $\delta \to 1$ ($\epsilon \to 0$), the CES utility nests a Cobb-Douglas utility with

$$U(c, D, R) = \vartheta u(c)^{\kappa} u(D)^{\alpha} u(R)^{\beta}. \tag{22}$$

The Cobb-Douglas utility slightly differs from our benchmark utility in (5) by two components. First, we have to add the weight parameter κ to consumption utility since the utility weight parameters in the CES must add up to 1 in order to nest a Cobb-Douglas function. Second, utility is shifted by the constant ϑ. This size of the parameter ϑ does not affect any choices of the individual but it will be adjusted to ensure that the absolute value of utility coincides with that obtained from the benchmark utility function of the main text.

We first establish the benchmark scenario for $\delta = 1$ ($\epsilon = 0$) with our slightly modified Cobb-Douglas utility function. The first row of Table A.1 shows the calibration results. The parameter σ has to be recalibrated in order to match the intertemporal elasticity of substitution with respect to consumption from the benchmark utility to meet the original calibration targets. The last three columns show the percentage change for the untested and tested case from the no-shock scenario as well as the welfare difference between these two cases expressed in 1000 dollars. Results for the testing decision are shown in the last three columns of Table A.1. The first row ($\delta = 1$) shows the results for the transformed Cobb-Douglas (which coincide with the results obtained with the benchmark utility function from Table 3 in the main text).

We now increase and decrease the substitution elasticity by 1/3, recalibrate the model to match the calibration targets, and rerun the experiments. When reducing the substitution elasticity, the components of the utility function become more complementary. Therefore, anticipation has c.p. a larger impact on utility since its impact on consumption utility $u(c)$ increases. We thus

Table A.1: Sensitivity Analysis – Utility Function

case	ρ	σ	α	β	A	θ	ϑ	untested	tested	Δ Vol
1) $\delta = 1.00$ ($\epsilon = 0.0$)	0.065	1.12	0.01	0.22	0.00146	0.10	1.27	-2.96	-4.45	-129
2) $\delta = 0.67$ ($\epsilon = -0.5$)	0.062	1.09	0.01	0.07	0.00146	0.10	0.71	-3.37	-4.76	-101
3) $\delta = 1.33$ ($\epsilon = 0.5$)	0.068	1.13	0.01	0.47	0.00146	0.10	1.82	-2.68	-4.28	-156
4) $\delta \to \infty$ ($\epsilon = 1$)	0.070	1.14	0.01	0.74	0.00146	0.10	2.62	-2.45	-4.18	-185

The values in the columns "'untested"' and "'tested"' are deviations in welfare (expected lifetime utility) in percent from the benchmark run without health shock. Δ Vol refers to the difference in welfare expressed in 1000 dollars.

need to reduce the utility parameter of anticipation β to 0.07 in order to match the calibration target of anticipation. Since β also affects the intertemporal elasticity of substitution with respect to consumption through $\kappa = 1 - \beta - \epsilon$, we need to adjust σ accordingly. In addition, the substitution elasticity enters the Euler equation for consumption. The more complementary the components are, the more the individual is willing to substitute future for present consumption in order to consume when he or she is still healthy. This drives down the consumption path which has to be counterbalanced by a lower ρ in order to match the calibration target of a flat consumption profile.

A lower ρ implies that individuals discount the future less and thus the health shock has a larger impact on welfare. The welfare reduction increases in the untested case from 2.96 % to 3.37 % and in the tested case from 4.45 % to 4.76 %. The willingness to pay for avoiding the test decreases from about $ 129,000 to $ 101,000, as shown in the ΔVol column of Table A.1

The aforementioned mechanisms hold vice versa for the case when the utility components become more substitutable ($\delta = 1.33$). In this case, the willingness to pay increases from $ 129,000 to $ 156,000. The last row in Table A.1 shows results for an infinite elasticity of substitution. For this case of an additively separable utility function, the willingness to pay increases to $ 185,000. Summing up the sensitivity analysis, we see that changing the substitution elasticity has a moderate impact on the willingness to pay for avoiding the test, but leaves the order of magnitude of the results and the decision to avoid testing unchanged.

