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Integrating large health shocks into life-cycle models - Modelling impacts of a cancer diagnosis

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Abstract

Most models on the life-cycle utilization of health care consider the expected development of an individual's health. However, health shocks with significant impact (e.g. the onset of a chronic disease or life-threatening accidents) should not be averaged, as they can put the life-course onto a different trajectory. This paper presents an optimal-control framework incorporating a stochastic health shock with individuals allocating their resources to consumption and various types of health care over their life-cycle. We distinguish between general health care and shock-specific prevention, acute, and chronic care. We investigate the optimal decisions (on consumption, saving, and health care) made in anticipation and the optimal reaction to the shock depending on the timing of its arrival. Furthermore, we extend the value of life concept to the multidimensionality of health and the scope for a shock and illustrate our findings by calibrating the model to an individual facing a potential cancer diagnosis in the US. In so doing we also investigate the role of possible bias in the subjective expectation of a health shock.

Keywords: Health care (preventive, acute, chronic), Health shocks, Life-cycle modelling, Optimal control, Regime switch, Value of life (health)

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Classification codes: C61, D15, I12

1 Introduction

Typically, life-cycle models of health behaviour take an ex-ante stance, where a representative individual is subject to the depreciation of a health stock (Grossman, 1972), subject to some mortality process (Ehrlich, 2000; Murphy and Topel, 2006; Hall and Jones, 2007; Kuhn et al., 2015), or subject to the accumulation of deficits (Dalgaard and Strulik, 2014). Individuals fix their life-cycle decisions in perfect anticipation of these processes, regardless of whether they are deterministic or stochastic. In a similar vein, the statistical value of a life is based on an ex-ante evaluation of survival (Shepard and Zeckhauser, 1984; Rosen, 1988; Murphy and Topel, 2006; Hall and Jones, 2007; Kuhn et al., 2010; Kuhn et al., 2011; Kuhn et al., 2015).

While hugely simplifying analytical complexity, the ex-ante perspective amounts to a gross stylization. In reality, health does not develop according to a smooth process shaped by health investments, but is rather subject to smaller or greater shocks. Some of these, such as severe life-threatening diseases (e.g. heart attacks, stroke, or cancer) or accidents as well as chronic diseases (e.g. diabetes or dementia), have the potential to put the entire life-course on a different trajectory. Even if individuals are anticipating the risk of such a shock, the optimization problem changes in as far as the shock subjects the individual to different constraints (e.g. a permanently lower income stream due to disability or a persistently higher mortality risk after the shock) and thus to a different behavioural regime. Consequently, individuals are also prone to alter their behaviour prior to a health shock by taking precautionary saving or preventive actions. The nature and intensity of such actions are shaped by institutions, such as the availability (or not) of health or disability insurance, as well as by policy interventions, such as the subsidization of preventive behaviour.

In this paper, we detail a life-cycle model, allowing us to study large singular shocks to health. Thus, we consider a life-cycle model with endogenous health and survival in the spirit of Kuhn et al. (2015) but allow for the onset of a disease or accident at some random time s. The individual is only aware of the risk, modeled as a hazard rate, that can be influenced by prevention. Thereby we also allow for bias in the expectation of such a health shock due to bounded rationality or limited/incomplete information available to the individual. If the health shock materializes at s, the health of the individual is affected, implying that (i) acute life-saving and/or disability-preventing health care may be required at s; (ii) mortality may be permanently elevated in the course of a chronic disease; (iii) a state of disease/disability emerges that may subsequently affect the individual's utility and earnings; (iv) chronic health care may be required to mitigate the adverse longer-term consequences of the shock. Since we model the life of an individual over time (i.e. age) and since the health shock can set in at any time during the life-course, the health state, assets, and the individual's consumption and health care choices are age and duration dependent.

A detailed analysis, including a complete mapping of state and control variables before and after a shock, a characterization of the linkages of preventive, acute, and chronic care at the point of shock, and a closed-form representation of the underlying valuations is challenging and has at best partially been achieved in previous contributions. Our contribution to the literature thus lies in the provision of such an integrated piece of analysis based on a novel method of

representing and solving optimal control models with stochastic regime change (Wrzaczek et al., 2020).

Our analysis allows us to generalize the value of life (VOL) and apply it to a setting with a health shock. Specifically, we derive the value of health before and after the health shock, the value of prevention, the value of surviving the shock, and the value of morbidity. We can also calculate an expected value of health. As it turns out, these values can be used to write the first order conditions for the different forms of health care in a compact form with a straightforward interpretation. Furthermore, the valuations are instrumental in understanding the dynamics of the different forms of health care. In doing so, we make a clear distinction between preventive health care (lowering the hazard rate directly or indirectly), acute health care (lowering the instantaneous impact of the shock on survival and/or subsequent disability) and chronic health care (lowering the disease/disability state after the shock).

We illustrate the age-duration specific dynamics by focusing on an application of our model to the onset of preventable cancer, such as lung, stomach, and cervical cancer among others (Fink et al., 2026), as one major type of health shock. For this purpose, we calibrate our model to reflect the dynamics of survival and health care spending related to cancer, based on US data. We find that individuals not only shift resources from consumption and general health care towards cancer care after a diagnosis, but also restructure the timing of consumption over the remaining life-cycle due to the impacts of cancer on mortality and utility. Applying our analytical results to this numerical exercise allows us to show that the preventive effects of good health with respect to the risk of cancer onset increase the classic value of life by about 50% at a young age.[1] These effects decrease with time and no longer contribute much to the value of health after the age of 70. Furthermore, we are able to identify the driving forces of consumption and health expenditures during different points in life and compare them in magnitude and timing. We summarize the numerical results in 6 corollaries throughout Section 4.

We conclude our numerical exercise with an analysis of the impact of incomplete information of the risk of cancer onset on the decision making throughout the life-cycle. We find that an overestimation of the hazard leads to an advancement of general health care investments, which reduces the probability of a cancer diagnosis throughout most of the life-cycle, but limits consumption and health expenditures at later stages in life. If on the other hand an individual underestimates the probability of a cancer diagnosis, we find a reduction of preventive health expenditures. However, this allows the individual to accumulate higher levels of wealth and therefore higher capabilities for mitigating a cancer diagnosis after the fact through higher health and consumption expenditures. Still, both individuals are substantially worse of compared to the rational individual with perfect information.

Our contribution to the literature lies in the provision of an analytical framework, rich in detail and nevertheless numerically tractable, that allows a detailed and comprehensive study of the allocations and valuations of distinct types of health care as well as consumption in the context of large shocks to health. Notably, this embraces the whole life-cycle context, al-

[1] We should stress that our model is equally applicable to the case of non-preventable cancer which is tantamount to a setting with an entirely exogenous hazard rate of the health shock.

lowing the assessment and valuation of different treatment pathways composed of preventive, acute and chronic health care, besides general health care, both depending on and (in case of preventive care) driving the timing of the shock. Thus, our analysis is rich enough to provide micro-economic foundations of medically relevant health care allocations in the context of life-changing conditions, such as cardio-vascular disease, cancer, degenerative diseases or debilitating accidents among others (Kul et al., 2012; Bergin et al., 2020; Van Hoeve et al., 2020). A second contribution lies in the generalization of the value of life (e.g. Rosen, 1988; Murphy and Topel, 2006) to a value of health, which embraces different aspects of health: general survival, as embraced by the value of life, but also acute survival (with respect to the shock), morbidity and prevention. Our framework allows an explicit representation of these values and the ways in which they combine to build the general value of health, and, in this, provides life-cycle foundations to medical evaluation (Rowen and Brazier, 2011). It also embraces an explicit analysis of the dependence of these values on the health shock and how they are linked to health care allocations. Finally, the framework allows an explicit study of health-related behaviours in anticipation and response to the shock, including the scope for possible biases in beliefs, which is foundational to health-policy making in these contexts.

The economic literature on health-related shocks to the life-cycle is disperse. The majority of this literature analyses the impact of health-related shocks on labour productivity, health care expenditure, and survival. Palumbo (1999), De Nardi et al. (2010), and Kopecky and Koreshkova (2014), for instance, focus on the impact of risky health care expenditure during old age on precautionary saving; while French (2005) and French and Jones (2011) focus on the impact of health shocks on labour supply and retirement.[2]

Capatina (2015) provides an overall assessment of how four channels of health-related risk (productivity, medical expenditure, time endowment, and survival) impact income inequality and precautionary savings. Common to all of these papers is that they model health risks as a sequence of possibly state-dependent shocks over the life-cycle without emphasizing the scope for "catastrophic" shocks with a propensity to drastically shift the life-cycle trajectory. Furthermore, and importantly, they do not provide scope for individuals to reduce the probability of shocks and/or mitigate their consequences by purchasing preventive and/or curative health care or engaging in other health-related actions. Reichling and Smetters (2015) allow for possibly large shocks when showing that stochastic mortality risks may explain the (rational) abstinence from private annuities when these shocks are correlated with medical expenses. This notwithstanding, the focus of their work is again not on the impact of such shocks on the utilization of health care and resulting health outcomes.[3]

Few works so far have studied how the risk of health shocks weighs on an individual's health investments, either in response to a shock or, more importantly, in anticipation of a shock. Cole et al. (2019) consider a setting in which (curative) health expenditure and labour productivity

[2]The literature on health shocks as motivation for precautionary savings ties in with a large literature on the savings response to (general) life-cycle risks (Eeckhoudt et al. (2005) and Eeckhoudt and Schlesinger (2008)).

[3]Smith and Keeney (2005) examine the valuation of health in a setting where individuals face lotteries over their health and income at distinct phases of their life-cycle. Although these lotteries may involve large shocks, the authors do not model the timing of these shocks; nor do they endogenize the health risks.

are subject to health shocks, the propensity of which depends on health status. The authors examine how the individual's incentive to engage in preventive efforts aimed at improving their health status are shaped by non-discriminatory health insurance and wage-setting. There is no mortality risk, and, as the authors themselves remark in their conclusions, the focus is on small (transitory) shocks to health. In Hugonnier et al. (2013) individual productivity and mortality depend both on an underlying health stock a la Grossman (1972). Individuals can invest in this stock of health, which is assumed to be subject to morbidity shocks following a Poisson process. The key distinction to our approach is that their modelling of uniform health investments and a sequence of (at least principally) transitory shocks (apart from death) does not allow them to discriminate between preventive health care (lowering the probability of a shock) and curative health care (reducing the damage from a shock). Neither do they distinguish between the valuation of preventive care as opposed to the value of curative or chronic care,[4] distinctions which will feature prominently in our work. Finally, Hugonnier et al. (2013) apply their model to understand the relationship between financial investments as opposed to health investments, whereas our focus lies on how the life-cycle allocation of preventive and curative care is shaped by the nature of the health shock. The notion of permanent health shocks is only taken up by Laporte and Ferguson (2007) who consider a version of the Grossman (1972) model in which the health stock is subject to a single irreversible shock, the arrival of which follows a Poisson process. They examine how the nature of this shock bears on the ex-ante path of health investments; however, health is assumed to bear on morbidity and, thus, on period utility but not on survival. Indeed, the length of life is assumed to be exogenous and deterministic.

This leads us to conclude that while the theoretical literature on health shocks has made considerable advances in terms of understanding the consequences of (a sequence of) small shocks for life-cycle patterns of labour supply, income, expenditure, and savings/consumption, comparatively little is known yet about the implications of shocks for the demand for health care and for health behaviours in regard to both their preventive and curative aspect. In particular, this applies to large shocks, such as the onset of severe chronic disease (diabetes, heart disease, cancer) or debilitating accidents, which induce permanent rather than transitory shifts in the mortality, morbidity and income patterns over the remaining life-cycle. These issues lie at the heart of the present work.[5]

Our work also relates to studies examining the costs of uncertainties in the health care or social systems. Caliendo et al. (2019) employ a similar mathematical approach to analyze the welfare costs of uncertainty regarding the timing of Social Security reforms, showing that delays and unclear reform schedules significantly affect individual saving and labor decisions. Caliendo et al. (2023) builds on empirical evidence on how uncertainty about retirement timing influences economic behaviour, and then models retirement as a stochastic decision to assess its impact on consumption and precautionary savings. More broadly, the literature on stochastic

[4] The same applies to the models in Picone et al. (1998), Jung and Tran (2016), Yogo (2016) and Fonseca et al. (2021).

[5] From an empirical perspective, the behavioural adjustments and consequences for income and well-being of health shocks have been studied extensively. Adjustments have been studied in regard to saving and consumption (Bíró (2013)) as well as in regard to health behaviours, in particular smoking (e.g. Smith et al., 2001; Khwaja et al., 2006; Marti and Richards, 2017).

switches and shocks is gaining popularity across several disciplines, including health economics, environmental and climate economics (see e.g., Tsur and Zemel (2021) for a recent overview), as well as management and operations research (for an overview see Freiberger et al. (2025)).

The remainder of the paper is structured as follows. The next section contains a description of the model. Section 3 presents the analytical solution, involving in particular the derivation of various value (of health) terms from the set of relevant shadow prices (Subsection 3.1) and their subsequent employment in the first-order conditions (Subsection 3.2). Section 4 then proceeds to present the foundations for the numerical analysis, with Subsections 4.1 through 4.3 setting out data, functional specifications, as well as details of the solution strategy. Section 5 examines the numerical solution starting with a comparison of the output of the calibrated model with the data in Subsection 5.1. Subsections 5.2 and 5.3 break down the numerical consumption, expenditure, health, and survival profiles and provide numerical valuations of different dimensions of health. An extensive decomposition analysis of the driving forces behind the optimal health expenditure profiles and valuations of health are provided in Subsection 5.4. Subsection 5.5 studies the life-cycle allocation for boundedly rational individuals who are subject to bias in their subjective expectation of the arrival rate of the health shock. Section 6 concludes.

2 Model

In this section, we present a framework that integrates a large shock to the health of an individual into a life-cycle model. For the timing of the shock we make the following assumptions (which apply throughout the paper).

(A1) A large health shock occurs at some age s, which is random. The probability rate of arrival is known by the individuals up to a multiplicative constant that represents possible bias in the individual's expectation.

(A2) The event at s occurs only once, so that the life-time of an individual can be separated into a stage before and a stage after s.

These two assumptions allow us to formulate the model as a stochastic optimal control model with a random stopping time (see Boukas et al., 1990) and analyze it in terms of a vintage optimal control model (see Wrzaczek et al., 2020). In both stages the individual chooses consumption and different types of health care in order to optimize (expected) life-time utility. Denoting by $t \in [0, T]$ the age of the individual, where $T > 0$ gives the maximum feasible age, we then have that $s \in [0, T]$ is the age at which the model switches from stage 1 (where $t < s$) to stage 2 (where $t > s$).

Using the index i ($i = 1, 2$) to denote the stage of the life-cycle, we assume that the survival probability $S_1(t)$ in stage 1 is determined by the stage-1 mortality rate $\mu^1(\cdot)$.[6] This rate is assumed to be equal to the base mortality rate $\mu^b(t, S_1, b_1)$, which depends on age t, decreases in survival $S_1(t)$, and decreases in the quantity $b_1(t)$ of general health care subject to decreasing returns. Here, $b_1(t)$ is a generic measure of all health care that is unrelated to the condition(s)

[6] Note that we will omit t wherever it is not of particular importance.

relating to the health shock. As described in Kuhn et al. (2015), the high correlation between survival and health implies that $S_1(t)$ can be interpreted as a proxy health state. We thus capture the negative dependency of mortality on health by including $S_1(t)$ as an argument in μ^b.[7] Altogether, the survival probability evolves[8] with age according to

$$\dot{S}_1(t) = -\mu^1(t, S_1(t), b_1(t)) \cdot S_1(t) = -\mu^b(t, S_1(t), b_1(t)) \cdot S_1(t), \qquad S_1(0) = 1. \tag{1}$$

The individual maximizes expected life-time utility

$$\mathbb{E}_s \left[\int_0^s e^{-\rho t} S_1(t) u^1(c_1(t))\, dt + e^{-\rho s} V^* \left(S_1(s), A_1(s), s\right) \right]. \tag{2}$$

The first term contains the aggregated utility from birth up to age s, where the period utility $u^1(c_1(t))$ from consumption $c_1(t)$ is weighted by survival $S_1(t)$ and a discount factor $e^{-\rho t}$ (the discount rate ρ is assumed to be exogenous).[9] The function $u^1(\cdot)$ is assumed to fulfill the assumptions of positive but diminishing marginal utility as well as the Inada conditions if consumption tends to zero or infinity. The second part of the expected life-time utility denotes the discounted aggregated utility over the remaining life-time conditional on the individual having suffered a health shock at age s. Here, $V^*(\cdot)$ denotes the optimal value (i.e. the value function) of the optimal control problem in the second stage which depends not only on the age s at the occurrence of the shock, but also on the survival/health state $S_1(s)$ and the assets $A_1(s)$ at that point. The expected value is built with respect to the random variable s. The likelihood of a shock occurring at a given age can be influenced by the individual through investments in preventive care h_1. Let the probability distribution of s be defined by $\mathcal{F}(t) = \mathbb{P}[s \leq t]$. The probability distribution of s can be characterized by the hazard rate η of the shock, which is generally defined by

$$\eta(t) = \frac{\mathcal{F}'(t)}{1 - \mathcal{F}(t)} \qquad \text{and} \qquad \mathcal{F}(t) = 1 - e^{-\int_0^t \eta(a) da}. \tag{3}$$

More specifically, we assume the hazard rate

$$\eta(t) = \eta(t, S_1(t), h_1(t), \pi) = \pi \cdot \widetilde{\eta}(t, S_1(t), h_1(t)) \tag{4}$$

to depend on age t, to decrease in survival/health $S_1(t)$, and to decrease in the utilization of preventive care $h_1(t)$ (again subject to diminishing returns). Note that h_1 has no direct impact on the health state S_1 of the individual and solely corresponds to measures reducing the propensity of the health shock.[10] For example, one might think of h_1 as the propensity to

[7] This formulation might be surprising at first sight, but following the explanations in Freiberger and Kuhn (2020) and under some weak assumptions it is consistent with a deficit accumulation model as developed in Schuenemann et al. (2017), or a classic Grossman-type (Grossman, 1972) model with a monotonously depreciating health stock over the life-cycle.

[8] In technical terms, the survival probability corresponds to a stock variable as its development is described through an ODE, while the health expenditures are a flow variable, which can be adjusted independently at each point in time.

[9] This formulation reaches back to Yaari (1965).

[10] Given that we only consider one singular large health shock, there is no role for preventive care after the

vaccinate against an infectious disease or as the propensity to attend precautionary screenings for cancer or heart disease. Furthermore, to obtain the subjective hazard rate, which the individual applies in their personal decision-making, the objective hazard rate $\widetilde{\eta}(t, S_1(t), h_1(t))$ is adjusted for a parameter π, representing possible bias in decisions due to imperfect information or bounded rationality. For $\pi = 1$ individuals are perfectly informed and rational in their decision-making, while they act completely oblivious with respect to a potential health shock for $\pi = 0$. For $\pi \in (0, 1)$ individuals underestimate the risk of a health shock, for $\pi > 1$ they assume a health shock to be more likely than objectively estimated.

The asset dynamics in stage 1 are given by

$$\dot{A}_1(t) = (r(t) + \theta\bar{\mu}(t))A_1(t) + w^1(t) - c_1(t) - p^b(t)b_1(t) - p^1(t)h_1(t) + B(t, \theta), \tag{5}$$

$$A_1(0) = 0 \qquad \text{and} \qquad A_1(T) = 0; \tag{6}$$

where assets $A_1(t)$ are partially annuitized and generate a return $(r(t) + \theta\bar{\mu}(t))$, with $r(t)$ and $\theta\bar{\mu}(t)$ denoting the interest rate and the mortality risk premium on annuities, respectively; with $w_1(t)$ denoting stage-1 earnings; with $p^b(t)$ and $p^1(t)$ denoting the prices for general and specific preventive health care, $b_1(t)$ and $h_1(t)$, respectively; and with the price for consumption $c_1(t)$ being normalized to one. Additionally, the individuals receive accidental bequests $B(t, \theta)$ throughout their life-cycle.[11] The parameter $\theta \in [0, 1]$ describes the degree of imperfection in the annuity market. While $\theta = 1$ corresponds to a perfect annuity market, for $\theta = 0$ all assets after death get redistributed through a lump-sum accidental bequest. This approach follows the work by Heijdra and Mierau (2012) and will discuss the imperfect annuity market in more detail in Section 4.1.2. Finally, we assume zero assets at birth and at the end of the maximum life-span T as usual.

In stage 1, the individual chooses the (non-negative) control variables $c_1(t)$, $b_1(t)$ and $h_1(t)$ so as to maximize the objective function (2) subject to the constraints (1) and (4)–(6).

Stage 2 is modeled in a similar vein, but we now consider a disease stock $M(t, s)$ as an additional state variable that bears on the individual's utility and constraints and reflects the long-term impacts specifically related to the health shock. For all variables in the second stage, t and s describe the age of the individual and the age at which the health shock has occurred, respectively. We assume that the condition setting in at s is associated with a specific mortality $\mu^m(t, s, M(t, s))$, depending on age, the time of shock (or onset of disease), and the disease stock. In addition, the individual continues to be subject to the base mortality $\mu^b(t, S_2(t, s), b_2(t, s))$ identical to the one in the first stage. With the total mortality rate μ^2 of the individual in the second stage being the sum of μ^b and μ^m, the dynamics of stage-2 survival $S_2(t, s)$ can be written as

$$\dot{S}_2(t, s) := \frac{dS_2(t, s)}{dt} = -\mu^2(t, s, S_2(t, s), b_2(t, s), M(t, s))S_2(t, s) \tag{7}$$

health shock in stage 2.

[11] From an individual point of view the annuity rate and accidental bequests are exogenous. In Section 4.1.3 we will expose the redistribution of individual assets after death in greater detail.

$$= -\left[\mu^b(t, S_2(t,s), b_2(t,s)) + \mu^m(t, s, M(t,s))\right] S_2(t,s). \tag{8}$$

The disease stock $M(t,s)$ evolves according to

$$\dot{M}(t,s) := \frac{dM(t,s)}{dt} = f(t, s, M(t,s), h_2(t,s)) \tag{9}$$

where f depends on age, the age at shock, the disease stock itself, and disease-specific (chronic) health care. While general health care after the shock $b_2(t,s)$ is still associated with reductions of the base mortality rate, the chronic health care $h_2(t,s)$ directly aims to lower the disease stock M (again subject to diminishing marginal effects) and has no other effects on general health/survival. Our general formulation of the disease dynamics allows for a range of different interpretations. These include, in particular, the cases of (i) an accident or acute disease at the point of shock, which leaves the individual disabled initially but where a natural healing process, in some cases supported by health care, leads to a gradual reduction of $M(t,s)$ (understood to be the extent of disability); and (ii) a progressive disease, such as cancer, diabetes or Alzheimer dementia, where $M(t,s)$ tends to increase unless kept in check or lowered by the consumption of health care.

To further account for the negative consequences of the onset of disease (or disability), $M(t,s)$ is assumed to lower stage-2 utility u^2, and stage-2 earnings, w^2, i.e.

$$\frac{\partial u^2(c_2, M)}{\partial M} \leq 0, \qquad \frac{\partial^2 u^2(c_2, M)}{\partial M^2} \leq 0, \qquad \frac{\partial w^2(t, s, M)}{\partial M} \leq 0, \qquad \frac{\partial^2 w^2(t, s, M)}{\partial M^2} \leq 0.$$

Finally, we assume the (initial) level of the disease state at the time of shock $t = s$ to be a decreasing function of the general health state, as proxied by $S_1(s)$, and of the one-time (acute) health care $d(s)$, i.e. $M(s,s) = M^0(S_1(s), d(s))$. We assume that acute care helps reduce initial deficits in addition to increasing the probability of surviving the health shock (see equation (12)). In the event of cardiac arrest, for instance, effective emergency care also improves the long-term prognosis (see Hassager et al., 2018).

The dynamics of stage-2 assets $A_2(t,s)$ are similar to those in stage 1 with the following differences. First, earnings are not exogenous but depend on $M(t,s)$, as detailed above. Second, expenditures for chronic health care, purchased at a price $p^2(t)$, substitute for preventive health care. Third, the initial stage-2 assets $A_2(s,s)$ (just after the shock) are equal to the stage-1 assets $A_1(s)$ (just before the shock) net of the expenditure for acute care that is purchased at a price $p^d(s)$. Thus,

$$\begin{aligned}\dot{A}_2(t,s) = (r(t) + \theta\bar{\mu}(t))A_2(t,s) + w^2(t, s, M(t,s)) - \\ - c_2(t,s) - p^b(t)b_2(t,s) - p^2(t)h_2(t,s) + B(t,\theta)\end{aligned} \tag{10}$$

$$A_2(s,s) = A_1(s) - p^d(s)d(s), \qquad A_2(T,s) = 0. \tag{11}$$

According to the last boundary condition, assets have to equal 0 at the end of life regardless of when the shock has occurred. The aggregated utility during stage 2 consists of the present

value of the expected (i.e. survival weighted) utility stream over the remaining life-course

$$P(S_1(s), d(s)) \cdot \int_s^T e^{-\rho t} S_2(t,s) u^2(c_2(t,s), M(t,s))\, dt, \tag{12}$$

where $P(S_1(s), d(s)) \in [0,1]$ is the probability that the individual survives the health shock, which increases with the stage-1 health state $S_1(s)$ and the quantity of acute care $d(s)$ (subject to diminishing returns). Note that for $P(\cdot) < 1$ the individual potentially does not survive the shock (e.g. an accident, a cardiac event, or a stroke), whereas $P(\cdot) = 1$ would reflect a disease that is not mortal upon its onset at s but only potentially so over time (e.g. cancer, diabetes, Alzheimer's disease). Our model also includes the case of health shocks that cause instantaneous death, $P(\cdot) = 0$. From now on, we will refer to $P(\cdot)$ as the "continuation probability".

Note that our modelling framework does not adopt an alternative approach to Yaari (1965); rather, it extends the model to a more general, larger *outer* problem that integrates life-cycle trajectories from multiple Yaari-type subproblems, to which the individual switches at the (stochastic) onset of cancer. However, we depart from Yaari (1965) by relaxing the assumption that individuals have perfect knowledge of their mortality process. We assume that awareness of the mortality process is blurred both by a large health shock (which increases the individual's mortality risk) and by a bias in the perception of the shock's occurrence. Together, these factors constitute a deviation from Yaari's key permanent foresight assumption. The implications of relaxing this assumption have been addressed, for example, by Caliendo et al. (2020) through the annuity market channel, which provides a complementary perspective to the approach taken in this paper.

The complete model is summarized in Equations (13) - (20).

$$\max_{c_1(t),h_1(t),b_1(t)\geq 0} \quad \mathbb{E}_s\left[\int_0^s e^{-\rho t} S_1(t) u^1(c_1(t))\, dt + e^{-\rho s} V^*\left(S_1(s), A_1(s), s\right)\right] \tag{13}$$

$$\dot{S}_1(t) = -\mu^1(t, S_1(t), b_1(t)) S_1(t), \tag{14}$$

$$\dot{A}_1(t) = (r(t) + \theta\bar{\mu}(t)) A_1(t) + w^1(t) - c_1(t) - p^b(t) b_1(t) - p^1(t) h_1(t) + B(t,\theta), \tag{15}$$

$$S_1(0) = 1, \qquad A_1(0) = 0, \qquad A_1(T) = 0 \tag{16}$$

$$V^*\big(S_1(s), A_1(s), s\big) := \max_{\substack{c_2(t,s), h_2(t,s),\\ b_2(t,s), d(s)\geq 0}} P(S_1(s), d(s)) \cdot \int_s^T e^{-\rho t} S_2(t,s) u^2(c_2(t,s), M(t,s))\, dt \tag{17}$$

$$\dot{S}_2(t,s) = -\mu^2\left(t, s, S_2(t,s), b_2(t,s), M(t,s)\right) S_2(t,s), \qquad S_2(s,s) = S_1(s) \tag{18}$$

$$\begin{aligned}\dot{A}_2(t,s) = {} & (r(t) + \theta\bar{\mu}(t)) A_2(t,s) + w^2(t,s,M(t,s)) - c_2(t,s) - p^b(t) b_2(t,s) - \\ & - p^2(t) h_2(t,s) + B(t,\theta), \qquad A_2(s,s) = A_1(s) - p^d(s) d(s), \qquad A_2(T,s) = 0\end{aligned} \tag{19}$$

$$\dot{M}(t,s) = f(t,s,M(t,s),h_2(t,s)), \qquad M(s,s) = M^0(S_1(s), d(s)) \tag{20}$$

Problem (13)-(16) can be interpreted as an optimal control model with random stopping time (Boukas et al., 1990). For the analysis and for the numerical solution we transform the model

into a vintage optimal control model, as this offers additional economic insights as well as an established numerical solution method (see Veliov, 2003).[12] For the theoretical background and other examples of the transformation method we refer to Wrzaczek et al. (2020). As the presentation of the model in vintage optimal control form is not immediately instructive, we relegate it to the Online-Appendix. Here, we only note that the vintage formulation implies that all second-stage variables are indexed by both age t and the time of the shock s, which can be interpreted as the arrival-date of a (potential) vintage of the remaining life-course (in disease). Indeed, the notation we have introduced earlier meets this criterion.

The transformation includes the introduction of two auxiliary variables which we will subsequently employ in our calculations and for which interpretations are straightforward. First, $Z_1(t)$ denotes the (subjective) probability that an individual assigns to not having suffered a health shock up to age t. The development of Z_1 can be formulated using the subjective hazard rate $\eta(t, S_1(t), h_1(t), \pi)$:

$$\dot{Z}_1(t) = -\eta(t, S_1(t), h_1(t), \pi) Z_1(t) \qquad , \qquad Z_1(0) = 1. \tag{21}$$

Second, we need the auxiliary variable $Z_2(s)$, which is defined by

$$Z_2(s) = Z_1(s) \cdot \eta(s, S_1(s), h_1(s), \pi) \cdot P(S_1(s), d(s)), \tag{22}$$

and can be interpreted as the joint (subjective) density of experiencing *and* surviving a shock at age s. For further reference, Table 1 summarizes the control and state variables in the two life-cycle stages. Furthermore, Figure 1 provides a graphical illustration of all interconnections in the modelling framework.

Table 1: Summary of all state and control variables in the basic framework

Control variables	***Stage 1***	***Stage 2***	***Shock time* s**
Consumption	$c_1(t)$	$c_2(t,s)$	-
General Health investments	$b_1(t)$	$b_2(t,s)$	-
Prevention expenditures	$h_1(t)$	-	-
Chronic care	-	$h_2(t,s)$	-
Acute treatment	-	-	$d(s)$
State variables			
Survival probability	$S_1(t)$	$S_2(t,s)$	$S_2(s,s) = S_1(s)$
Assets	$A_1(t)$	$A_2(t,s)$	$A_2(s,s) = A_1(s) - p^d(s)d(s)$
Survival in good health	$Z_1(t)$	-	-
Joint "probability" of shock *and* survival at s	-	$Z_2(s)$	$Z_2(s) = P(S_1(s), d(s))Z_1(s)\times$ $\times\eta(s, S_1(s), h_1(s), \pi)$
Severity of health deficits	-	$M(t,s)$	$M(s,s) = M^0(S_1(s), d(s))$

[12] The solution process is, nevertheless, not trivial as Veliov (2003) sets up a general framework, requiring multifaceted adaptations for the solution of specific problems.

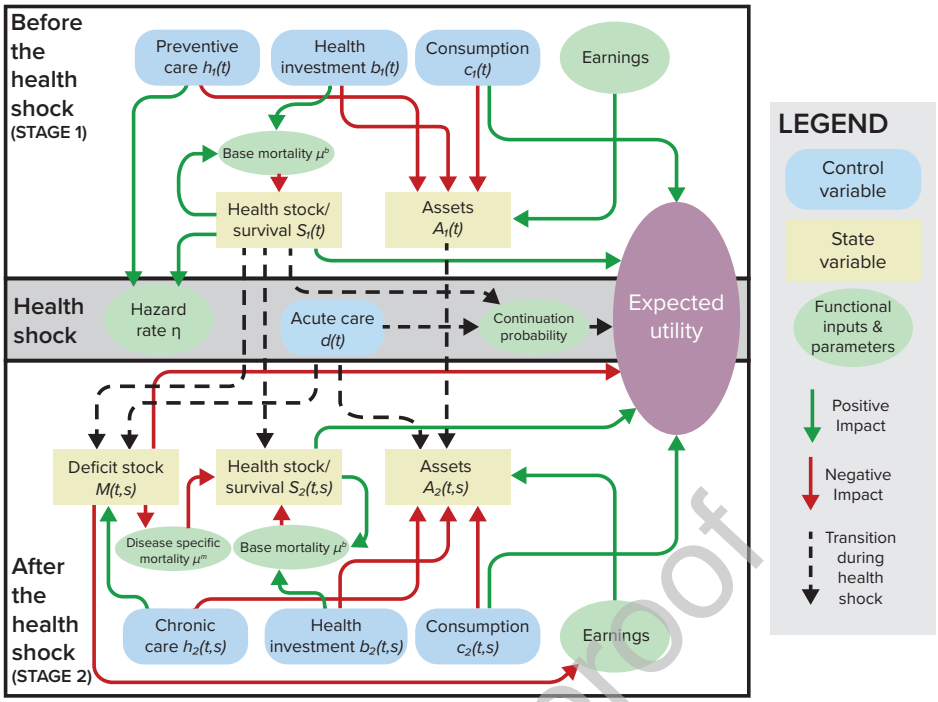

Figure 1: Flow diagram of the model structure

3 Analytical results

For the vintage optimal control model, we apply the Maximum Principle presented in Feichtinger et al. (2003) to arrive at a set of necessary optimality conditions. The differential equations and transversality conditions for the adjoint variables, together with the state equations (with initial and boundary conditions) and the first order optimality conditions (at every point in time/age and every age at shock occurrence), are used to find the optimal solution. In the following section, we employ the first order conditions and the adjoint variables to identify and characterize the behavioural channels of the model.

3.1 Valuations of Health

Following Rosen (1988), Murphy and Topel (2006), Hall and Jones (2007), Kuhn et al. (2015) and others we investigate the individual willingness to pay for changes in health. However, while the original work focuses on a reduction in mortality risk, i.e. the value of life (VOL), we distinguish between the willingness to pay for changes in a range of different aspects of health.

Definition 1. For the analysis of problem (13) - (20), we define the following valuations of health, which we will use throughout. Here, $\mathcal{V}$ denotes the value function of problem (13) - (20).

Value of health ψ_H^i in stage $i = 1, 2$: Willingness to pay for a reduction in the mortality rate (depreciation rate of the survival stock) μ^i in stage $i = 1, 2$.

$$\psi_H^i := \left(-\frac{d\mathcal{V}}{d\mu^i}\right) / \left(\frac{d\mathcal{V}}{dA_i}\right).$$

Value of prevention ψ_P: Willingness to pay for a reduction in the risk of a health shock by

reducing η, $\psi_P := \left(-\frac{d\mathcal{V}}{d\eta}\right) / \left(\frac{d\mathcal{V}}{dA_1}\right)$.

Value of acute survival ψ_{AS}: Willingness to pay for an increase in the probability P of surviving the shock, $\psi_{AS} := \left(\frac{d\mathcal{V}}{dP}\right) / \left(\frac{d\mathcal{V}}{dA_2}\right)$.

Value of morbidity ψ_M: Willingness to pay for a reduction in the disease/disability stock M, $\psi_M := \left(-\frac{d\mathcal{V}}{dM}\right) / \left(\frac{d\mathcal{V}}{dA_2}\right)$.

In addition, it is convenient to define the value of second-stage life as

$$\psi_L^2(t,s) = \int_t^T R(t,\tau)\frac{u^2(\tau,s)}{u_{c_2}^2(\tau,s)}\,d\tau, \qquad \text{with} \qquad R(t,\tau) := \exp\left(-\int_t^\tau r(\tau') + \theta\bar{\mu}(\tau')d\tau'\right), \tag{23}$$

i.e. as the present value at age t of the stream of consumer surplus over the remaining life-course in stage 2, with the return on annuities being applied as discount rate. Note the similarity to the 'conventional' value of life in earlier works (e.g. Kuhn et al., 2015). Based on this, Proposition 1 presents an explicit analytical formulation of the valuations defined above.

Proposition 1. *Assume the existence of optimal trajectories of consumption and (the various) health investments in both stages of the individual life-cycle model (13) - (20) together with an interior solution for the consumption profiles c_1 and c_2. The valuation terms in Definition 1 can then be written as follows.*[13]

Stage-2 valuations:

$$\psi_H^2(t,s) = \int_t^T R_H^2(t,\tau,s)\frac{u^2(\tau,s)}{u_{c_2}^2(\tau,s)}\,d\tau, \tag{24}$$

$$\psi_M(t,s) = \int_t^T R_M^2(t,\tau,s)\left\{\mu_M^2(\tau,s)\psi_H^2(\tau,s) - w_M^2(\tau,s) - \frac{u_M^2(\tau,s)}{u_{c_2}^2(\tau,s)}\right\}\,d\tau. \tag{25}$$

Stage-1 valuations:

$$\psi_{AS}(t) = \psi_L^2(t,t)/P(S_1(t),d(t)), \tag{26}$$

$$\psi_P(t) = \int_t^T R_P^1(t,\tau)\left\{\frac{u^1(\tau)}{u_{c_1}^1(\tau)} + \eta(\tau)P(\tau)\frac{u_{c_2}^2(\tau,\tau)}{u_{c_1}^1(\tau)}\psi_L^2(\tau,\tau)\right\}\,d\tau - \\ - \frac{u_{c_2}^2(t,t)}{u_{c_1}^1(t)}P(t)\psi_L^2(t,t). \tag{27}$$

$$\psi_H^1(t) = \int_t^T R_H^1(t,\tau)\left\{\frac{u^1(\tau)}{u_{c_1}^1(\tau)} - \eta_{S_1}(\tau)S_1(\tau)\psi_P(\tau) + \eta(\tau)P(\tau)\frac{u_{c_2}^2(\tau,\tau)}{u_{c_1}^1(\tau)}\times \right. \\ \left. \times\left[\psi_H^2(\tau,\tau) + P_{S_1}(\tau)S_1(\tau)\psi_{AS}(\tau) - S_1(\tau)M_{S_1}^0(\tau)\psi_M(\tau,\tau)\right]\right\}\,d\tau, \tag{28}$$

[13] For notational convenience, we omit the state and control arguments in all functions and just indicate at which time-point the state and control values should be evaluated, e.g. $u_{c_2}^2(\tau,s) \equiv u_{c_2}^2(c_2(\tau,s),M(\tau,s))$.

The various discount factors are defined by

$$R_P^1(t,\tau) := R(t,\tau)\cdot\exp\left(-\int_t^\tau \eta(\tau')P(\tau')\frac{u_{c_2}^2(\tau',\tau')}{u_{c_1}^1(\tau')}d\tau'\right), \tag{29}$$

$$R_H^1(t,\tau) := R(t,\tau)\cdot\exp\left(-\int_t^\tau \mu_{S_1}^1(\tau')S_1(\tau')+\eta(\tau')P(\tau')\frac{u_{c_2}^2(\tau',\tau')}{u_{c_1}^1(\tau')}d\tau'\right), \tag{30}$$

$$R_H^2(t,\tau,s) := R(t,\tau)\cdot\exp\left(-\int_t^\tau \mu_{S_2}^2(\tau',s)S_2(\tau',s)d\tau'\right), \tag{31}$$

$$R_M^2(t,\tau,s) := R(t,\tau)\cdot\exp\left(\int_t^\tau f_M(\tau',s)d\tau'\right). \tag{32}$$

Proof. Following Rosen (1988) we derive the following terms for the valuations:

$$\psi_H^1(t)=\frac{S_1(t)\lambda_S(t)}{\lambda_A(t)} \quad , \quad \psi_H^2(t,s)=\frac{S_2(t,s)\xi_S(t,s)}{\xi_A(t,s)} \quad , \quad \psi_M(t,s)=\frac{-\xi_M(t,s)}{\xi_A(t,s)}$$
$$\psi_P(t)=\frac{Z_1(t)(\lambda_Z(t)-P(t)\xi_Z(t,t))}{\lambda_A(t)} \quad , \quad \psi_{AS}(t)=\frac{Z_1(t)\eta(t)\xi_Z(t,t)}{\xi_A(t,t)}.$$

The remaining proof makes use of the FOC for consumption and the integral representations of the costate variables. Following tedious calculations which can be found in the Online-Appendix delivers the results presented in the proposition. □

Although the above expressions look involved, the individual terms can be assigned to the forward and backward incentives that shape the qualitative behaviour of the optimal solution, as discussed in the following.