Consumption Path. We next check sensitivity with respect to the consumption profile. In the benchmark case, we assume a constant consumption trajectory over the life course according to Browning and Ejrnaes (2009). In fact, the shape of the consumption profile should not matter per se since the individual has unrestricted access to the capital market. In such a setting, what

matters is the present discounted value of lifetime income rather than current income (permanent income hypothesis) and thus there is no contemporaneous tradeoff between consumption and health expenditure. Therefore, as long as the present discounted value of lifetime income remains unchanged, the shape of the consumption path should not make a big difference for the lifetime utility of the individual.

The parameter ρ is the parameter that, given the other calibration targets, governs the shape of the consumption profile (see Euler equation in (9)). Therefore, we check the sensitivity of the results to changes in ρ and thus the shape of the consumption profile. The results are shown in Table A.2.

Table A.2: Sensitivity Analysis – Consumption Path

case	σ	α	β	A	θ	$c(T)/c(0)$	untested	tested	Δ Vol
1) $\rho = 0.65$	1.16	0.01	0.217	0.00146	0.10	1	-2.96	-4.45	-129
2) $\rho = 0.59$	1.12	0.01	0.217	0.00146	0.10	0.73	-3.56	-5.03	-97
3) $\rho = 0.72$	1.20	0.01	0.217	0.00146	0.10	1.40	-2.46	-3.94	-171

The values in the columns "'untested"' and "'tested"' are deviations in welfare (expected lifetime utility) in percent from the benchmark run without health shock. $c(T)/c(0)$ denotes the difference between the final and initial consumption level. Δ Vol refers to the difference in welfare expressed in 1000 dollars.

The first row reiterates the results from the benchmark run for $\rho = 0.65$. The second and third row show the results for a 10 % decrease and increase in ρ. The model is then recalibrated to fit the other calibration targets from the benchmark run. When increasing ρ and keeping the interest rate r at the benchmark level, we observe a decreasing consumption profile. The ratio between the final and initial consumption level amounts to 0.73. At the same time, the future gets discounted more heavily. For that reason, the individual would c.p. spend less on health compared to the benchmark run. In order to match health investments and life expectancy, a higher value of σ is needed. A higher σ means that marginal utility declines faster with increasing consumption, a feature that motivates the individual to spend more on health in order to increase longevity rather than per-period consumption. The argument holds vice versa for a reduction in ρ. The other parameters remain unchanged or only change mildly.

As expected, the shape of the consumption path does not alter the welfare differences between the test and no-test scenario. In all cases considered, the welfare difference amounts to approximately 1.5 percentage points. The willingness to pay for avoiding the test, however, ranges

from about $ 100.000 to $ 170.000. The intuition for these large deviation in absolute value is as follows. Since σ changes when recalibrating the model, the marginal utility of consumption ($c^{-\sigma}u(D)u(R)$), which is used to convert the welfare differences into monetary equivalents, changes as well. A higher σ implies c.p. a lower marginal utility of consumption. Dividing the same welfare difference by a lower marginal utility then leads to a higher willingness to pay and vice versa.

External Parameters. In this section, we check sensitivity of the results with respect to changes in the externally set parameters shown in Table 1b. To this end, we recalibrate the model following an upward and downward shift in each external parameter and analyze the effect on the willingness to pay for avoiding a test. For descriptive measures like the wage or the interest rate, we apply the standard deviation as our measure of variation, while for estimated parameters (D_0, D_T, μ) we apply the standard error. As far as the latent parameters a and γ are concerned, we vary the parameters by 10 % of its benchmark value. Results are shown in Table A.3 where the first row reiterates the parameter values and results from the benchmark run.