3.1.1 Value of health in the second stage: $\psi_H^2(t,s)$

The value of health in the second stage ψ_H^2 (see equation (24)) is closely related to the value of life, i.e. the discounted value of consumer surplus over the remaining life-cycle, as defined in equation (23). The only difference is that the value of health takes into additional account a term $\mu_{S_2}^2 S_2$ in the discounting function R_H^2, reflecting that better health, as measured by S_2, contributes to lower mortality over the remaining life-course. Given that $\mu_{S_2}^2 < 0$, the new term decreases the discount factor and, thereby, raises the value of health over and above the conventional value of life. Intuitively, this reflects the additional value of health/survival as an asset that by lowering mortality yields a return in terms of additional consumer surplus over the remaining life-course.

3.1.2 Value of acute survival: $\psi_{AS}(t,s)$

The value of marginally increasing the continuation probability, ψ_{AS}, (see equation (26)) at the time of the shock directly corresponds to the conventional value of life in the second stage, ψ_L^2.[14] The weighting of ψ_L^2 by $1/P(t)$ implies that the willingness to pay for acute survival increases with the risk of not surviving the health shock.

[14] The discounting term $\mu_{S_2}^2 S_2$ is not present here, as a marginal change in $P(t)$ has no effect on subsequent survival S_2.

3.1.3 Value of morbidity: $\psi_M(t,s)$

The willingness to pay for a (marginal) reduction in the disease/disability stock, ψ_M, (see equation (25)) depends on the interaction of three effects: (i) the first part $\mu_M^2\psi_H^2$ captures the impact of the disease/disability stock on mortality or, equivalently, on the depletion of the health stock. A marginal increase in the disease stock increases the mortality risk by μ_M^2, which consequently leads to a change in second-stage survival, S_2, which is valued at the willingness to pay ψ_H^2. (ii) the part $-w_M^2$ contains the impact of the disease/disability on earnings, which directly translates into a willingness-to-pay to lower the disease/disability stock. (iii) The last part $-\frac{u_M^2}{u_{c_2}^2}$ measures the reduction in consumer surplus in the presence of disease/disability, or, equivalently, the marginal rate of substitution between consumption and degree of illness/disability. The greater the marginal impact of disability on utility, the greater the willingness-to-pay for reductions in morbidity. Finally, we have to adjust the standard discount rate R for the direction and speed of disease progression, as measured by the impact f_M of the disease stock on its own accumulation. Thus, the value of future changes in morbidity tends to be discounted more heavily if the disease is accelerating, i.e. $f_M > 0$, and less heavily if it is decelerating, i.e. $f_M < 0$.

3.1.4 Value of prevention: $\psi_P(t)$

The willingness to pay for a reduction in the hazard rate of the health shock, ψ_P, (see equation (27)) is equivalent to the net value of remaining in (the healthy) stage 1 as opposed to transitioning to (the diseased/disabled) stage 2. Accordingly, the integral term measures the value of remaining in stage 1, which in itself is composed of two distinct factors. The first part $\frac{u^1(c_1(\tau))}{u_{c_1}^1(c_1(\tau))}$ amounts to the consumer surplus for each year that continues to accrue in stage 1, whereas the second part adds the expected value of stage 2 utility should a transition occur at rate $\eta(\tau)$ in some future year $\tau > t$. Note that this value corresponds to the stage-2 value of life, ψ_L^2, weighted with the probability $P(\tau)$ of surviving a health shock at τ. As ψ_L^2 is counted in units of stage-2 consumption, a conversion into units of stage-1 consumption takes place by multiplication with $\frac{u_{c_2}^2(t,t)}{u_{c_1}^1(t)}$. Notably, the discount factor R_P^1 applied to the utility stream associated with remaining in stage 1 through t now takes into account the (weighted) risk of a transition into stage 2.

The value of remaining in stage 1 (integral part in equation (27)) is then offset against the value of switching to stage 2 at t (last term in (27)). This value corresponds to the stage-2 value of life, again weighted with the survival probability, $P(t)$, at the time of the shock and converted into stage-1 values. Note that the value of avoiding a deadly health shock, for which $P(\tau) = 0$ for all $\tau \in [t, T]$, exactly corresponds to the conventional stage-1 value of life.

3.1.5 Value of health in the first stage: $\psi_H^1(t)$

After having introduced all other valuations of health we can finally analyze the value of health in the first stage ψ_H^1, which contains multiple terms presented above (see equation (28)). In total we separate five distinct impact channels. (i) The stream of stage-1 consumer surplus,

$\frac{u^1}{u^1_{c_1}}$, that underlies the value of survival. (ii) The value of health/survival in reducing the hazard rate and, thus, preventing the shock, $\eta_{S_1} S_1 \psi_P$. The remaining three parts capture the value of stage-1 health for reaching and living through a stage 2 life-cycle conditional on surviving a shock at age τ. Thus, all three factors are weighted with ηP, the joint probability of experiencing and surviving the health shock at τ as well as with the conversion factor $\frac{u^2_{c_2}}{u^1_{c_1}}$. (iii) The term ψ^2_H measures the direct value of health upon entering stage 2; (iv) the term $P_{S_1} S_1 \psi_{AS}$ captures the value of stage-1 health in enhancing acute survival following a shock; and (v) the term $M^0_{S_1} S_1 \psi_M$ captures the value of stage-1 health in lowering the intensity of disease/disability and, thus, morbidity at the point of the shock. We conclude by noting that the discount factor R^1_H includes the long-run impacts of survival on future mortality $\mu^1_{S_1} S_1$ like R^2_H as well as the (weighted) risk of entering stage 2 upon survival of a shock, $\eta P \frac{u^2_{c_2}}{u^1_{c_1}}$.

3.1.6 Expected value of health

From an ex-ante stance the future development of the individual's health status is stochastic. Thus, the value of health in its general form, comparable to the well-known value of life in other contributions, should account for this uncertainty. Hence we define the expected value of health as the expectation of the different values of health in stages 1 and 2, weighted with the corresponding probabilities. This is summarized in the following definition.

Definition 2. Assuming the existence of optimal trajectories of consumption and (the various) health investments in both stages of the individual life-cycle model (13)-(20), the expected value of health can be defined as

$$\Psi_H(t) := Z_1(t)\psi^1_H(t) + \int_0^t Z_2(s)\psi^2_H(t,s)ds. \tag{33}$$

Following from this definition, the expected value of health at age t can also be interpreted as an averaged value of health across individuals who have not experienced a shock up to age t and individuals who, at age t, have experienced different stages of disease progression following a shock experienced at an earlier age $s < t$.

Note that such a measure is useful when it comes to assessing the value of health at population level.[15] In particular for normative purposes, it is considered unethical to distinguish individuals according to their value of life. Thus, the value of life is typically averaged across income strata, health states and often age (for an exception see Aldy and Viscusi, 2008). The expected value of health would provide a value that is averaged across the possible health states at age t, including the potential health-driven inequality in earnings.

3.2 First order optimality conditions

The first-order optimality conditions (FOCs) (for the full set of optimality conditions see the Online-Appendix) give insight into the economic trade-offs between the different control vari-

[15] Note that from a population perspective $\int_0^t Z_2(s)ds$ corresponds to the prevalence of the disease in age-group t.

ables. Using the valuations of health presented in the previous section, we can formulate these FOCs in a compact and intuitive way.

Proposition 2. *Assume the existence of optimal trajectories of consumption and (the various) health investments in both stages of the individual life-cycle model (13)-(20) together with interior solutions for the (various) choices of health care. The first-order optimality conditions can then be written as follows.*

$$\textit{Stage 1:} \qquad \left[-\mu^1_{b_1}(t)\right] \cdot \psi^1_H(t) = p^b(t) \tag{34}$$

$$[-\eta_{h_1}(t)] \cdot \psi_P(t) = p^1(t) \tag{35}$$

$$\textit{Stage 2:} \qquad \left[-\mu^2_{b_2}(t,s)\right] \cdot \psi^2_H(t,s) = p^b(t) \tag{36}$$

$$[-f_{h_2}(t,s)] \cdot \psi_M(t,s) = p^2(t) \tag{37}$$

$$\textit{At the time of shock } s: \qquad [-B_d(s)] \cdot \psi_M(s,s) + P_d(s) \cdot \psi_{AS}(s) = p^d(s) \tag{38}$$

Proof. A simple rearrangement of the FOCs presented in the Online-Appendix and usage of the formulas shown in the proof of Proposition 1 directly imply the presented results. □

Consequently, the optimal allocation of health care involves that for each age/point in time t and for every possible onset of the shock s, the unit price for each type of health care equals the corresponding marginal benefit, consisting of the respective marginal effectiveness and the respective valuation of the health dimension involved. Thus, the price $p^b(t)$ for general first-stage health care, $b_1(t)$, has to equal its marginal impact on mortality ($-\mu^1_{b_1}$) (tantamount to the depreciation rate of health) multiplied with the first-stage value of health, ψ^1_H. The interpretation for the other types of health care is analogous. Furthermore, we note that the marginal benefits of acute care, d, consist of the sum of two separate terms, as acute care does not only (potentially) increase the chances of surviving the shock but also (potentially) reduces the initial level of morbidity following the shock.

The FOCs provide immediate and intuitive information on the (relative) drivers of health care choices. Thus, the individual will demand a higher quantity of health care if it is more effective, if it has a higher value or if it has a lower price. Notably, the FOCs can also be read as reflecting the optimal trade-off between the different types of health care and consumption. Here, the left-hand side (LHS) of each condition reflects the marginal rate of substitution between the particular type of health care and consumption, whereas the right-hand side (RHS) gives the price ratio, with the price of the consumption good normalized to one. Thus, a higher price for health care would have to be offset by an increase in effectiveness and/or an increase in the value of this care, the latter reflecting greater need. The system of FOCs also allows to trace the allocation across different types of health care in light of relative effectiveness and relative valuations. Consider e.g. the condition

$$\frac{-B_d(s)}{-f_{h_2}(s,s)} + \frac{P_d(s) \cdot \psi_{AS}(s)}{[-f_{h_2}(s,s)] \cdot \psi_M(s,s)} = \frac{p^d(s)}{p^2(s)}$$

as implied by the FOCs (37) and (38). To understand the intuition, assume first a setting

in which the health shock does not impose a risk to survival, implying there is no role for acute care in enhancing acute survival, i.e. $P = 1$ and $P_d(s) = 0$, as would be the case e.g. with cancer. In such a case the condition would tell us that a higher price for acute care (e.g. immediate surgery) as opposed to chronic care (e.g. pharmaceutical therapy) at the point of the onset of the disease would need to be offset by greater effectiveness of acute care in containing the disease (i.e. the progression of cancer). If acute care also bears on survival, the price ratio does not only reflect differences in the effectiveness of care in curbing morbidity but, in addition, the marginal rate of substitution between the (valued) change in acute survival for the acute treatment and the change in morbidity for the chronic treatment. Thus, a higher price for acute care at the point of the shock is supported to the extent that it not only reduces initial morbidity but also improves survival chances by $P_d(s) > 0$. Note that this argument extends to settings, where the provision of acute care, e.g. cancer surgery, may carry a risk to survival, such that $P_d(s) < 0$. Here, the survival risk lowers the willingness to pay for acute care, implying that its utilization is lower relative to chronic care for a given price ratio and/or a given level of utilization is supported only at a lower price.[16] Similar trade-offs between other dimensions of health care, e.g. between general health care and preventive health care in stage 1 or between preventive health care in stage 1 and chronic health care in stage 2, can be constructed by appropriate combination of the relevant FOCs.

Isolating the value terms on the LHS of the FOCs, as e.g. in $\psi_P(t) = \frac{p^1(t)}{[-\eta_{h_1}(t)]}$ for preventive care or $\psi_M(t,s) = \frac{p^2(t)}{[-f_{h_2}(t,s)]}$ for chronic care allows us to interpret the FOCs in terms of the underlying dimensions of health as a final good rather than health care as an intermediate good. Thus, we find that for an optimal allocation, the value of prevention should equal the effective price of prevention, as given by the price of preventive health care adjusted for its effectiveness in curbing the arrival rate of a shock. Similarly, the value of (reducing) morbidity should be equal to the effective price of lowering morbidity, as given by the price of chronic health care adjusted for its effectiveness in curbing or reversing the progression of the disease. Analogous expressions can be derived for other dimensions of health care. Following Frankovic et al. (2020b) who undertake this analysis in the context of survival, we can infer that medical progress that raises the effectiveness of a certain type of health care, e.g. prevention, may be associated with a decline in the value of this dimension of health. Notably, this reflects the greater consumption of more effective preventive health care leading to reductions in the health risk to a level for which any further reduction is less valuable (or in micro-economic terms, the decline in the effective price of prevention relative to consumption is associated with a decline in the marginal rate of substitution between prevention and consumption). We conclude with the following empirical observation: In many practical settings, the valuation of different types of health is difficult to observe. A revealed preference argument would then suggest that the willingness to pay equals the effectiveness-adjusted price of health care, which can be calculated on the basis of observable nominal prices of health care and scientific evidence on medical effectiveness.[17]

[16] Recall here our assumption that all controls exhibit diminishing returns.

[17] See e.g. Cutler et al. (1998); Lakdawalla et al. (2015); Hult et al. (2018) for the construction and application

Given the multi-dimensionality of health, we can extend the previous argument to examine the relative valuation of different types of health care. Drawing on the FOCS (36) and (37), for instance, we can write

$$\frac{\psi_M(t,s)}{\psi_H^2(t,s)} = \frac{p^2(t)/\left[-\mu_{b_2}^2(t,s)\right]}{p^b(t)/\left[-f_{h_2}(t,s)\right]}.$$

Thus, the value of lower morbidity relative to the value of survival can be understood to reflect the relationship between the effective price of morbidity relative to the effective price of survival. This has important repercussions from a practical point of view, where many studies in medical evaluation seek to probe into consumer/patient assessment of improvements to the health-related quality of life relative to survival (see e.g. Rowen and Brazier, 2011:for a survey). While such studies are often carried out in the context of an abstract decision-framework, we note that when resulting from market assessments (e.g. when asking expert physicians to state these trade-offs), differences (across individuals, populations or over time) in the relative valuations of health may be as reflective of differences in the underlying preferences as of differences in the relative effectiveness of the different types of health care. Again, revealed preference analysis may be brought to bear to deduce relative valuations.

4 Numerical analysis: an application to cancer

Within the next two sections, we illustrate insights from our model by conducting a calibration exercise and calculating optimal individual behaviour numerically. To provide a concrete context, we study the impact of a (potential) diagnosis of some preventable cancer on the life-cycle allocation of health care and consumption.

To avoid adding the substantial complexity involved with the hidden progression of cancer up to the point of diagnosis, we assume that (i) a diagnosis at age/time s coincides with the onset of cancer. Thus, we disregard the building-up of the cancer-stock prior to s, which is reasonable when assuming that a diagnosis coincides with the point of first symptoms. As there is no clear empirical correlation between the stage of cancer at the time of diagnosis and the general health status (or the respective age)[18], we assume (ii) that the individual enters with a constant positive disease stock $M(s,s) = \phi_0 > 0$ independent of its stock of health, $S_1(s)$, at the point of diagnosis. Although it is also reasonable to assume that (iii) a diagnosis at s is not associated with an acute risk to survival, such that $P(S_1, d) \equiv 1$, we also assume for the purpose of this analysis that (iv) there is no role of acute care at the time of diagnosis, i.e. $d(s) \equiv 0$. This is justified when assuming that the diagnosis is early enough to rule out a significant role for surgery. To avoid the complexity involved with the timing of diagnosis, we abstract from (v) screening measures. Finally, in order to limit the number of states and

of quality-adjusted (effective) prices.

[18] Goodwin et al. (1986) find a positive correlation between the stage of cancer and the age at diagnosis for some types of cancer, while for other types this correlation turns out to be negative. Even for breast cancer alone the results turn out to be unclear. While Satariano et al. (1986) and Mandelblatt et al. (1991) find a positive relationship between age and severity, Yancik et al. (2001) find it to be non-significant.

controls we assume (vi) that healthy and unhealthy behaviours such as smoking, drinking, eating habits or exercising that affect the risk of cancer are captured by "'generic"' investments in health, b_1. Note that these feed into the hazard rate $\eta(t, S_1(t), h_1(t))$ through their impact on the health stock/survival and thereby generate health benefits beyond cancer, a reasonable assumption. We, thus, assume (vii) that there is no further role for specific cancer prevention, such that $h_1(t) \equiv 0$. We also assume that age does not have a direct bearing on the hazard rate, implying that we work with $\eta(S_1(t))$.

Altogether, we obtain a slightly reduced model, which nevertheless enables us to analyze comprehensively the impacts of cancer on the utilization of health care before and after the diagnosis, including a detailed characterization of the utilization pattern of chronic care, which from now on we refer to as cancer care.

4.1 Data sources

We calibrate our model to the data of an individual in the United States in the 2010s, using a number of data sources for the input parameters.[19] We assume that an individual receives earnings from age 20, which we take to be the age at which the individual begins to make life-cycle decisions. For the calibration of the model parameters, we also assume that the individual acts perfectly informed and rational, i.e. $\pi = 1$. We will discuss in Section 5.5 how the outcomes vary for individuals with imperfect information and bounded rationality.

4.1.1 Input parameters

One of the few fully exogenous inputs is the base age-profile of earnings $\{w_1(t) \,|\, t \in [20, 110]\}$, which in our model we assume to be the average profile for the US in 2011, as taken from the National Transfer Accounts (NTA) database[20]. We account for the fact, that there are no public pensions and all health care expenditures are paid out of pocket in our model, by adding the net health and pension transfer profiles (which are also contained in the NTA database) to the base earnings to obtain a profile of disposable income.[21] Figure 2 shows this data along the life-cycle as well as the equilibrium value for the accidental bequests.

For the income profile after a diagnosis, we abstract from the impact of cancer and assume that earnings remain unchanged relative to the first stage. The reason for this decision is twofold: (i) Income profiles that are contingent on both age and duration of cancer are not readily obtainable and therefore ignored for the purpose of this illustrative analysis. (ii) Ignoring cancer-specific earnings impacts allows us to identify the impact of a cancer diagnosis without concerns about differences the life-time budget.

[19] Due to limited data availability, we are unable to use all data from the exact same year.

[20] `www.ntaccounts.org`, see Lee and Mason (2011) and United Nations (2013) for details.

[21] The US exhibit a significant life-cycle deficit in the period cross-population data, implying that the profile for disposable income would not be able to cover the expenditure profiles for consumption and health care in a longitudinal setting. Thus, the raw data used for calibration contradicts our assumption of zero assets at the end of life. Hence we adjusted the income profile by 16.1%, such that the empirical profiles fulfill the life-time budget constraint.

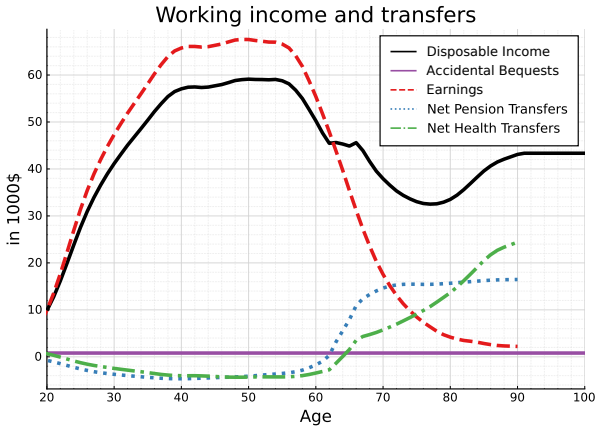

Figure 2: Earnings, transfers and disposable income

Hansen and İmrohoroğlu (2008) have shown that the typical hump-shape pattern of (non-health) consumption over the life-cycle can only be explained consistently within a life-cycle model when assuming that annuity markets are absent (or severely incomplete). For this reason, we follow Heijdra and Mierau (2012) and assume that individuals draw on a combination of annuitized assets and accidental bequests as shown in more detail in Section 4.1.2. To achieve a hump-shaped consumption profile, we have to assume that the interest rate $r(t)$ surpasses the discount rate ρ to some degree. Therefore, we propose a constant interest of $r(t) = 3\%$ and a time discount rate of $\rho = 2.5\%$.

4.1.2 Annuity market and accidental bequests

To explain the mixed annuity/accidental bequest set-up we used for the numerical solution we first describe each individual component and subsequently their combination.

Annuity rate In most models annuity markets are assumed to be either fully absent or complete with the equilibrium annuity rate being equal to the mortality rate. Our setting is more complex, as it contains two mortality rates, reflecting different health regimes: one with cancer and one without. Thus the rate of return for annuities $\theta\bar{\mu}(t)$ depends on the way the annuity market is structured.

For the present calibration we assume that the insurer has no information about whether or when an individual has been diagnosed with cancer. As a result the annuity rate (in a perfect annuity market) at time t equals the expected mortality rate (averaged across the population), and can be calculated as

$$\begin{aligned}\bar{\mu}(t) &= \frac{Z_1(t)(-\dot{S}_1(t)) + \eta(t)\cdot[1-P(t)]\,S_1(t)Z_1(t) + \int_0^t Z_2(s)(-\dot{S}_2(t,s))ds}{Z_1(t)S_1(t) + \int_0^t Z_2(s)S_2(t,s)ds} \\ &= \frac{Z_1S_1\left(\mu^1 + \eta\cdot[1-P]\right) + \int_0^t Z_2S_2\mu^2 ds}{Z_1S_1 + \int_0^t Z_2S_2 ds}. \end{aligned} \tag{39}$$

The numerator adds up all deaths across the two groups with and without cancer and relates them to the total population in the denominator. Note that we included for sake of generality

the deaths at the point of the shock/diagnosis. This third type of mortality is not relevant for our example of a cancer diagnosis, but becomes important in the analysis of e.g. cardiovascular diseases. One of multiple alternative assumptions would be that the insurer has perfect information about a cancer diagnosis, allowing a reclassification of individuals with a diagnosis. In this specific case (with $P = 1$) the annuity rate would be equal to the mortality rate for each type of individual, i.e. $\bar{\mu}^1(t) = \mu^1(t)$ for those without diagnosis and $\bar{\mu}^2(t) = \mu^2(t)$ for those with. Individuals would then need to factor in the reclassification on their asset income as part of the health shock. Investigating the implications of such a differently structured annuity market is an interesting task in its own right that goes beyond the scope of the present paper.[22]

Accidental bequests Accidental bequests describe the redistribution of (positive and negative) assets left behind by individuals after their death. The aggregated stranded assets in absence of an annuity market can be calculated as

$$\begin{aligned}\text{Stranded Assets} = \int_0^T \Big[\mu^1(t)S_1(t)Z_1(t)A_1(t) + \eta(t)\cdot[1-P(t)]\,S_1(t)Z_1(t)A_1(t)\Big]dt + \\ + \int_0^T \int_0^t \mu^2(t,s)S_2(t,s)Z_2(s)A_2(t,s)dsdt. \end{aligned} \tag{40}$$

Furthermore, we assume that at each point in time the stranded assets, which have not been annuitized, are distributed in equal proportion across the population.

$$B(t,\theta) = \frac{(1-\theta)\cdot\text{Stranded assets}}{\text{Total population}} = \frac{(1-\theta)\cdot\text{Stranded assets}}{\int_0^T \Big[S_1(t)Z_1(t) + \int_0^t Z_2(s)S_2(t,s)ds\Big]dt}. \tag{41}$$

Mixed setting Following the results of Heijdra and Mierau (2012) we assume that the annuity rate reaches 70% of the actuarially fair values and the remaining assets are redistributed through accidental bequests, i.e. we set $\theta = 0.7$.

4.1.3 Calibration targets

We use health expenditure data from the NTA-Database (available for the year 2011) and combine it with age-specific information about the share of total cancer specific health care expenditures from `healthdata.org`[23] (out-of-pocket and co-payments) to construct age profiles for (i) the general (non-cancer) health expenditure and (ii) cancer specific expenditures against which we the respective average profiles generated by our model.[24] Due to limited data availability on age- and duration specific cancer care expenditure and their dependence on the specific type of cancer, we focus on replicating qualitative aspects of the cancer care expenditure profiles, which are numerically within the plausible range of empirical expenditures.

[22] Note that one might in this context interpret the cancer risk as one source of survival ambiguity as modeled by Caliendo et al. (2020), with our choice for the annuity market exhibiting asymmetric information between the individuals and insurance companies.

[23] Institute for Health Metrics and Evaluation (IHME) (2016) (Accessed 2020-01-13)

[24] We define the average health expenditure at age t as $\overline{b}(t) := Z_1(t)b_1(t) + \int_0^t Z_2(s)b_2(t,s)ds$ and, similarly, the average cancer care expenditure as $\overline{h_2}(t) := \int_0^t Z_2(s)h_2(t,s)ds$.

Furthermore, we draw on the age-specific mortality profile for the US in 2011 from the human mortality database[25] as a basis for establishing survival profiles. Using the mortality rate directly to calculate the corresponding survival profile within our model, we obtain the equivalent of the average survival $\overline{S}(t) := Z_1(t)S_1(t) + \int_0^t Z_2(s)S_2(t,s)ds$ in our model. To obtain the appropriate data against which to compare first-stage survival in our model, we take age-specific cancer mortality rates from the SEER-database[26] and subtract them from the corresponding general mortality rates. From the resulting age-profile of non-cancer mortality we construct a cancer-free survival profile as the appropriate comparison for the S_1 profile in the model.

We then employ the cancer-free survival profile from the data together with age-specific cancer incidence rates,[27] again taken from the SEER-database, to calibrate the hazard rate $\eta(S_1)$, which we assume to depend only on the survival state. Finally, we use information from the SEER-database about cancer-specific survival depending on the duration since the cancer diagnosis to calibrate the cancer-specific mortality rates for four different age-groups over the first ten years after the diagnosis. For further details on the estimation and calibration strategy for general and cancer-specific mortality rates (μ^b and μ^m) and the cancer hazard rate (η) we refer to the Appendix A.

4.2 Solution strategy

The numerical solution of a two-stage optimal control problem with random switching time is a far-from-trivial problem. As indicated in Section 2, the transformation into a vintage-structured optimal control problem (see Wrzaczek et al. (2020) for further details) allows our numerical strategy to rest on an existing gradient-based optimization algorithm, as described by Veliov (2003). For an extended discussion of the algorithm and the adaptations necessary for capturing particular features of our model, we refer to the Online-Appendix.

4.3 Functional specifications

To generate a numerical solution of our model, as summarized in equations (13) - (20), we need to specify the following functional forms. Details on the calibration strategy can be found in the Online-Appendix.

Utility: Following Hall and Jones (2007) and many others, we employ an adjusted CRRA-utility function for instantaneous utility from consumption:

$$u^1(c) = \frac{c^{1-\sigma}}{1-\sigma} + \overline{u}, \qquad 0 < \sigma \neq 1 \qquad .$$

Here, we assume the constant $\overline{u}$ to be sufficiently large, guaranteeing $u^1(c) > 0$ for all reasonable

[25] University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany) (2020) (accessed 2019-10-04)

[26] Surveillance Research Program, National Cancer Institute (2020)

[27] We take average incidence rates between 2012 and 2016.

consumption levels.[28] We set $\sigma = 1.13$ as has been chosen in Frankovic et al. (2016) and which lies in the range of empirically estimated values found in Chetty (2006).

For the second stage, we assume that the cancer stock enters utility in a multiplicative form:

$$u^2(c, M) = u^1(c)v(M) = u^1(c) \exp\{\kappa_0 \cdot M^{\kappa_1}\},$$

with $v(M) \in [0, 1]$ and $v'(M) < 0$ for $\kappa_0 < 0$. The mixed derivative $u^2_{cM} = u^1_c(c)v'(M)$ is negative, which implies that a higher cancer stock reduces the marginal utility of (non-health care) consumption, as is consistent with empirical evidence (see Finkelstein et al., 2013).[29]

Mortality: To enable us to disentangle cancer specific mortality in the second stage, we assume here that the non-cancer mortality rate $\mu^b(t, S_1, b_1)$ does not depend on the state of survival S_1.

$$\mu^b(t, b_1) = g(t)b_1^{\varepsilon(t)} = \exp\left\{\gamma_0 + \gamma_1 t + \gamma_2 t^2 + \gamma_3 t^3\right\} \cdot b_1^{\alpha_0 + \alpha_1 t}$$

Here, the parameters γ_i and α_i are calibrated to reproduce the survival profile without a cancer diagnosis and the empirical profile of general health expenditures b_1.

Cancer hazard: Comparing survival data adjusted for cancer mortality and cancer incidence rates from the SEER-database, we find that the function

$$\widetilde{\eta}(t, S_1) = \widetilde{\eta}(S_1) = \beta_0 \cdot \left(1 + \beta_1 \left(\frac{1 - S_1}{S_1}\right)^{\beta_2}\right)^{-1}$$

delivers the best fit for the objective hazard rate. Furthermore, we calibrate the model for a perfectly informed and rational individual, i.e. $\pi = 1$.

Cancer stock: For the progression of the cancer stock,

$$f(t, s, M, h_2) = \delta_0 M - \delta_1 h_2^{\delta_2} M^{\delta_3}, \tag{42}$$

we draw on Talkington and Durrett (2015) who present multiple established processes for the untreated spread of cancerous cells. We use the simplest version (which is sufficient for our purposes) and therefore assume that the number of cancer cells grows over time at a constant rate δ_0. Following Heuser et al. (1979), who estimated the average doubling time for breast cancer cells to be 327 days, we propose a rate of $\delta_0 = 0.774$ (per year) and normalize the initial number of cancerous cells when diagnosed to one, i.e. $M^0(S, d) = \phi_0 = 1.0$ (see footnote 18).

For the effects of cancer care, we lean on the health deficits approach by Dalgaard and Strulik (2014). Depending on the intensity of cancer care h_2 (subject to diminishing returns with $\delta_2 \in (0, 1)$) and the available technology (captured by δ_1), we propose that growth can be

[28] Specifically, we assume $\overline{u} = 11.370$ (see Table 3). For the lower consumption bound of \$50/year that we impose, this generates a positive utility of 0.0148 in our numerical calibration.

[29] Due to the limited availability of data on the direct impact of cancer deficits on individual utility, the numerical values are chosen to improve overall calibration. We conducted a sensitivity analysis by solving the model without any impact of cancer deficits on utility and found no significant impact on the qualitative structure of the results; a summary of these results is available upon request.

slowed or reversed. In contrast to Dalgaard and Strulik (2014) we propose that the effectiveness of cancer care increases with the number of cancerous cells. Including M linearly is not able to replicate the empirical data. However, assuming a convex impact of M is sufficient in this regard, and we therefore multiply by M^{δ_3} with $\delta_3 > 1$.

Cancer specific mortality: For the cancer specific mortality function $\mu^m(t, s, M)$, we eliminate the dependence on age t and assume that the whole mortality process is driven by the number of cancerous cells M and the age at which cancer was diagnosed. As we find in the data, mortality increases with age at diagnosis. Hence, we propose the following functional form:

$$\mu^m(s, M) = \psi_0 \cdot M \cdot \exp\left\{\psi_1 \cdot \left(\frac{s}{T}\right)^{\psi_2}\right\} \tag{43}$$

We estimate the parameters ψ_i using data on cancer incidence and survival data, as described in Appendix A.

Prices for health care: We set the prices for general and chronic health care goods and services equal to 1, such that b_1, b_2, h_2 can be seen as the relevant monetary expenditures.

Table 3 summarizes the functional forms and parameter choices. We wish to stress that we did not attempt to obtain a full calibration of the model, since this would have required the introduction of further state and control variables to represent the complex nature of cancer risk, prevention, progress and treatment. Instead, we aimed for a reduced model formulation, which nevertheless replicates key patterns of cancer progression and cancer care, and thereby enables new insights into the behavioural patterns regarding health care and consumption in the presence of cancer as a health shock.

$u^1(c) = \frac{c^{1-\sigma}}{1-\sigma} + \overline{u}$	σ	1.13^{**}	$M^0(S,d) = \phi_0$	ϕ_0	1.0
	$\overline{u}$	11.370^{**}	$f(t,s,M,h_2) = \delta_0 M - \delta_1 h_2^{\delta_2} M^{\delta_3}$	δ_0	0.7737^{**}
$\varepsilon(t) = \alpha_0 + \alpha_1 \cdot t$	α_0	-0.2473^{+}		δ_1	2.0^{++}
	α_1	0.0672^{+}		δ_2	0.03^{++}
$g(t) = e^{\gamma_0+\gamma_1 t+\gamma_2 t^2+\gamma_3 t^3}$	γ_0	-7.004^{+}		δ_3	1.3^{++}
	γ_1	-0.100^{+}	$\mu^m(t,s,M) = \psi_0 \cdot M \cdot$	ψ_0	0.1705^{*}
$\left(\mu^b(t,b) = g(t) \cdot b^{\varepsilon(t)}\right)$	γ_2	15.571^{+}		ψ_1	1.8474^{*}
	γ_3	-7.984^{+}	$\cdot \exp\left\{\psi_1 \cdot \left(\frac{s}{T}\right)^{\psi_2}\right\}$	ψ_2	1.5572^{*}
$\widetilde{\eta}(t,S) = \frac{\beta_0}{1+\beta_1(\frac{1-S_1}{S_1})^{\beta_2}}$	β_0	0.0243^{*}	$v(M) = \exp\{\kappa_0 \cdot M^{\kappa_1}\}$	κ_0	$\ln(0.7)^{**}$
	β_1	0.0087^{*}		κ_1	1.2^{**}
	β_2	-2.020^{*}	Interest rate	r	0.03^{**}
Annuity market completeness	θ	0.7	Discount rate	ρ	0.025^{**}

Table 3: Summary of functional specifications and parameters in the model

Parameters indicated by * are estimated using empirical data. Parameters marked with ** are taken from (or are in line with) the empirical literature. Parameters indicated with $^{+}$ result from automated calibration within the solution process so that the model profiles (survival, expenditures) match the empirical data. The parameters indicated with $^{++}$ are calibrated to match the empirical data on the duration dependent cancer-specific mortality rates. For further details on the calibration process we refer to Appendix A.

5 Numerical results

We will first show to what extent we were able to replicate the data and thereby meet our calibration targets (Section 5.1). In a next step, we discuss and compare the profiles of consumption before and after a cancer diagnosis and use the respective Euler-equations to identify how these profiles are impacted by the different aspects of the (potential) diagnosis. Section 5.3 presents the profiles of general health investments and cancer care together with the resulting developments of the health state in both stages and the disease stock that captures the progression of cancer. In doing so, we also examine the relevant valuations of health introduced in Section 3.1 and their respective decomposition. Finally, Section 5.4 presents Euler-type equations for the different types of health care both in the most general formulation, as well as for their numerical evaluations for the optimal solution. These calculations allow us to pin down the several impacts a potential diagnosis has on the individual's decision making.

5.1 Expenditure and survival patterns: Model vs. data

Figures 3 and 4 compare, for a range of variables, model outcomes with data from the US, as discussed in previous sections. More specifically, Figure 3 summarizes the different types of expenditure in our model. The left panel shows the expected values for general (non-cancer) health expenditures as well as for the spending on cancer care. Overall, the expected expenditure profiles follow the data reasonably well. The right panel illustrates that we are able to match the qualitative hump-shape of the consumption profile well. This is feasible through our choice of an incomplete annuity market.

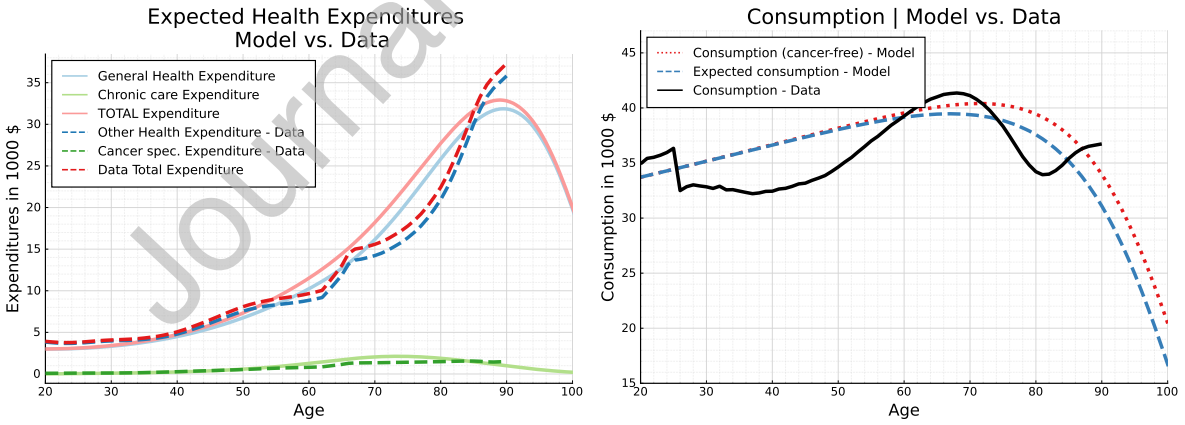

Figure 3: Calibration results: Comparison between expenditures predicted by the model and data

In Figure 4 we focus on the fit of the survival and mortality data. The left panel shows that we match remarkably well both average survival, $\overline{S}(t) = Z_1(t)S_1(t) + \int_0^t Z_2(s)S_2(t,s)ds$ and survival conditional on remaining cancer-free, $S_1(t)$. Furthermore, we see that the cancer-free survival profile, $Z_1(t)$, in our model matches the corresponding profile derived from the data relatively well, if slightly underestimating it. The right panel shows cancer-specific mortality data over the first 10 years after a diagnosis for 4 different age-groups. Note that we plot

the logarithm of the mortality rate to account for the strong differences in magnitude of these values. The model and data profiles match very well, despite some discrepancy for the youngest and oldest age groups around the point of diagnosis.

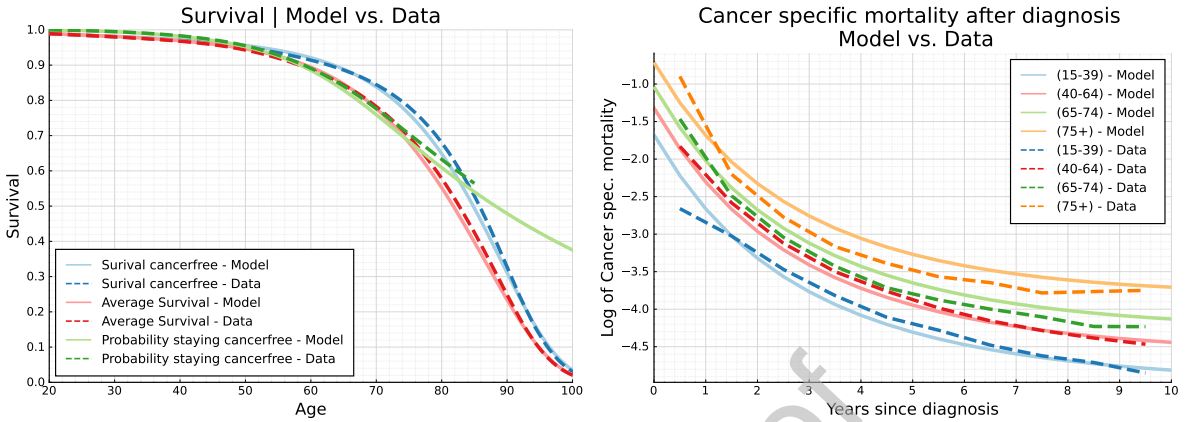

Figure 4: Calibration results: Comparison of survival and mortality profiles between model and data

5.2 Consumption profiles

Consider now the optimal consumption profiles, as plotted in Figure 5. The grey dashed line depicts the consumption profile in the absence of cancer. The solid lines represent the consumption profile following a cancer diagnosis at a given age, where for illustrative purposes we move the age at diagnosis in five year steps. Finally, the black dotted line illustrates the consumption level chosen immediately after a diagnosis, the distance between the dotted and dashed lines representing the instantaneous consumption change following a cancer diagnosis. The consumption profile in the absence of cancer follows a hump-shaped pattern and increases

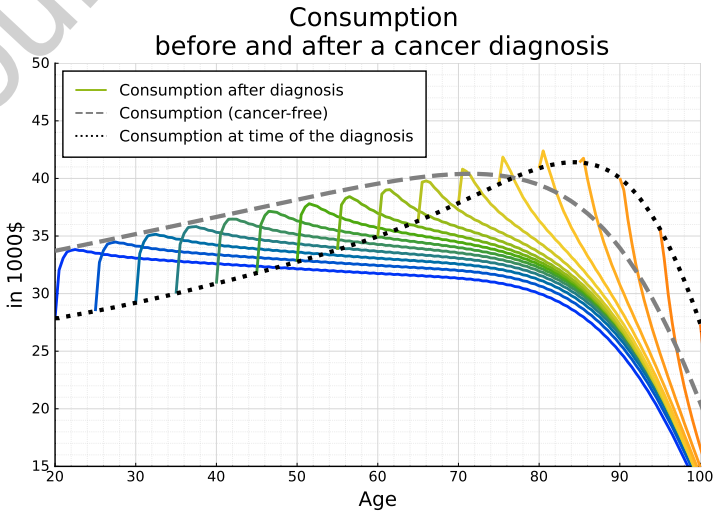

Figure 5: Consumption profiles in the first and in the second stage

from about $35,000 to slightly above $40,000 at the age of 70. Afterwards the consumption

decline accelerates and drops to below 50% of peak consumption at age 100. The hump shape is a result of the interest rate surpassing the individual's discount rate (explaining the increase early in life) and the incomplete annuity market, i.e. the difference between annuity returns based on averaged mortality and the individual's state-contingent mortality (explaining the decrease in later stages of life).