We explain the effects of the parameter changes on the other parameter values and results step by step. We always focus on an increase in the parameter value, the explanation for a reduction in the parameter value holds vice versa. We first investigate a change in the initial deficit level. Motivated by the standard errors reported in Mitnitski et al. (2002b), we vary the initial deficit level from the benchmark value of 0.0273 to 0.278 and 0.269. An increase in D_0 c.p. reduces longevity since the critical deficit level D_T is reached earlier. To reestablish benchmark longevity, the first-order effect is on the health technology, which increases from 0.00146 to 0.00157. As stated above, the welfare difference between the test and no-test scenario remains virtually unchanged. In order to convert this welfare difference into dollar units, the welfare difference is divided by the marginal utility of consumption. Since the recalibration of the model requires a mild reduction in σ, which leads, for given consumption levels, to an increase in the marginal utility of consumption and thus a moderate decrease in the willingness to pay from $ 129.000 to $ 120.000. The opposite effects hold for a change in D_T which we vary, again motivated by the study by Mitnistksi et al. (2002a), by one standard error from 0.1059 to 0.1009 and 0.1112.

A similar effect is observed when varying the force of aging μ. Mitnitski et al. (2002a) report a standard error of 0.001 which is also consistent with estimates by Abeliansky and Strulik

Table A.3: Sensitivity Analysis – External Parameters

case	ρ	σ	α	β	A	θ	untested	tested	Δ Vol
1) bench	0.065	1.16	0.01	0.217	0.00146	0.10	-2.96	-4.45	-129
2) $D_0 \uparrow$	0.065	1.15	0.01	0.220	0.00157	0.10	-2.97	-4.40	-120
3) $D_0 \downarrow$	0.065	1.17	0.01	0.214	0.00137	0.10	-2.96	-4.45	-138
4) $D_T \uparrow$	0.055	1.18	0.01	0.209	0.00136	0.10	-2.96	-4.55	-150
5) $D_T \downarrow$	0.065	1.14	0.01	0.225	0.00156	0.10	-2.97	-4.43	-112
6) $\mu \uparrow$	0.065	1.15	0.01	0.215	0.00155	0.10	-2.96	-4.43	-120
7) $\mu \downarrow$	0.065	1.17	0.01	0.219	0.00137	0.10	-2.96	-4.46	-140
8) $a \uparrow$	0.065	1.18	0.01	0.216	0.00120	0.10	-2.96	-4.46	-150
9) $a \downarrow$	0.065	1.14	0.01	0.218	0.00173	0.10	-2.97	-4.43	-109
10) $\gamma \uparrow$	0.065	1.14	0.01	0.218	0.00126	0.10	-2.97	-4.44	-112
11) $\gamma \downarrow$	0.065	1.18	0.01	0.216	0.00171	0.10	-2.96	-4.45	-144
12) $w \uparrow$ (low)	0.065	1.15	0.01	0.217	0.00146	0.10	-2.96	-4.45	-129
13) $w \downarrow$ (low)	0.065	1.17	0.01	0.217	0.00146	0.10	-2.96	-4.44	-129
14) $w \uparrow$ (high)	0.065	1.12	0.01	0.217	0.00146	0.10	-2.97	-4.44	-129
15) $w \downarrow$ (high)	0.065	1.20	0.01	0.217	0.00146	0.10	-2.69	-4.28	-130
16) $r \uparrow$	0.075	1.16	0.01	0.220	0.00154	0.10	-2.27	-3.75	-120
17) $r \downarrow$	0.055	1.15	0.01	0.215	0.00141	0.10	-3.87	-5.33	-127

Each line of the table considers an upward or downward shift of a parameter by one standard deviation (or one standard error) and shows the recalibrated model parameters and the results for the testing decision. The values in the columns 'untested' and 'tested' are deviations in welfare (expected lifetime utility) in percent from the benchmark run without health shock. Δ Vol refers to the difference in welfare expressed in 1000 dollars.

(2018) for European populations. We thus vary the aging parameter from 0.043 to 0.042 and 0.044. When increasing μ, the health technology parameter has to be increased to match the targeted life expectancy. Through a mild decrease in σ, the marginal utility of consumption increases and the willingness to pay reduces to $ 120.000. A similar (but opposite) effect is found when changing the parameter a that also enters the deficit accumulation equation. We vary the parameter by 10 % of its benchmark value and find that increasing a increases the

willingness to pay to $ 150.000. The variation in the willingness to pay is greater since the recalibration requires a larger variation in σ.

A similar adjustment of σ is also observed when varying γ by 10 % of its benchmark value. Since a higher γ increases the effectiveness of health investments, individuals spend c.p. more on their health. In order to match targeted life-cycle health investments, σ has to be reduced. A smaller σ implies higher marginal utility of consumption and people spend more on consumption rather than on health. Since health investments are more effective now, the other health technology parameter A has to be reduced in order to match the targeted life expectancy. Due to the lower σ, the willingness to pay decreases to $ 112.000.

As far as the wage rate is concerned, BLS (2011) reports a relative standard deviation of 4.76 %. We also consider a variation of 20 % of its benchmark value to illustrate that results are also robust to larger variations in wages. Lines 12 and 13 show the results for the first case and lines 14 and 15 for the latter case. An increase in the wage rate leads to higher health investments such that σ has to be reduced to match the calibration target for health expenditure. The welfare difference and willingness to pay remain unchanged. Naturally, a higher wage rate c.p. increases the willingness to pay. A lower σ and thus a higher marginal utility of consumption, however, offset this effect when converting the welfare difference into monetary equivalents.

Finally, we vary the interest rate r according to the standard deviation reported in Jorda et al. (2019). The relative standard deviation is found to be between 19.17 % and 8.12 %, depending on the type of asset. We take an intermediate value and vary r by 13.65 % of its benchmark value. A higher value of r increases the growth rate of consumption and thus the slope of the consumption path (see consumption Euler in (9)). In order to match a flat consumption profile, ρ needs to be increased accordingly. Since a higher ρ implies higher discounting of the future, the effect of the health shock reduces to 2.27 % in the untested case and to 3.75 % in the tested case. The difference in welfare and willingness to pay, however, change only mildly.

The general takeaway from the sensitivity analysis is that the welfare difference between the test and no-test scenario remains virtually unchanged across specifications. For all considered parameter changes, the difference accounts to around 1.5 percentage points. Despite the rather constant welfare differences, the willingness to pay varies moderately between $ 110.000 and $ 150.000. This is so because the parameter adjustments change marginal utility, which is used to convert the welfare differences into willingness to pay, measured in dollar units.

Appendix D: Comparative Dynamics

Huntington Disease. In this section we perform comparative dynamic analyses in order to account for heterogeneity of individuals in the key parameters. While in the main text we reported results for the average American, we now check how the willingness to pay for avoiding the test changes with respect to the endogenous parameters. To this end, we vary each parameter by 50 % of its calibrated benchmark value while leaving the other parameters unchanged. In contrast to the sensitivity analysis, we thus do not recalibrate the model after a parameter change to fit the behavior and outcomes of the average American, but account for individual-specific heterogeneity in the model parameters. Table A.4 shows the results for this experiment where the first two rows denote percentage deviations from the no-shock scenario. The first

Table A.4: Huntington Disease: Comparative Dynamics – Welfare and Willingness to Pay

	bench	$\rho\uparrow$	$\rho\downarrow$	$\sigma\uparrow$	$\sigma\downarrow$	$\alpha\uparrow$	$\alpha\downarrow$	$\beta\uparrow$	$\beta\downarrow$	$\theta\uparrow$	$\theta\downarrow$
untested	-2.96	-1.21	-7.76	-3.07	-2.95	-2.98	-2.96	-3.11	-2.79	-3.03	-2.83
tested	-4.45	-2.58	-9.17	-4.90	-4.28	-4.47	-4.43	-5.31	-3.53	-4.01	-4.63
Δ VOL	-129	-135	-81	-941	-12	-130	-129	-195	-64	-86	-155

The values in the columns "'untested"' and "'tested"' are deviations in welfare (expected lifetime utility) in percent from the benchmark run without health shock. Δ Vol refers to the difference in welfare expressed in 1000 dollars.

observation is that the largest impact on the welfare reduction relative to the no-shock scenario is triggered by changes in the time preference rate ρ. A higher ρ implies that the future health shock is discounted more heavily and thus has a lower effect on lifetime utility. The parameter β displays the highest effect on the welfare difference between the untested and tested case. While in the benchmark specification the welfare difference amounts to 1.49 %, it ranges from 0.74 % to 2.2 % when varying β by 50 %. Naturally, a higher weight of anticipatory utility translates into a stronger anticipation effect when being tested and thus makes testing less favorable and vice versa. This can be also seen when inspecting the willingness to pay for avoiding the test which ranges from $ 64.000 to $ 195.000. A similar yet somewhat weaker effect can be observed for the case of θ. A higher θ implies that future deficits are discounted more heavily and thus have lower weight in the anticipation stock. Therefore, anticipation in the tested case is less pronounced such that the welfare reduction and the willingness to pay for avoiding the test decrease relative to the benchmark specification. A lower θ has the opposite effect.