After a cancer diagnosis optimal consumption follows a qualitatively different path. Right after the diagnosis individuals decrease their consumption if the diagnosis occurs during the early or middle stages of the life-cycle, i.e. before around age 85. This immediate drop by some 17 percent is followed by an increase over the next two years which reduces the gap to consumption levels of cancer-free individuals of the same age to insignificant levels. Subsequently, the consumption of diagnosed individuals decreases independent of the age of diagnosis, while for later diagnoses in life this decline becomes more pronounced. In every case the gap in consumption between diagnosed and cancer-free individuals widens.

If the diagnosis occurs in late stages of life after the age of 85, individuals boost their consumption directly after the cancer diagnosis after which the consumption path follows a permanent decline over the remaining life-course. The upward jump is increasing with the age at diagnosis and reaches levels of up to 128 percent of the cancer-free consumption level. Following this initial boost, the consumption of individuals with late diagnoses then declines rapidly to levels of up to 30 percent below the consumption of cancer-free individuals at age 100.

To explain these striking patterns in the second stage we can disentangle multiple factors using the Euler-type equations (44) and (45) for the dynamics of consumption before and after the diagnosis,[30,31]

$$\frac{\dot{c_1}}{c_1} = \frac{u^1_{c_1}}{-u^1_{c_1c_1}c_1}\left[r-\rho+\theta\bar{\mu}-\mu^1-\eta+\eta P(S_1,d)\frac{u^2_{c_2}}{u^1_{c_1}}\right], \tag{44}$$

$$\frac{\dot{c_2}}{c_2} = \frac{u^2_{c_2}}{-u^2_{c_2c_2}c_2}\left[r-\rho+\theta\bar{\mu}-\mu^2+\frac{u^2_{c_2M}}{u^2_{c_2}}f\right]. \tag{45}$$

We can identify two shared and two distinct contributing factors between the drivers of consumption before and after the diagnosis. In both stages, a positive gap between the interest and discount rate, $r-\rho$, tends to induce a deferral of consumption, while the mortality risk, μ^i, shifts consumption towards younger ages to the extent that it is not offset by annuity returns, $\theta\bar{\mu}$. In addition, we find the hazard rate and the change in marginal utility from consumption after the diagnosis shaping the profile in the first stage, while in the second stage the disutility from the disease stock comes into play. In Figure 6 we present a numerical attribution of the (log) difference between current consumption at time/age t and initial consumption at $t_0 = 20$

[30] Note that all Euler equations (here and in the following sections) are presented in their general form, and the interpretations also apply to a general health shock. Here, we discuss them in the context of the numerical results based on our calibration for cancer and discuss which terms are non-existent in our specified set-up.

[31] For the derivations we use the first-order optimality conditions for consumption together with the dynamics of the adjoint variables of assets. The relevant equations as well as the derivations are presented in the Online-Appendix.

to the different components of the Euler equation. The left panel shows the decomposition before a cancer diagnosis, the right panel after a cancer diagnosis at age 30. In this representation, the different terms $F_i(t)$ in the Euler equations are aggregated from the beginning of the life-cycle (resp. from the time of diagnosis) up to each point in time t. Taking into account, for instance, stage-1 consumption $c_1(t)$ and defining $\frac{\dot{c_1}(t)}{c_1(t)} =: \sum F_i(t)$ we can write $c_1(t) = c_1(t_0)\exp\{\int_{t_0}^{t}\sum F_i(s)ds\}$ and, thus, $\log(c_1(t)) - \log(c_1(t_0)) = \sum\int_{t_0}^{t} F_i(s)ds$. Hence, the aggregated terms are direct expressions of the (log) difference between current consumption $c_1(t)$ and initial consumption $c_1(t_0)$.[32] In both panels we see that the positive difference

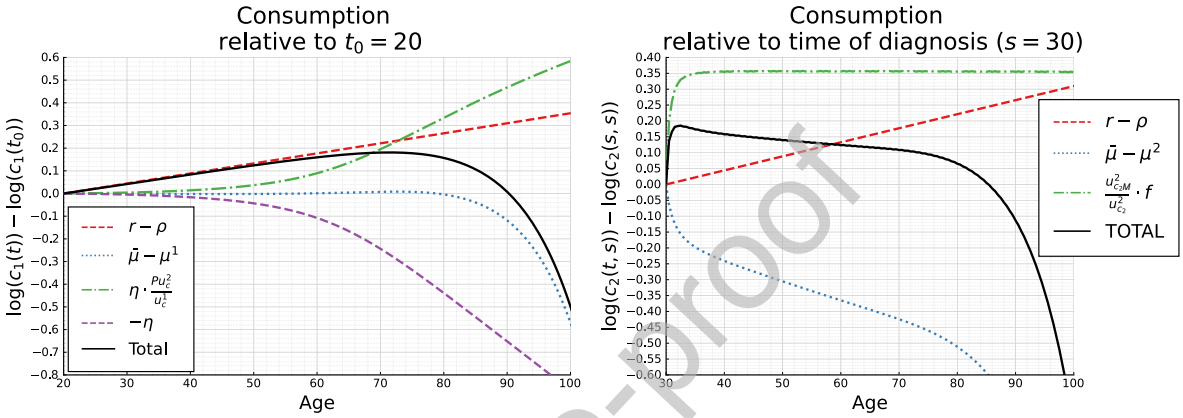

Figure 6: Decomposition of (log) difference between current consumption and initial consumption before (left panel) and after (right panel) a diagnosis.

between interest and discount rate leads to a deferral of consumption (red dashed line). Considering now cancer-free individuals (left panel), we find that although the annuity market is incomplete, the annuity rate nearly perfectly covers the mortality risk until age 80.[33] Afterwards, however, the mortality rate substantially surpasses the annuity rate even for cancer-free individuals and incentivizes an advancement of consumption.

The hazard of a cancer diagnosis ($-\eta$) triggers two offsetting incentives: A desire to self-insure against the cancer risk induces individuals to shift consumption to earlier ages (dashed purple line), which is pitted against an incentive to accumulate precautionary savings, $\eta P(S_1, d)\frac{u_{c_2}^2}{u_{c_1}^1}$ (dash-dotted green line). Notably, the incentive to advance consumption for reasons of self-insurance becomes significant after the ages around 60, and strongly increases towards the end of life.

Turning to the drivers of the consumption profile after a cancer diagnosis (at age 30), as depicted in the right panel, we can identify three distinctive channels that explain the pronounced difference of consumption choices compared to a cancer-free individual. (i) The strong decline of the blue dotted line in the beginning indicates a strong early-on advancement of consumption. This is because the average annuity rate only compensates to a small extent the strong initial increase in mortality in the first years after diagnosis. (ii) The subsequent

[32] We use the same decomposition technique when presenting the dynamics of health expenditure in Section 5.4.

[33] As the annuity rate is defined through the average mortality of individuals with and without cancer, it somewhat overpays cancer-free individuals such that even an incomplete annuity market covers their mortality rate.

more gradual decrease of the same line towards later in life reflects the permanent but much slighter increase in the mortality of cancer-survivors over their remaining life-cycle, as compared to cancer-free individuals. The resulting permanent incentive to advance consumption explains the widening gap between consumption levels of cancer and non-cancer individuals. (iii) The green dash-dotted line indicates a strong tendency to postpone consumption early on. This is reflecting the negative impact of the cancer stock on the marginal utility of consumption shortly after diagnosis (see Figure 8 for the development of the cancer stock) under our assumption that good health and consumption are complements. As the individual expects a relatively fast reduction in the cancer stock due to the early intensive consumption of cancer care (see Figures 7 and 8), it tends to defer consumption only for a few years.

Combining these three channels we find that for diagnoses early in life channel (iii) dominates (i) within the first two years after diagnosis, implying a sharp increase in consumption early on, whereas channel (ii) tends to become dominant as time progresses, which in turn implies its gradual decline. While the profile in the RHS panel of Figure 6 helps us to explain the profile of stage-2 consumption, we need to recall from Figure 5 that the onset of cancer itself leads to a strong discrete decline in consumption below its pre-diagnosis level. This decline relates to both the reallocation of spending to cancer care and the instantaneous drop in the marginal utility of consumption in the presence of (perceived) cancer deficits.

Finally, we note from Figure 5 that spending patterns differ for individuals at advanced ages. For individuals who are diagnosed at advanced age channel (i), i.e. the incentive to advance consumption given higher mortality directly after the shock, dominates channel (iii), i.e. to deferral of consumption to ages with higher marginal utility. Furthermore, the effect of channel (i) is too strong to make a clear distinction between effects (i) and (ii).[34]

We summarize our findings on consumption in the following two corollaries.

Corollary 1 (Consumption before diagnosis)**.** *Accounting for the risk of a cancer diagnosis implies a consumption profile with a more pronounced hump shape. This results from the incentive to defer consumption early in life in order to benefit from a (partial) annuity return that overcompensates the mortality risk of a cancer-free individual (up until age 80) and the desire to advance consumption later in life to self-insure against the loss in marginal utility from consumption for the case of contracting cancer.*

Corollary 2 (Consumption after diagnosis)**.** *The increased mortality risk after a cancer diagnosis implies that individuals strongly advance their consumption. This effect is dampened or even overcompensated by the incentive to postpone consumption to ages where a reduction in the intensity of cancer allows for a higher marginal utility of consumption.*

5.3 Health expenditure and health state profiles

We now focus on the optimal choices for health expenditure and the resulting profiles of the health/survival and cancer stock. We also calculate, for our numerical specification, the valu-

[34] The decompositions as presented in Figure 6 for other times of diagnosis are available upon request. Furthermore, an extended analysis of the Euler equations and the numerical evaluation can be found in Freiberger (2022). Additional robustness checks are available upon request.

ations of health from Definition 1 and decompose them according to Proposition 1.

5.3.1 Health expenditure profiles

As shown in the upper left panel of Figure 7, a cancer diagnosis does not imply a great difference with respect to the qualitative (hump-shaped) profile of general, i.e. non-cancer specific, health expenditures over the life-cycle. However, we obtain some significant quantitative differences.

For early diagnoses, general health expenditures initially drop by around $1,300 per year, which amounts to around 43% of the expenditures before a diagnosis for the youngest groups. Up until an age at diagnosis of 60, this initial gap even increases to up to $3,000 per year. Furthermore, for all cohorts, the initial gap tends to widen over the life-cycle and becomes specifically pronounced later in life. For individuals with an early diagnose, the gap at higher ages may lie in excess of $10,000.

The instantaneous reduction in general health care spending (difference between the dashed and dotted lines) can be traced back to the sudden and large increase in cancer care expenditure triggered by a diagnosis (see the upper right panel of Figure 7). For all age groups, expenditure is highest immediately after diagnosis and then declines rapidly over a time span of around ten years to a residual spending level for the purchase of care (e.g. regular screenings) that is necessary to avoid the reemergence of cancer. Interestingly, (initial) expenditures on cancer-care (dotted line) increase slightly with age from around $67,000 at young ages to some $76,000 for a diagnosis occuring at age 55. As the time horizon quickly shortens when cancer is diagnosed closer to the end of life, the benefits of cancer care are less pronounced, and therefore spending at the point of diagnosis declines steeply to insignificant levels at high ages. [35]

The lower panel of Figure 7 shows the financial burden associated with the high expenditures for cancer care. Right after diagnosis, assets decline steeply, where young individuals (with negative assets already (dashed line)) go further into debt to finance cancer treatment, despite concomitantly reducing general health expenditure and consumption, as in Figure 5. We need to stress, however, that the structure of the annuity market plays a crucial role. As we assume that cancer patients are not identified by the market, they obtain the same annuity rate as a representative member of the population. Despite their higher mortality risk, individuals with a cancer diagnosis can therefore go into debt to finance cancer care, facing the same full interest rate as the average population. In contrast, adjustments in the annuity price to the individual health state and associated mortality ((Reichling and Smetters, 2015)) would strongly compromise the individual's ability to fund effective cancer care. In this respect, our model illustrates the scope and need for health insurance to shield the individual against the financial repercussions of large scale health shocks.

[35] On inspection, the spending levels on cancer care might appear high, but they are in line with the average per-patient costs that are reimbursable for breast cancer. Blumen et al. (2016) estimate the allowed costs to lie between roughly $60,000 and $134,000 within the first 12 months after a diagnosis (depending on the stage of cancer) and between $13,000 and $70,000 through the second year. Unfortunately, more extensive information on cancer care expenditures depending on the age at diagnosis and the duration since diagnosis is unavailable. This notwithstanding, our model is not only able to replicate the sharp decline in cancer care expenditure within the first two years after diagnosis, but also matches the expected expenditure for cancer care at every age as shown in Figure 3.

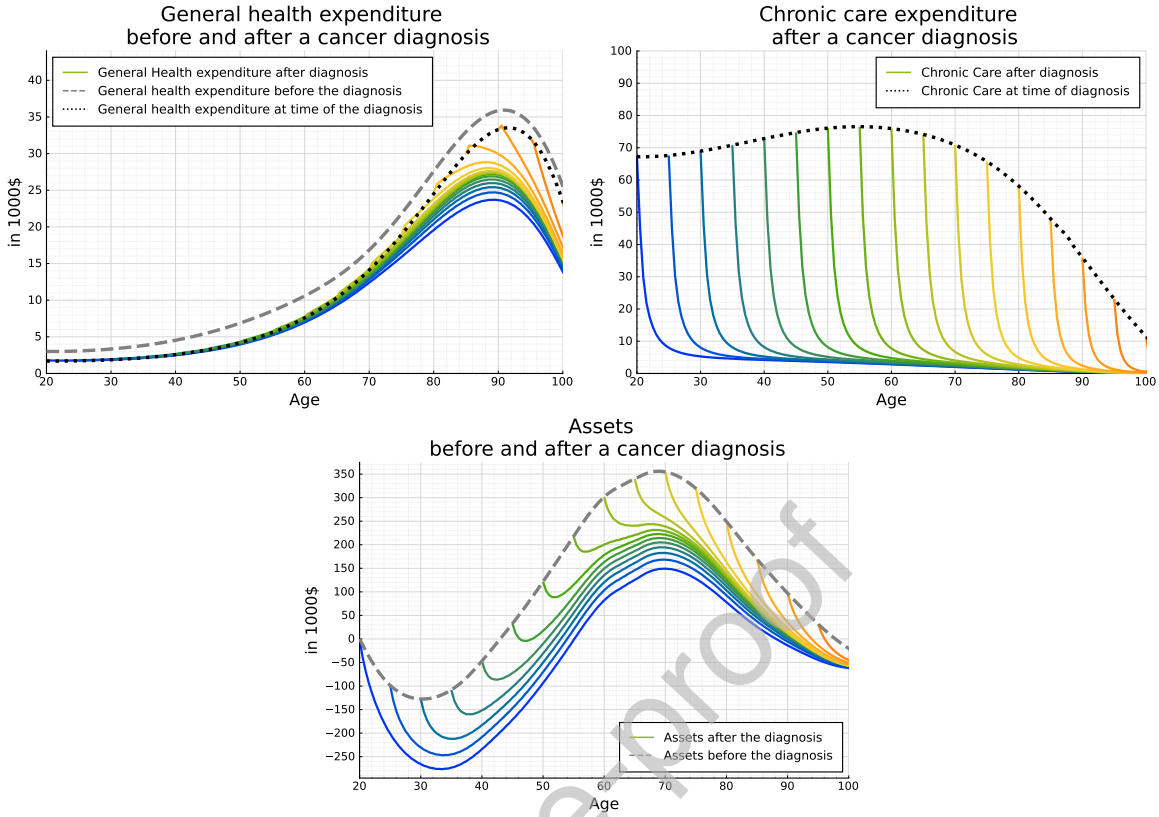

Figure 7: First row: General and cancer specific health expenditures before and after a diagnosis. Second row: Financial assets before and after a diagnosis.

5.3.2 Cancer stock dynamics

In Figure 8 we see that the (optimal) pattern of cancer care is effective in decreasing cancer stock for all ages at diagnosis. This suggests that for our specification, cancer kills patients relatively early on and is otherwise transformed into a chronic disease, where a small residual stock is not eliminated, but rather held in check by chronic care. This can be interpreted as a situation in which cancer can, indeed, be reduced to negligible levels but where the risk of its resurgence requires a small amount of care in the form of regular screenings. The residual cancer stock also induces a mortality risk (depending on the age at diagnosis), which covers the chance of dying through a potential relapse. Meanwhile, the impact of residual cancer stock on consumption utility is insignificant in the long run. For our calibration, the cancer stock that remains after ten years decreases utility by less than one percent.

5.3.3 Valuations of Health

In this section, we provide numerical illustrations of the valuations of the various components of health derived in Section 3.1. By entering the first-order conditions presented in Proposition 2, these profiles play a crucial role in the shape of the health investment profiles.

In the upper left panel of Figure 9, we compare the different valuations. The solid black line shows the value of health (VOH), ψ_H^1, in the first cancer-free stage. The VOH amounts to

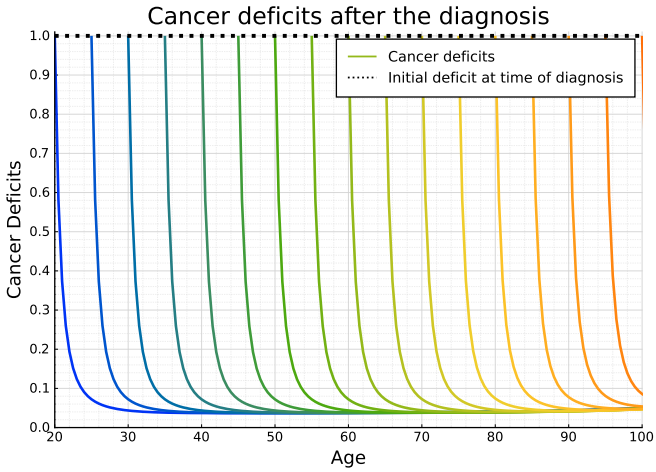

Figure 8: Cancer deficits after diagnosis.

around \$17.5 million at age 20 and follows a concave-convex decline throughout the life-cycle. The expected or average VOH, which accounts for the weighted prevalence of cancer at age t (see Definition 2), is illustrated by the dotted (blue/dark) line and differs only slightly from the first-stage VOH. The dash-dotted (green) line indicates the value of acute survival, which is equal to the initial VOH after the shock under our specific assumptions. Hence, we can see that the VOH drops dramatically after a diagnosis before age 70, this difference being less pronounced for diagnoses later in life. The dashed (red) line indicates the value of prevention, i.e. the willingness to pay for a reduction of the hazard rate. This value is significantly smaller, but still starts around \$3 million at age 20 and stands around \$1 million at the age of 85. Finally the dotted (purple/light) line represents the value of reducing morbidity (VOM) at the time of the diagnosis. This value stays fairly constant around \$1 million up to age 70 and then declines slowly towards the end of the time-horizon. The remaining panels contribute to understanding second stage valuations and how they relate to the first stage. Here, the upper right panel shows the development of the VOH in the second stage, i.e. the stage with cancer, for different ages at diagnosis as compared to the VOH in stage one. Apart from its general decline with age, there is little additional variation in the second-stage VOH with respect to age at diagnosis. However, one may wonder why the strong drop in the VOH at diagnosis for all ages up to 70 does not exacerbate the gap between the first-stage VOH, ψ_H^1, and the expected VOH. The answer lies in the low probabilities of being diagnosed with cancer early in life, as can be seen in the lower right panel of Figure 9. This implies a low weight of ψ_H^2 in the calculation of the expected VOH (see equation (33)). In contrast, the difference in the first- and second-stage VOH has become small for the age-groups older than 70 who are subject to significant prevalence of cancer. Thus, the difference between the "cancer-free" first-stage VOH and the expected VOH remains remarkably small throughout the full life-cycle. This has an interesting policy implication:

Corollary 3 (Expected Value of Health)**.** *Even health shocks that are wide-spread (such as cancer) and have large impacts on the VOH from an individual perspective exert little influence on the VOH from a population perspective (as represented by the expected or average VOH) if*