The strongest effect on the willingness to pay is observed for changes in σ. As already explained in the sensitivity analysis, σ affects the marginal utility of consumption which converts welfare differences into monetary equivalents. A larger σ implies a lower marginal utility and thus c.p. a higher willingness to pay. Since we consider a substantial change in the parameter, the willingness to pay varies between $ 12,000 and $ 941,000.

7.1. **Breast and Ovarian Cancer.** We focus the comparative dynamic analysis of the breast and ovarian cancer on the threshold wage that hold at the point of indifference with respect to testing. For this purpose, we allow for heterogeneity in the key parameters and determine for each parameter change the threshold wage w^* for which the anticipating individual is indifferent between the untested and tested-and-treated scenario. Table A.5 shows the results when varying each parameter value by 50 % of its benchmark value. The table shows a quantitatively

Table A.5: Breast and Ovarian Caner: Comparative Dynamics – Threshold Wage

	bench	$\rho\uparrow$	$\rho\downarrow$	$\sigma\uparrow$	$\sigma\downarrow$	$\alpha\uparrow$	$\alpha\downarrow$	$\beta\uparrow$	$\beta\downarrow$	$\theta\uparrow$	$\theta\downarrow$
w^*	3283	2,265	5,870	1,963	26,755	3,293	3,272	3,355	3,218	3,240	3,338

The values in the columns refer to the threshold wage w^* for which the anticipating individual is indifferent between testing and non-testing.

significant impact of the time time preference rate ρ. A higher time preference rate increases the threshold wage (to $ 5,870) since individuals discount more heavily the future health shock, thereby reducing the incentive to get treatment. Therefore, individuals remain untested for higher wages as compared to the benchmark specification. The quantitatively largest impact on the threshold wage is observed for changes in σ. A lower σ increases the marginal utility of consumption such that forgoing consumption to pay for the treatment bears a higher welfare burden for the individual. Therefore, the threshold wage below which the individual remains untested rather than being tested and potentially treated increases to $ 26,755. In fact, we find that a woman with a 50 % lower σ that is endowed with the average wage of $ 17,303 would choose to remain untested.

Summarizing, this section shows that heterogeneity in the key parameters may imply changes in the willingness to pay and the threshold wage for different individuals in the population as compared to the calibrated average American.

Appendix E: Adaptation to Anticipation

In order to integrate adaptation to anticipation into the model, we modify the utility function to

$$U(c, D, R) = \left(\frac{\bar{D}}{D}\right)^{\alpha} \left(\frac{S}{R}\right)^{\beta} \cdot u(c) \tag{23}$$

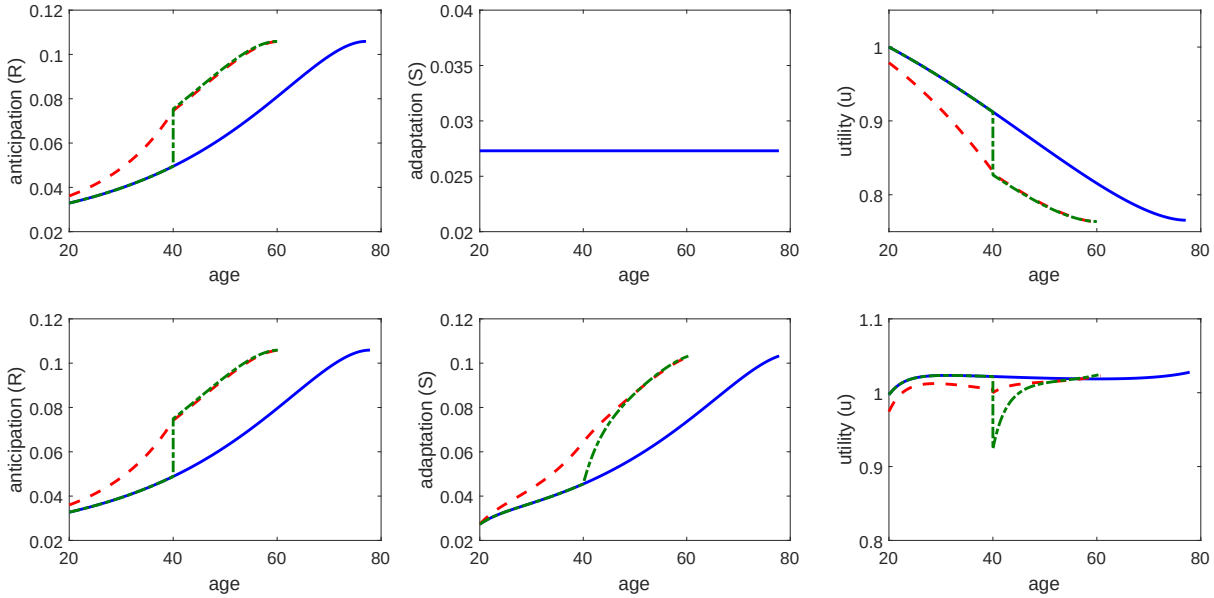
where S represents the state of adaptation. The higher S, the more the individual has adapted to the anticipation of future health deficits, thereby reducing the negative impact of anticipation. We rely on the study by Schünemann et al. (2017a) which investigated adaptation to bad health in the context of the health deficit model. Specifically, we introduce the process of adaptation to anticipation by the differential equation

$$\dot{S} = \xi(R - S) \tag{24}$$

where ξ denotes the speed of adaptation. This way of modeling adaptation is consistent with the modeling of adaptation and habit formation in macroeconomics and economic growth. Note that for spontaneous and perfect adaptation ($\xi \to \infty$), $R = S$ for all t and the individual does not draw any disutility from anticipation. We normalize $R(0) = \bar{R} = \bar{D}$ such that for no adaptation ($\xi = 0$), the utility function nests our benchmark utility function in (5).

Figure A.1 shows the results for the anticipation stock, the adaptation stock and instantaneous utility for anticipating types without adaptation ($\xi = 0$) as in the benchmark specification (upper panels) and with adaptation ($\xi = 0.3$ (Schünemann et al., 2017a)) (lower panels). Blue (dashed) lines refer to the no-shock scenario, red (dashed) lines to the expected-shock scenario, and green (dash-dotted) lines to the unexpected-shock scenario. While the stock of anticipation looks similar for both types, the stock of adaptation is constant for non-adapting types and increases for adapting types. The adaptation stock increases along with the anticipation stock illustrating the fact that individuals get used to the anticipation of future health deficits. In case of the expected shock, the stock of adaptation is higher than in the no-shock scenario already before the shock because the individual knows that the shock will arrive and starts getting used to the idea of developing HD. In case of the unexpected shock, the adaptation stock evolves as in the no-shock scenario before the shock and then increases when the shock arrives and the individual starts anticipating the increased future health deficits.

Figure A.1: Adaptation to Anticipation



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).

The evolution of the adaptation stock directly translates into instantaneous utility. First note that instantaneous utility is not monotonously decreasing as in the benchmark specification because adaptation leads to an increasing consumption path. Recall from the Euler Equation in (9) that anticipation drags down the consumption path. Since adaptation alleviates the impact of anticipation, the flat consumption from the benchmark specification turns to an increasing consumption path when adaptation is included.

Again, instantaneous utility coincides for the unexpected shock and the no-shock scenario before the shock and reduces on impact when the shock sets in. After the shock, however, instantaneous utility recovers due to adaptation. The most important observation from the last panel is, however, the difference between instantaneous utility in the expected-shock vs. the unexpected-shock scenario. As the figure shows, this difference decreases significantly through adaptation. Although the individual still draws disutility from anticipating the health shock, the individual gets used to this anticipation which reduces the willingness to pay for avoiding a test.

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