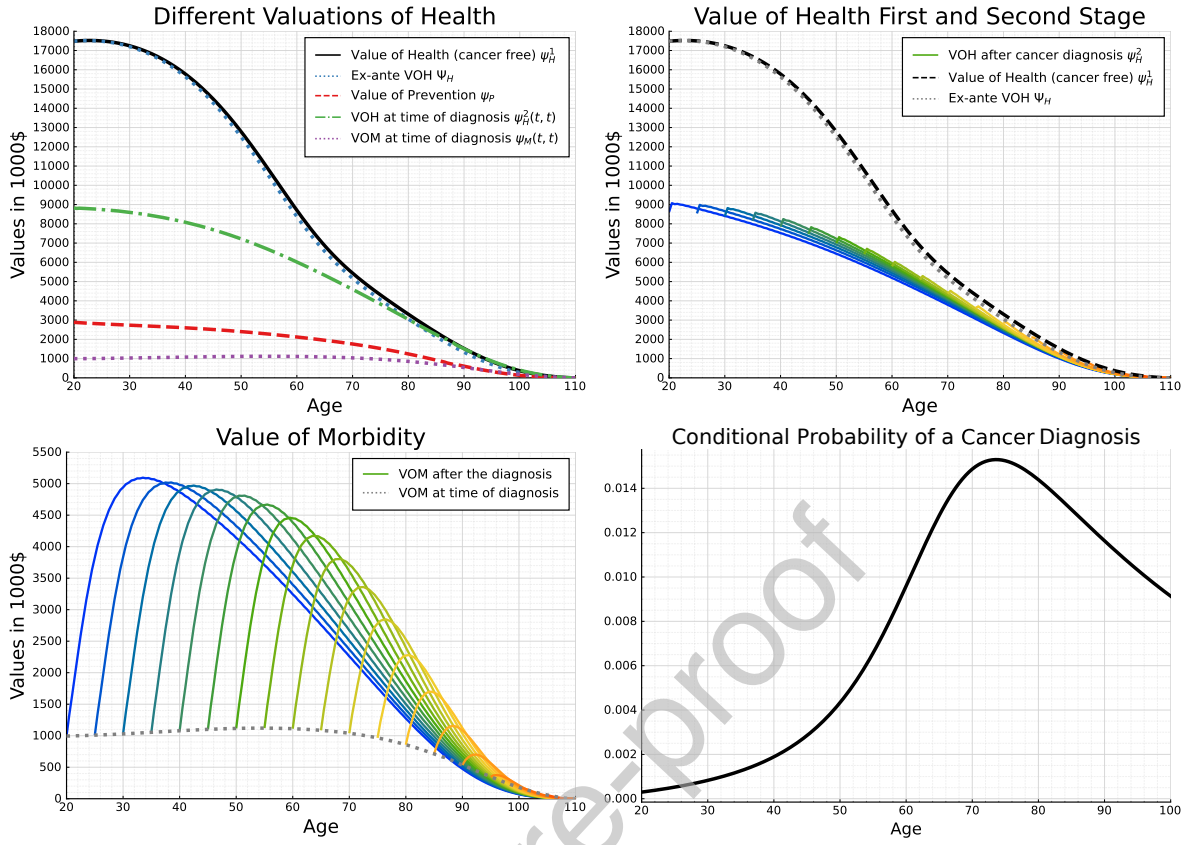

Figure 9: Valuations of Health

their prevalence is centered on high ages.

The lower left panel in Figure 9 shows the VOM after a cancer diagnosis for varying ages at the time of diagnosis. Notably, the VOM increases over the first 10-12 years to values more than four times the VOM at the time of diagnosis, followed by a continuous decline over the remaining life-course. These patterns can be understood by recalling from the FOC (37) that in the optimum, the VOM equals the effective price of reducing the cancer stock by one unit, $\psi_M = \frac{p^2}{-f_{h_2}}$. As cancer care becomes less effective with decreasing cancer stock, this implies an increasing effective price of reducing cancer (further). For an optimal allocation, this, in turn, implies that the VOM (i.e. the marginal rate of substitution between reductions in the cancer stock and consumption) must increase as well. Once the cancer stock has been reduced to its residual value after around ten years, the effective price of controlling it remains constant, and the VOM (and efforts towards controlling cancer) now decline in line with the reduction of the remaining life expectancy. These considerations have important implications when comparing the VOM across different types of cancer. Here, cancers which are more difficult to treat and, thus, exhibit a lower value of $-f_{h_2}$ also exhibit a higher VOM as willingness to pay for what is a "scarce" remedy. Notably, this willingness to pay will translate into a higher value of preventing such cancers (to the extent this is possible) and a high willingness to pay for medical progress against these particular cancers.

In Figure 10, we present a decomposition of the first-stage VOH for an optimal allocation

into the constituent valuations of the different aspects of first-stage health, as measured by S_1. [36] The solid (black) line equals the sum of the respective constituents and corresponds to the curve in the upper left panel in Figure 9. Unsurprisingly, the conventional VOL (dashed

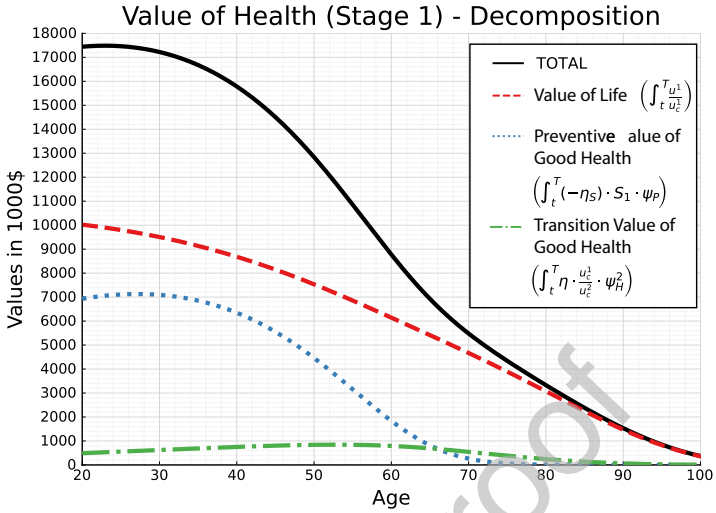

Figure 10: Decomposition of the value of Health before a cancer diagnosis according to Proposition 1

red line) adds the main share to the VOH over the whole life-cycle. Figure 10 shows, that our model implies a value of life of roughly \$8.7 million at age 40.[37] This is closely in line with the estimate of \$8.5 million (2015 U.S.dollars) derived in a meta-analysis by Masterman and Viscusi (2020). The VOL decreases nearly linearly with age and accounts for most of the VOH after age 70. Up to that age, the benefit of first-stage health through better prevention of cancer (dotted blue line) contributes significantly and explains more than a third of the cancer-free VOH at age 20. The remaining component of the VOH captures the fact that first-stage health, S_1, directly translates into initial health in the second stage, S_2, in the case of a diagnosis. Valued below \$1 million this part is relatively small compared to the others, but still far from insignificant in absolute terms.

Corollary 4 (Value of Health before diagnosis)**.** *The risk of a cancer diagnosis significantly increases the value of health for relatively young ages if health has a preventive effect on the hazard rate.*

5.4 Euler equations for health care

From the first-order optimality conditions for the different types of health expenditures presented in Proposition 2 we can derive Euler type equations for the evolution of general health care (before and after the diagnosis), chronic care, and preventive care over the life-cycle. In the following, we focus on general health care and chronic (cancer) care and relegate the analysis of

[36] Similar decompositions for other valuations as presented in Proposition 1 can be easily produced and are available upon request.

[37] According to Hammitt (2023) the statistical value of life is primarily estimated for individuals at age 40.

preventive care to the Online-Appendix. We will present the Euler equations in their general form but then cast the discussion in the context of our numerical analysis on (preventable) cancer.

The Euler equations for all types of health care share a similar pattern as they consist of three main terms: (a) the dynamics of the valuation of the relevant dimension(s) of health affected by the particular type of health care; [38] (b) changes in the effectiveness of health care due to changes in the relevant health or disease stocks but also due to external changes with age and time (medical progress); (c) the price path of the specific type of health care.

5.4.1 General health care

The Euler equations for general health care before and after a cancer diagnosis in equations (46) and (47) directly illustrates the three main contributing factors. Note that all terms are scaled with $\frac{-\mu^i_{b_i}}{\mu^i_{b_i b_i} b_i}$ respectively, which is equivalent to the inverse of the elasticity of mortality the mortality rate with respect to general health care, which in principle is available from medical studies.

$$\frac{\dot{b_1}}{b_1} = \frac{-\mu^1_{b_1}}{\mu^1_{b_1 b_1} b_1}\Bigg[-\frac{\mu^1_{b_1 S_1}}{\mu^1_{b_1}}\mu^1 S_1 + \frac{\mu^1_{b_1 t}}{\mu^1_{b_1}} - \frac{\dot{p^b}}{p^b} + \underbrace{+r + \theta\bar{\mu} + \mu^1_{S_1} S_1 - \frac{u^1/u^1_{c_1}}{\psi^1_H} + \eta_{S_1} S_1 \frac{\psi_P}{\psi^1_H} - \eta P \frac{u^2_{c_2}}{u^1_{c_1}}\Bigg\{ \frac{(\psi^2_H - \psi^1_H)}{\psi^1_H} + \frac{P_{S_1} S_1}{P}\frac{\psi^2_{life}}{\psi^1_H} + (-M^0_{S_1}) S_1 \frac{\psi_M}{\psi^1_H}\Bigg\}}_{=\frac{\dot{\psi^1_H}(t)}{\psi^1_H(t)}}\Bigg] \tag{46}$$

$$\frac{\dot{b_2}}{b_2} = \frac{-\mu^2_{b_2}}{\mu^2_{b_2 b_2} b_2}\Bigg[-\frac{\mu^2_{b_2 S_2}}{\mu^2_{b_2}}\mu^2 S_2 + \frac{\mu^2_{b_2 t}}{\mu^2_{b_2}} - \frac{\dot{p^b}}{p^b} + \underbrace{r + \theta\bar{\mu} + \mu^2_{S_2} S_2 - \frac{u^2/u^2_{c_2}}{\psi^2_H}}_{=\frac{\dot{\psi^2_H}(t,s)}{\psi^2_H(t,s)}}\Bigg] \tag{47}$$

We will now present the economic interpretations of the three driving forces (a), (b), and (c) in detail.

(a) Value of Health: The rate of change of the VOH, $\dot{\psi^1_H}(t)/\psi^1_H(t)$ resp. $\dot{\psi^2_H}(t,s)/\psi^2_H(t,s)$, plays a crucial part in the Euler equations. Using the results of Proposition 1 these rates can be decomposed into several parts. Parts (i) and (ii) discussed below show up in both stages and the interpretations are relevant for general health expenditures in both stages. Meanwhile the effects discussed in (iii)-(vi) are only present before a cancer diagnosis.

(i) The discount rate of the VOH ($r + \theta\bar{\mu} + \mu^i_{S_i} S_i \gtreqless 0$) incentivizes later health investments for high market returns ($r+\theta\bar{\mu}$). This effect is partially offset as the individuals take the effect of better health on the mortality rate into account ($\mu^i_{S_i} S_i \leq 0$).

[38] Hence, for the life-cycle pattern of general health care, the value of health is the decisive valuation, while for chronic care, the value of morbidity is a determining factor.

(ii) The year-by-year depreciation $-\frac{u^i/u^i_{c_i}}{\psi^i_H} \leq 0$ of the value of life (as part of the VOH) motivates health investments earlier in life.

(iii) The term $\eta_{S_1} S_1 \frac{\psi_P}{\psi^1_H} \leq 0$ implies the advancement of health investments towards younger ages for reasons of prevention, since S_1 lowers the hazard rate and decreases the probability of a cancer diagnosis. The extent of this effect depends on the value of prevention relative to the value of health.

(iv) According to $-\eta P \frac{u^2_{c2}}{u^1_{c_1}} \frac{(\psi^2_H - \psi^1_H)}{\psi^1_H} \gtreqless 0$ health investments are delayed (for precautionary reasons) to the later less-risky stage in life if the value of health is subject to the risk of instantaneous depreciation due to a health shock (corresponding to a drop $\psi^2_H - \psi^1_H) < 0$ as shown in Figure 9). This factor becomes more pronounced at ages at which a cancer diagnosis is more likely (i.e. when η is higher).

(v) The term $-\eta \frac{u^2_{c2}}{u^1_{c_1}} P_{S_1} S_1 \frac{\psi^2_{life}}{\psi^1_H} \leq 0$ accounts for the advancement of health care for the purpose of boosting the protective effect of health S_1 against instantaneous mortality at the point of a shock and, thus, for increasing the continuation probability, P. *This effect is equal to zero in our scenario, as health has no impact on the continuation probability*

(vi) The term $-\eta P \frac{u^2_{c2}}{u^1_{c_1}} (-M^0_{S_1}) S_1 \frac{\psi_M}{\psi^1_H} \leq 0$ accounts for the advancement of health care for the purpose of boosting the protective effect of health S_1 against a high level of disability or chronic disease, M^0, in the aftermath of a shock and when entering the second stage. *This effect is equal to zero in our scenario, as health has no impact on the initial deficits.*

(b) Effectiveness: Changes in the effectiveness of health care over the life-cycle also play a decisive role for the shape of the health care profile. This aspect contains two distinct parts:

(i) The term $-\frac{\mu^i_{b_i S_i}}{\mu^i_{b_i}} \mu^i S_i$ relates the effectiveness of health care to the health state. In the plausible case that the effectiveness of health care decreases in the level of health, such that $(\mu^i_{b_i S_i} > 0)$, individuals tend to defer the utilization of health care into (later) life stages where a poorer health state renders the use of (medical) health care more effective. *This effect is equal to zero in our scenario, as health has no impact on the base mortality rate.*

(ii) According to $\frac{\mu^i_{b_i t}}{\mu^i_{b_i}}$ individuals have an incentive to postpone their health investment to later ages, if the marginal effectiveness of health expenditures increases directly with age or if there is an expectation of increasing effectiveness over time due to medical progress $(\mu^i_{b_i t} < 0)$.

(c) Price: Depending on the rate of medical price change $-\frac{\dot{p^b}}{p^b}$ individuals advance the utilization of health care if they expect medical price inflation over time, and vice versa for expected reductions in prices. *This effect is equal to zero in our scenario, as prices are assumed to be constant.*

To quantify the relevant drivers for our numerical example[39] we present a numerical attribution of the (log) difference between the current utilization of stage-1 general health care at age/time t and its initial utilization at $t_0 = 20$ to the different components of the Euler equation. In so doing, we follow an analogous decomposition approach to the one taken in Section 5.2 when attributing consumption change to its drivers in Figure 6.

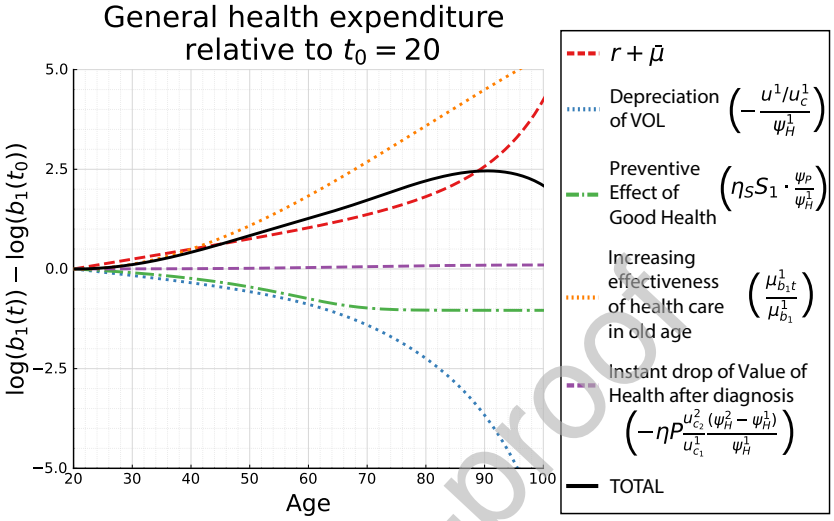

Figure 11: Decomposition of (log) difference between current health expenditures and initial health expenditures at age 20 before a diagnosis.

We can easily identify the two main driving forces behind the overall increase in health expenditures over the life-cycle. First, the sum of market and annuity interest strongly incentivizes a postponement in health expenditure (dashed red/dark grey line) especially towards the end of life, where the annuity rate increases in tandem with mortality. The second incentive to postpone the utilization of health care is the increasing effectiveness of general health care with age (dotted orange/light grey line), which by itself would imply consistently increasing health expenditures after the age of 45.

The effect of these two drivers is dampened by two counterveiling factors. While the preventive aspect of better health leads to an advancement of health care only up to around age 70 (dash-dotted green/grey line), the depreciation of the value of life (dotted blue/grey line) gains in strength over the life-course. This leads to a flattening-out and subsequent decrease of health care expenditures by around age 90. Finally, we see that the risk of losing VOH in the course of a cancer diagnosis has no significant impact throughout the life-course. While the difference between the first- and second stage VOH is significant for ages below 70, the hazard of contracting cancer is so low that this does not translate into a sizeable delay of health care spending.

The analogous numerical evaluation of general health expenditure after a diagnosis is relegated to the online appendix. However, we again summarize our findings for general health care expenditure in both stages in the following corollary.

[39] As outlined above, some of the terms are equal to zero due to the assumptions made for the scenario of a cancer diagnosis and are therefore omitted in the presentation in the figures below.

Corollary 5 (General Health Expenditure)**.** *The main driving forces behind increasing health expenditures over the life-course are the market interest and annuity rate and the increasing effectiveness of health care in older ages. The dampening effect of the depreciation of the value of life is enhanced by an additional term in the presence of a potential cancer diagnosis: if better health leads to a reduction in the cancer hazard, this leads to front-loaded health care spending for preventive reasons. This term is effective only before a cancer diagnosis and, for this reason, only shapes the behaviour of cancer-free individuals.*

5.4.2 Cancer-specific chronic care expenditure

Finally, we focus on cancer care after diagnosis, present the corresponding Euler equation, and again attribute the expenditure profile to its drivers along the optimal numeric solution. The dynamics of chronic care depends on the age at diagnosis in quantitative terms, but is always shaped by the same forces. As with the prior Euler-type equations, all terms in equation (48) are scaled with $\frac{-f_{h_2}}{f_{h_2h_2}h_2}$, the inverse elasticity of the disease state with respect to chronic care.

$$\frac{\dot{h_2}}{h_2} = \frac{-f_{h_2}}{f_{h_2h_2}h_2}\Bigg[\underbrace{r+\theta\bar{\mu}-f_M+\frac{w_M^2}{\psi_M}+\frac{u_M^2/u_{c_2}^2}{\psi_M}-\mu_M^2\frac{\psi_H^2}{\psi_M}}_{=\frac{\dot{\psi_M}(t,s)}{\psi_M(t,s)}}+\frac{f_{h_2t}}{f_{h_2}}+\frac{f_{h_2M}f}{f_{h_2}}-\frac{\dot{p^2}}{p^2}\Bigg] \tag{48}$$

We obtain the same three main components of the dynamics: (a) change in the value of morbidity, (b) change in the effectiveness of chronic care; and (c) change in price.

(a) Value of Morbidity: As shown in Figure 9 the value of morbidity follows a hump-shaped pattern and we provided an intuitive explanation for this somewhat surprising pattern. However, decomposing its rate of change $\frac{\dot{\psi_M}(t,s)}{\psi_M(t,s)}$ can enhance our understanding further:

(i) The discount rate of the VOM ($r+\theta\bar{\mu}-f_M$) leads to chronic care postponement for high market rates ($r+\theta\bar{\mu}$). If deficits (or the disease stock) accumulate at a decreasing (increasing) rate, i.e. if $f_M<0$ ($f_M>0$) individuals further delay (advance) their chronic care expenditures.

(ii) The negative impact of the deficit/disease stock on earnings $\frac{w_M^2}{\psi_M}\leq 0$ induces individuals to utilize chronic care closer to the time of the diagnosis. *This term is equal to zero in our scenario, as the cancer stock is assumed to have no impact on earnings.*

(iii) The utility loss from disease $\frac{u_M^2/u_{c_2}^2}{\psi_M}\leq 0$ is another reason for individuals to invest into chronic care earlier. The extent of this effect depends on the ratio of the marginal disutility of disease and the marginal utility of consumption, which is tantamount to a stage-2 willingness-to-pay for avoiding disutility of disease.

(iv) The term $-\mu_M^2\frac{\psi_H^2}{\psi_M}\leq 0$ implies an advancement of chronic care as an increasing deficit/disease stock elevates mortality and the depreciation of the general health

stock S_2. The strength of this effect depends on the stage-2 VOH relative to the VOM.[40]

(b) Effectiveness: Changes in the effectiveness of chronic care over the disease-course also play a decisive role for the shape of the chronic care profile:

(i) The term $\frac{f_{h_2 M} f}{f_{h_2}} \gtreqless 0$ relates the effectiveness of chronic care to the deficit/disease state. In the case that the effectiveness of chronic care increases in the level of deficits for a progressive disease with $f > 0$ we have $\frac{f_{h_2 M} f}{f_{h_2}} > 0$ and individuals tend to defer the utilization of health care into (later) life stages where a more advanced disease state renders the use of (medical) health care more effective. However, alternative constellations can be conceived of, where treatment of the disease is most effective in its early stage, such that $\frac{f_{h_2 M} f}{f_{h_2}} < 0$ (which is the case in our setting of cancer treatment) and the utilization of chronic care would be advanced. Further cases need to be considered for recessive disease with $f < 0$.

(ii) The term $\frac{f_{h_2 t}}{f_{h_2}} \gtreqless 0$ describes the change of chronic care effectiveness with age and over time if medical progress is taken into account. While in the latter case, $\frac{f_{h_2 t}}{f_{h_2}} > 0$ implies a deferral of chronic care, the relationship with age is less clear-cut. *This term is equal to zero in our scenario, as the progression of cancer is assumed to be independent of age and no medical progress is considered.*

(c) Price: According to $-\frac{\dot{p^2}}{p^2}$, expected increases of the price of cancer care advance its utilization closer to the diagnosis (and vice versa for an expected lowering in price). *This term is equal to zero in our scenario, as prices are assumed to be constant.*

To quantify the various drivers of cancer care in our numerical example, Figure 12 presents the numerical attribution of the (log) difference between the current utilization of cancer care at age/time t and its initial utilization at $t_0 = 20$ to the different components of the Euler equation for an early (30 years; left panel) and late (70 years; right panel) age of diagnosis.[41]

Similarly to general health care expenditures, the total interest rate $r + \theta\bar{\mu}$ alone would lead to a deferral of cancer care, especially in late life (dashed red/dark grey line). As $f_M < 0$ can be shown for our parameterization, the decreasing rate of accumulation of cancer stock implies a further delay in the utilization of cancer care (dotted blue/dark grey line). Notably, this impact is a particularly strong force for both an early and late age at diagnosis.

These forces are offset by three factors that push towards an advancement of cancer care. In particular, the positive impact of cancer stock on mortality induces a strong incentive for advancing cancer care (dashed violet/grey line). This incentive is complemented by the individual's desire to contain the impact of cancer on utility (dash-dotted green/light grey line), albeit this incentive is comparatively small and only increases in magnitude at very late

[40] This term has an intuitive interpretation for the case of an asymptomatic diseases, i.e. diseases which are not associated with earnings or utility loss. Here, the extent to which such a disease leads to the erosion of general health continues to provide a strong case for early intervention, as is captured by this term.

[41] Again, we follow the decomposition approach taken in Section 5.2 when attributing consumption change to its drivers in Figure 6.

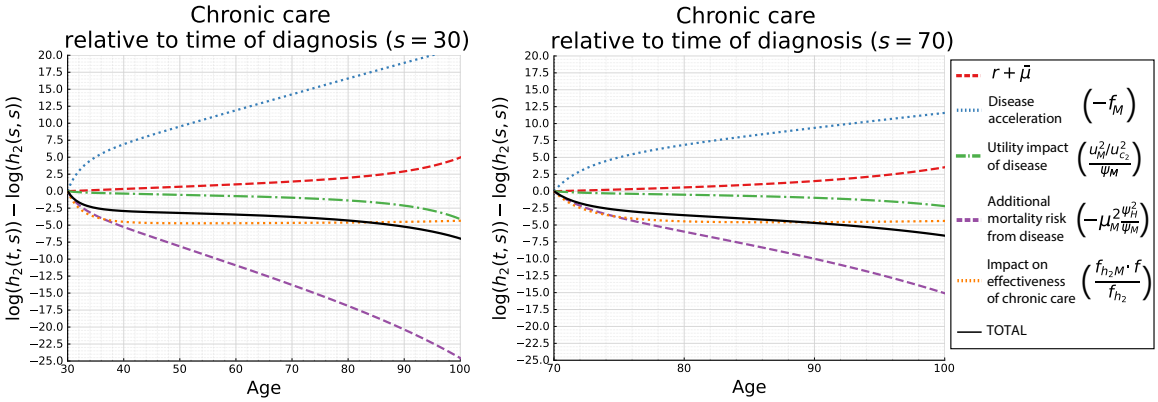

Figure 12: Decomposition of (log) difference between current chronic care and initial chronic care expenditures after diagnosis for an early (30 years, left panel) and late (70 years, right panel) age at diagnosis.

ages. Finally, the dotted orange/light grey line shows that for the increasing effectiveness of cancer care in cancer stock ($f_{h_2M} < 0$) that is implied by our functional specification (see Equation (42)) implies that cancer care is advanced to an early stage close to the diagnosis, where the deficit stock is particularly large. Following the reduction of the cancer stock to a residual level, this channel has no significant impact anymore after around ten years since diagnosis. Adding all terms up, we obtain the total profile (solid black line), which highlights the dominance of negative terms over the positive ones. The strongest decrease in the cancer expenditure profile occurs right after the diagnosis, which is followed by a period of almost flat (if slightly diminishing) expenditures.

Corollary 6 (Cancer treatment)**.** *The initiation of intensive cancer treatment immediately after the diagnosis and its subsequent reduction to a low level (recall the pattern in Figure 7) is mainly driven by the need to curb the high additional mortality risk early on as well as by decreasing effectiveness of cancer care for a decreasing cancer stock. The combined impact is attenuated as the direct effect of the cancer stock on cancer progression (under the optimal cancer care regime) acts in an opposing way. The age at diagnosis has no substantial impact on the qualitative structure of the relative chronic care profiles. However, due to differences in the initial levels of chronic care, the decline is more pronounced among individuals diagnosed at younger ages.*

5.5 Bounded Rationality

This section addresses the impact of bounded rationality and incomplete information on the life-cycle allocation under the risk of acquiring a preventable cancer. For our numerical exercise so far, we have assumed a perfectly rational individual who is fully aware of the hazard of a cancer diagnosis. As shown in Equation 4 this corresponds to the value of $\pi = 1$. To isolate the effect of bounded rationality with respect to the cancer diagnosis we will illustrate the differences in life-cycles decisions and outcomes for individuals exhibiting values of $\pi = 0.1$ ("the oblivious individual") and $\pi = 2$ ("the anxious individual") keeping the rest of the model

unchanged. While the oblivious individual consistently underestimates the hazard rate of a cancer diagnosis the anxious individual overestimates this risk.[42] Both variations could arise either through irrational individual assessment of cancer risk or through limited/incomplete information available to the individual.[43]. We should stress at this point that the nature of this analysis is explorative. While the role of biased believes is probably most salient in regard to the "large-scale" health risks that are the subject of our analysis, one would expect that similar bias (in either direction) applies to other health risks and, thus, to the more general survival risk as well. Not least for the purpose of a "clean" comparison we abstract from such correlated beliefs and assume that belief bias applies to the hazard rate alone.

5.5.1 Differences in behaviour before diagnosis

It is easy to see that belief bias in regard to the hazard rate of a cancer diagnosis directly affects the behaviour in the cancer-free first stage with only indirect repercussions on behaviours in the (second) stage after diagnosis. For this reason, we contain our presentation to first-stage behaviours while only hinting at the second-stage knock-on effects, some of which are presented in greater detail in the Online-Appendix.

We begin by focusing on the differences in expenditure allocations of the oblivious and anxious individuals as compared to the rational one. The upper panel of Figure 13 shows the general health expenditure profiles for the cancer-free stage, and we can identify a rather intuitive pattern. The anxious individual frontloads health expenditure while the oblivious individual defers them to later periods of the life-cycle. The lower three panels provide a more formal explanation for these shifts. Each panel shows the decomposition of the value of health (which directly determines the health expenditures) as already presented in Figure 10.[44] It is apparent that the preventive aspect of health (blue dotted line) gets overvalued (resp. undervalued) by the anxious (oblivious) individual. This leads to substantial shifts in the total value of health which consequently drive health expenditure decisions. Notably, the anxious (oblivious) individual is spending less (more) on health care than both the fully rational individual during the later periods of life. This reversal in spending patterns can be inferred from the total VOH which can be verified to be lower (higher) for the anxious (oblivious) individuals from around age 65 onwards.[45] This reversion in valuations is down to the fact that greater (lower) early-life utilization of health care by anxious (oblivious) individuals is also lowers (increases) their scope for savings and thus lowers (boosts) their later life resources (as can be seen in the upper right panel of Figure 14).

[42]The terms oblivious and anxious are used solely as descriptive labels. They are not meant to imply a psychological interpretation but are introduced to provide an intuitive distinction between the two scenarios and to simplify the discussion in this section.

[43]Note that we do not calculate a new balanced annuity market for these individuals, but assume that they are facing the exact same internal (mortality, hazard rate, preferences) and external (annuity rate, interest rate) conditions as a rational individual.

[44]Note that the value of health is a subjective evaluation of the individual and therefore depends on the subjective hazard rate of a cancer diagnosis.

[45]While the VOH of a rational individual drops to $4 million at age 76, the VOH of the anxious individual has reached this mark already at age 74. However, the oblivious individual exhibits a subjective VOH above $4 million until age 80.

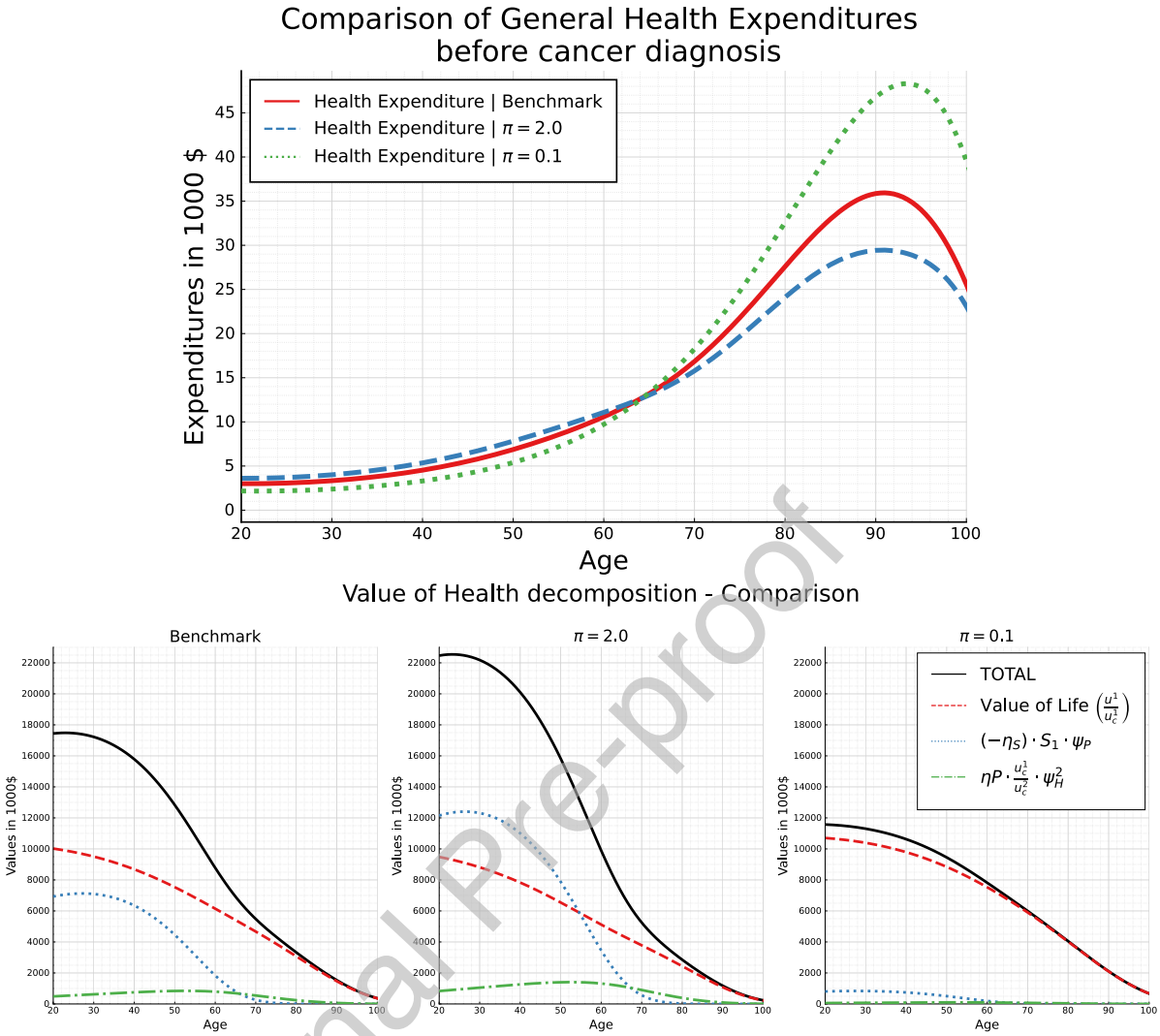

Figure 13: Comparison of the health expenditure profiles and the decomposition of the value of health of rational, anxious, and oblivious individuals before a cancer diagnosis.

Switching to the consumption profiles illustrated in the upper left panel of Figure 14 we find that interestingly, the consumption paths look almost identical until the age of 50, at which point a gap emerges and continues to widen to the disadvantage (advantage) of the anxious (oblivious) individual. Here, the decomposition of the Euler equations as presented in Section 5.2 provides helpful information. The lower three panels show that the two effects related to the (subjective) hazard rate (green dash-dotted and purple dashed lines) drive the differences late in life. Furthermore, analyzing the asset profiles in the right upper panel shows that the increase in consumption for the oblivious individual is possible, as she is able to accumulate more wealth over time given the lower health expenditures in early ages. These increased financial resources also allow the oblivious individual to respond to a cancer diagnosis with substantially higher cancer care but also with higher general health expenditures late in life.[46] Thus, by underspending on health care early-on in life, the oblivious individual is able to

[46]The relevant figures are available in the Online-Appendix.

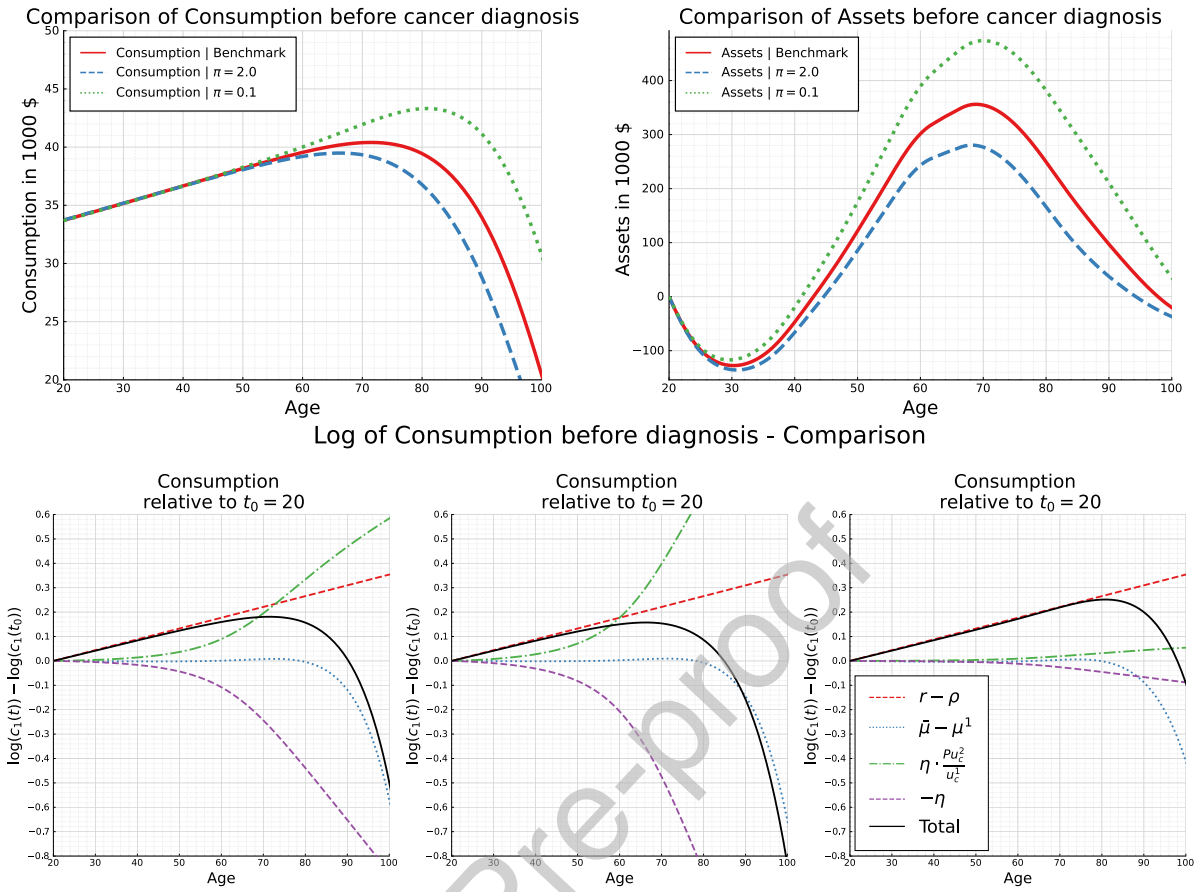

Figure 14: Comparison of the consumption profiles and their Euler decompositions of rational, anxious, and oblivious individuals before a cancer diagnosis.

accumulate more resources for mitigating cancer in case of a diagnosis and, more generally, for enhancing health in advanced life years. In more general and intuitive terms, this corresponds to a shift from the prevention of damage towards its mitigation under the perception of too low a risk.

Taking into account that the differences in health S_1 and (objective) survival in good health Z_1 across anxious, oblivious and rational individuals are all in the order of 10^{-2} and given that the higher financial capabilities allow oblivious individuals to sustain both higher consumption and health care spending after a diagnosis and in later life more generally,[47] the question arises as to whether an oblivious individual is actually better off than a rational individual. To investigate this issue, we will employ the concept of consumption-equivalent welfare.

5.5.2 Consumption-equivalent welfare

As employed by Jones and Klenow (2016) for the comparison of life-cycle allocations, consumption-equivalent welfare describes by which percentage the consumption profile of the reference scenario (the rational individual in our case) needs to be adjusted to match the life-time utility

[47] Again we refer to the Online-Appendix for all the relevant illustrations.

of the comparison scenario (corresponding to the anxious or oblivious individual).[48]

In our framework, the objective function directly depends on 4 different variables: (i) the health/survival profiles $S_1(t)$ and $S_2(t,s)$; (ii) the profiles of survival in good health and cancer incidence, $Z_1(t)$ and $Z_2(s)$, respectively; (iii) the consumption profiles $c_1(t)$ and $c_2(t,s)$; and (iv) the cancer deficit profiles $M(t,s)$. In Table 4, we present the percentage change in the consumption of a rational individual that would make her indifferent to the life-cycle allocation of the respective bounded rational individual. In so doing, we consider both the difference between the full allocation and the contributions of the individual channels (i)-(iv) described above. The first row highlights that, unexpectedly perhaps, the expected life-time utility of the oblivious individual actually corresponds to a larger reduction in consumption than the expected life-time utility of an anxious individual.[49] While a rational individual's consumption profile needs to be reduced by 0.67% to match the life-time utility of the oblivious individual, the reduction amounts to only 0.25% for the anxious individual. Although both of these values are below 1% and thus appear rather small, we should stress that these differences only arise from the bias in the assessment of the hazard rate (which in its own right is low during the relevant early years of life), while all other characteristics of the individuals are identical. Hence, we nevertheless consider these effects to be substantial. Furthermore, note that the monetary values of these relative consumption changes correspond to some 19,500$ (oblivious individual) and 7,300$ (anxious individual). These values can be seen as the values of information for the anxious and oblivious individuals and provide a strong argument for the societal value of cancer awareness and information programs (given these values hold on per-capita basis).[50]

The remaining rows of Table 4 provide a decomposition analysis that attributes the equivalent-consumption adjustment to the four different channels. In each row we evaluated the life-time utility of the rational individual with just a single type of profile switched to the one of the bounded rational individual. Here we find that it would take a 1.18% increase in consumption of the rational individual to match the oblivious individual's consumption profile and an increase of 0.01% would be necessary to compensate for the faster decreasing cancer deficits (enabled through higher chronic care). However, these advantages of obliviousness are overcompensated by a reduction in consumption by some 0.72% to offset the early loss in general health in the cancer-free stage for an oblivious individual and an even higher reduction by 1.13% to offset the increased incidence of cancer.

For the anxious individual, the direction of these individual effects is reversed. The anxious individual benefits from better health and lower cancer incidence (by 0.1% and 0.56% of

[48] The anxious and oblivious individual use their subjective expectations about the cancer incidence in their maximization of life-time utility. However, the realized utility is of course determined by the true cancer hazard rate (which the rational individual uses). The life-time utility values discussed in this section always describe the actual life-time utility rather than the subjective values of the bounded rational individuals.

[49] We conjecture that the ordering between the oblivious and anxious outcomes may reverse for a set-up in which individuals face significantly lower earnings. This is because of the income gradient in the value of life (and health) (see e.g. Hall and Jones, 2007; Frankovic et al., 2020b). Poorer individuals would thus prefer the greater volume of consumption that comes with the under-estimation of a cancer risk.

[50] The finding that bounded rationality lowers individual welfare in a partial equilibrium setting with given annuity prices may be reversed in general equilibrium owing to the presence of fiscal externalities (Feigenbaum et al., 2013; Heijdra et al., 2014).

equivalent consumption) but looses through the lower consumption level (by 0.9% of equivalent consumption). A full table with all possible combinations of the four channels can be found in the Online-Appendix, but the individual effects are close to linear if activated simultaneously.

Counterfactual Decomposition				**Equivalent Consumption Adjustments %**	
Survival	Cancer Incidence	Consumption	Cancer Deficits	$\pi = 2$	$\pi = 0.1$
✓	✓	✓	✓	**-0.25**	**-0.67**
✓				0.10	-0.72
	✓			0.56	-1.13
		✓		-0.90	1.18
			✓	-0.01	0.01

Table 4: Evaluation of the equivalent consumption adjustments for anxious and oblivious individuals and a decomposition into the four driving forces.

6 Conclusions

We have constructed a life-cycle model in which individuals respond to the risk of a singular, life-changing shock to their health (e.g. heart attack, stroke, cancer, diabetes, disabling accident) by utilizing and investing in a range of distinct forms of health care: preventive care to reduce, directly or indirectly, the arrival rate of the shock; acute care to lower instantaneous survival and the extent of morbidity/disability at the point of the shock; and chronic care to lower mortality and morbidity in the follow-up of the shock; as well as general health care. We solve the underlying stochastic optimal control problem with a random time horizon by applying an innovative transformation into an age-structured control model. This enables us to derive intuitive expressions of the first-order conditions for the choice of health care based on their respective (monetary) valuations. These conditions affirm the intuitive notion that the optimal choice of health care equalizes the marginal costs of any additional effort with the monetary valuation of the respective aspect of health both across types of health care and time.

Calibrated to US data, our model provides a good fit between (i) general and cancer-specific health care expenditure, (ii) age-specific survival and incidence of cancer; (iii) age- and duration-specific cancer mortality rates, as well as (iv) average cancer and non-cancer mortality. Under the assumption of an incomplete annuity market, where the return of annuities is based on the average mortality rate, relatively young individuals respond to a cancer diagnosis by adjusting their consumption in light of an increased mortality risk, from a hump-shaped pattern into a gradually decreasing pattern (after a brief initial adjustment phase). To finance intensive cancer care over a certain time frame after the diagnosis, individuals with cancer reallocate resources from consumption and health expenditures, placing both respective profiles at a lower level compared to cancer free individuals. Given the possibility to do so in an incomplete annuity market, individuals with a diagnosis run-up substantial debt to finance cancer-specific health care. With the duration of the disease, cancer-specific health care is reduced, reflecting an intuitive tendency of such care to evolve from urgent life-protecting care into less intensive

chronic care, conditional on the individual's survival. For individuals who are diagnosed with cancer late in life, a boost to consumption immediately after diagnosis coincides with a drop in general health expenditures, as well as with a smaller investment in cancer-specific care, showing that a cancer diagnosis affects individuals differently at different points in the life-cycle. However, consumption of individuals diagnosed during old age subsequently falls quickly to levels below the consumption of healthy individuals.

We calculate various expressions for the value of health. For our calibration, the value of health before a cancer diagnosis starts from a level of around $17.5 million at age 20 and then declines steadily. Our analysis shows that up to around age 70 a cancer diagnosis drastically reduces the value of health. Although both the cancer-free value of health and the value of health at the point of diagnosis decline with age, the decline is much more pronounced for the cancer-free value of health. From around age 85 onwards, the cancer-free value of health no longer differs much from the value at the onset of diagnosis, reflecting the much diminished remaining life-time in either case.

Contrasting the cancer-free value of health with the expected value of health, which averages across health states - cancer-free vs. the distribution of cancer states according to their arrival time - we find little difference between the two. This may come as a surprise given the strong drop in the value of health for individuals diagnosed at a young age. However, since cancer is an unlikely diagnosis for young individuals, the loss in the value of health carries little weight in the calculation of the expected value of health. Although prevalence is higher for advanced age groups, a cancer diagnosis in these groups no longer leads to a strong decrease in the value of health. For these offsetting tendencies, the expected value of health remains close to the cancer-free value of health, for both young and old age-groups. Our analysis also shows that the close match between the expected and cancer-free value of health is likely to hold only for diseases for which incidence and prevalence are clustered at high ages. For debilitating diseases, such as diabetes, for which the incidence is high even earlier in life, as well as for infectious diseases, the drop in the value of health that occurs at the point of an early onset of disease may receive a higher prevalence weight and, thus, lead to a more substantial gap between the expected and disease-free value of life.

Considering the components of the cancer-free value of health, we find that while the largest part falls on the value of survival (tantamount to the conventional value of life), the preventive value of good health (i.e. the value of prevention weighted with the health-related reduction in the incidence of cancer) makes up for a significant share in excess of 30 percent of the total value at age 20. From age 50 onward, the preventive value of good health diminishes quickly and vanishes almost entirely for the highest ages.

Finally, turning to the Euler equations, we see that three forces emerge as drivers of consumption in different stages of the life-cycle while in the cancer-free state. With the interest rate surpassing the discount rate individuals have an incentive to increase consumption throughout their life-course. As the return to partial annuitization does not fully compensate the steep increase in mortality risk after the age of 80, this induces individuals to lower consumption during the risky oldest ages, and consequently leads to the overall hump-shaped profile of

cancer-free consumption. On top of this general pattern, uninsured cancer risk leads to an additional incentive for the advancement of consumption within the cancer-free stage, especially for higher ages, thus, intensifying the hump-shaped pattern. After the onset of cancer, two main forces determine the consumption path: With cancer reducing the marginal utility of consumption, individuals tend to defer consumption into the future when (conditional on survival) they expect to have overcome or at least controlled cancer. However, for the most part, this effect is offset by the desire of individuals to consume instantaneously given the high mortality risk, following the onset of cancer.

The demand for all types of health care is shaped by the return to annuities, which tends to imply an increase, and the writing-off of life-years from the respective valuation of health (value of health resp. value of morbidity), which tends to imply a decline. For general health care, these effects are moderated by age-related changes in effectiveness. Furthermore, for cancer-free individuals the role of health for the prevention of cancer implies an additional incentive to advance the utilization of health care. Overall, we observe a moderate increase in the utilization of general health care over the life-course both in the absence and, from a lower level, in the presence of cancer. For cancer-specific health care, we identified two additional effects: On the one hand, individuals have an incentive to delay the investment in cancer care, given it is more effective for a disease that has (somewhat) progressed already; on the other hand, the mortality risk that increases with the progression of cancer provides a strong incentive to advance cancer care; and this is, indeed, the dominating effect.

Relaxing the assumption of a perfectly rational and informed individual, we find that individuals who overestimate the hazard of a cancer diagnosis further advance the utilization of health care for preventive reasons, which lowers the incidence of cancer below the level of a fully-informed rational individual but also draws down assets to a point that both later-life consumption and health care spending fall short of what is achieved by a rational individual. If, by contrast, the individual underestimates the risk of a health shock, health care utilization in early years is reduced. A side-effect of the under-utilization of health care is the increased accumulation of financial assets to a point that allows an individual that is under-estimating the cancer risk to expend more on consumption and health care both when remaining cancer-free and in case of a diagnosis. Surprisingly, such an individual then even faces a somewhat better survival prospect upon the contraction of cancer. However, our analysis of welfare-equivalent consumption adjustments proves that the rational and perfectly informed individual is nevertheless better off due to lower incidence and higher unconditional survival from an ex-ante perspective.

At the micro-level, our model provides a useful framework for understanding resource allocations across different types of health care as well as along treatment pathways. While we illustrate the allocation patterns for pre- and post-shock general health care, including its preventive aspect, and chronic care for the case of cancer, our framework allows a broader understanding of the patterns of four types of health care, including dedicated preventive care, acute care, chronic care and general health care, which cover both preventive and curative aspects. Notably, this understanding is informed by an understanding of the levels, time-age

trajectories, and health-state contingencies of the valuations of different aspects of health, including the value of life (survival), the value of prevention and the value of morbidity (reductions). These specific monetary valuations can be combined with estimates of the effectiveness and nominal prices of treatments to provide allocation rules that, in principle, can be grounded in real-world data. Our analysis also shows how a revealed preference argument can be used to elicit values of certain aspects of health care from information on prices, medical effectiveness, and the demand for the relevant type of health care. When aggregating the valuations to population level our framework provides, in extension to the pioneering work by Murphy and Topel (2006), a basis for the comparative valuation of (novel) treatments and health care programs that address certain diseases from different angles, e.g. the comparison of a screening or vaccination program that helps to prevent a disease as opposed to an intensive method that helps to cure the disease as opposed to a chronic treatment pathway. Here, our framework is general enough to account for side-effects and risks associated with different treatments.

Furthermore, our framework is prone to inform broader health policy-making by allowing for an assessment of how health-related behaviours and outcomes in respect to large health shocks vary with the particular disease, the institutional context and, importantly, socio-economic context (income, education) of the individual. Such an understanding can inform health policy in particular in respect to the design of health and disability insurance, where the specific arrangements drive the consumption of particular types of health care - a typical example being the potential under-consumption of preventive health care if the cost of curative health care is insured. By explicating the underlying valuations and dynamic aspects of distinct types of health and health care, our framework allows the scope to go beyond more stylized models of health insurance. Finally, we have illustrated the scope of our framework to study the role of bounded rationality and missing information for the choice of health care in such complex medical settings. This includes, the possibility to provide an assessment of the value of information and ultimately the monetary value of health awareness programs.

Although mostly to be understood as a micro-economic framework, our model allows an important perspective on the adequacy of more stylized representative agent life-cycle models which feature no shocks or shocks that are modeled in a more parsimonious fashion but may be used to study more macro-oriented issues. Here, we can draw on whether the value of health in a representative agent model without health shocks, which is tantamount to the first stage value of health in our model, is a good proxy for the expected value of health when taken across a population of disease-free and affected individuals. As our findings show, this is the case for diseases that have their onset and, thus, their prevalence late in life, which is arguably true for most non-communicable diseases. With a view to these diseases, the express modelling of health shocks is nevertheless important to understand medical allocations and health policies at the micro-level but can be abstracted from for the purpose of macro-models, such as the approaches taken in Murphy and Topel (2006) who relate to heart disease and cancer and Frankovic et al. (2020a) who relate to heart disease without modelling the underlying shocks. A more cautious stance is warranted if the incidence of disease is more evenly distributed, implying significant prevalence also among the younger population.

Beyond the policy applications discussed above, there is scope for further extensions of our model. First, we are planning to provide analysis for other types of health shocks, in particular cardio-vascular events that involve an instantaneous threat to survival and, thus, warrant the consumption of acute health care. Second, especially in the case of cancer, there is an important issue about the lag between onset and diagnosis, which we plan to address in future work. Third, there is scope for employing the model to study the role of the annuity market and the role of health and disability insurance on the financial side; the role of medical progress in regard to different treatment regimes on the provision side; both of these set against the back-drop of socio-economic heterogeneity. Fourth, there is scope to extend our work to allow for imperfect information and, thus, ambiguity, on the underlying hazard and mortality rates. Finally, we are considering extensions of the model to involve multiple shocks and differences in the severity of the health shock as a second dimension of uncertainty.

7 Declaration of interests

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

Michael Freiberger reports financial support was provided by Austrian Science Fund. Stefan Wrzaczek reports financial support was provided by Austrian Science Fund. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendices

Appendix A Estimation and calibration strategies

Note that some parameter values shown in Table I are not the result of an automated calibration process, but are manually chosen to improve the calibration fit. The high complexity of the model and intricacy of the solution process in addition to relatively long computation times for one set of parameters did not allow us to conduct standard calibration methods. Hence the parameters $\delta_1, \delta_2, \delta_3$ and κ_0, κ_1 are not chosen through a process minimizing a strictly defined quality of calibration criterion (e.g. minimizing the maximum absolute difference in the cancer mortality rates), but are manually set to obtain a qualitatively appropriate fit of the several profiles presented. For all the other parameters we are going to present the calibration strategy in the next subsections. Furthermore empirical calibration targets are not readily available and have to be constructed by combining several different data sources.

A.1 Construction of general and cancer specific health care expenditure

As general health expenditure data is not available conditional on the time of the cancer diagnosis in general, we are not able to construct separate empirical data profiles for b_1 and b_2 directly. However, we can construct an approximation for the average non-cancer health care expenditure $\bar{b}(t)$, which is defined in our framework as

$$\bar{b}(t) = Z_1(t)b_1(t) + \int_0^t Z_2(s)b_2(t,s)ds.$$

We use the general health expenditure profile created by the National Transfer Accounts (NTA) initiative, which include the total health care expenditures by an individual (out-of-pocket and insurance covered) at every age throughout the life-cycle. Consequently we subtract the age-specific share of cancer-specific health expenditure sourced from `healthdata.org` to obtain an estimated profile for the general health care expenditure in our model. Similarly we can compute an estimate for the average cancer specific health expenditures, which is defined in our framework as

$$\bar{h}(t) = \int_0^t Z_2(s)h_2(t,s)ds. \tag{49}$$

A.2 Construction of the cancer-free survival profile

Taking the total age-specific mortality rates from the Human Mortality Database (HMD) does not provide an adequate estimate for the general mortality rate and the survival profile in first stage respectively. Luckily, the SEER database provides age-specific mortality rates for cancer. Building the difference between the two mortality rates allows us to construct a non-cancer mortality rate and consequently an empirical profile for the non-cancer survival function of the first stage.

A.3 Calibration of the base mortality function $\mu^b(t,b)$

We use a very general form of the mortality function, so we are able to match the health expenditure profile and the survival profile at the same time, i.e. we assume the functional form

$$\mu^b(t,S_1,b_1)=\mu^b(t,b_1)=g(t)b_1^{\varepsilon(t)} \qquad g(t)=\exp\left\{\gamma_0+\gamma_1 t+\gamma_2 t^2+\gamma_3 t^3\right\}$$
$$\varepsilon(t)=\alpha_0+\alpha_1\cdot t \quad (<0)$$

As the data delivers better information about health expenditure rather than health care utilization we directly connect the mortality rate to health expenditures by normalizing the price $p^b=1$. To find fitting values for γ_i and α_i we use an iterative procedure:

(i) We start with a reasonable estimate for the value of health profile in the first stage.

(ii) Now consider the first-order optimality condition

$$(-\mu^1_{b_1}(t,b_1(t)))\psi^1_H(t)=p^b(=1)$$

and the fact, that for our functional specification it holds that

$$\mu^1_{b_1}(t,b_1(t))=\varepsilon(t)\frac{\mu^1(t,b_1(t))}{b_1(t)}$$

(iii) Consequently we can rewrite the optimality condition as:

$$\varepsilon(t)=\frac{b_1(t)}{-\mu^1(t,b_1(t))\psi^1_H(t)}$$

Inserting the general expenditure data $b_1^{Data}(t)$ obtained from the NTA database, the calculated value of health profile and the non-cancer mortality rates from the data, we get an estimated profile for $\varepsilon(t)$, which can then be used to estimate the parameters α_1 and α_2.

(iv) Having found the parameters for $\varepsilon(t)$, we can then simply use the definition of the mortality function to find the parameters $\gamma_i, i\in\{1,2,3,4\}$ by using

$$\ln(g(t))=\ln\left(\frac{\mu^1(t)}{b(t)^{\varepsilon(t)}}\right)$$

and inserting the mortality and health expenditure data together with the estimate for $\varepsilon(t)$ on the right hand side of the equation.

(v) As a next step we undertake several iterations in our general optimization algorithm to get closer to the optimal solution.

(vi) After a certain number of steps we stop and calculate the a-priori value of health (average

value of health in the first and second stage) for the current solution of controls, states and costates and return to step (ii).

As the optimal solution converges during the process, also the calibration for the parameters γ_i and α_i should converge for proper guesses of the initial choices.

A.4 Estimation of cancer hazard rate $\eta(S_1)$

For the estimation of the cancer hazard rate we adjust the total age-specific mortality rate obtained from the human mortality database (University of California, Berkeley (USA) and Max Planck Institute for Demographic Research (Germany) (2020)) for the average age-specific cancer mortality provided in the SEER-database (Surveillance Research Program, National Cancer Institute (2020)). Using this adjusted mortality profile, we can derive the empirical equivalent of the S_1-profile in our model. Using the age-specific cancer incidence rate (again from the SEER database), we wanted to find a functional form, which uses as few free parameters as possible (to avoid overfitting) and still provides a good fit for the relationship between health and cancer incidence. The functional form

$$\widetilde{\eta}(t,S) = \beta_0 \left(1 + \beta_1 \left(\frac{1-S_1}{S_1}\right)^{\beta_2}\right)^{-1}$$

turns out to be an appropriate choice under these prerequisites. Consequently we use a general least square fitting function for non-linear functions to obtain estimates for β_0, β_1, and β_2.

A.5 Estimation of cancer specific mortality rate $\mu^m(s,M)$

The estimation procedure for the parameters of the cancer-specific mortality rate is more involved. Here we face the challenge to find a functional relationship between cancer mortality and the progression of the cancer stock, without having sufficient empirical data for the latter.

Using the duration dependent cancer specific survival data for different ages at diagnosis (4 broad age groups) in the SEER database, we first derive the corresponding empirical cancer mortality rates depending on the time since diagnosis for each of the four age-groups. As the calculated mortality rates represent the average mortality within each of the first 10 years after diagnosis, we assign them to the midpoint of each year, i.e. 6 months, 1.5 years, 2.5 years, etc. Thereby, we obtain the data presented in Table 5.

Recalling that our specified cancer mortality function

$$\mu^m(s,M) = \psi_0 \cdot M \cdot \exp\left\{\psi_1 \cdot \left(\frac{s}{T}\right)^{\psi_2}\right\} \tag{50}$$

only depends on the age of diagnosis s and the cancer stock M, the remaining procedure is less straightforward, as we are missing an empirical counterpart for the development of M.

In a first step we observe, that for each age-group j we are able to approximate to a high

Years since diagnosis	Age-Group 15-39	40-64	65-74	75+
0.5	0.0700	0.1603	0.2309	0.4072
1.5	0.0485	0.0755	0.0830	0.1105
2.5	0.0312	0.0441	0.0478	0.0626
3.5	0.0220	0.0302	0.0325	0.0422
4.5	0.0164	0.0233	0.0244	0.0337
5.5	0.0138	0.0186	0.0207	0.0282
6.5	0.0113	0.0157	0.0183	0.0261
7.5	0.0098	0.0138	0.0165	0.0228
8.5	0.0090	0.0125	0.0145	0.0232
9.5	0.0078	0.0115	0.0145	0.0235

Table 5: Cancer mortality rates depending on duration since diagnosis

degree the mortality profile with a function depending on the time since diagnosis $d := t - s$:

$$\mu^m_{Data}(d, j) \approx N^j(d) := \exp\left\{-a_{1,j} + a_{2,j} \cdot e^{-a_{3,j} \cdot d}\right\}$$

After estimation of $a_{i,j}$ using a non-linear least-square estimation for each age-group separately, we will use the smoothed data

$$\mathcal{N} := \left\{N^j(d) \text{ with } j \in \{1, 2, 3, 4\}\ ,\ d \in \{0, 1, \ldots, 10\}\right\}$$

for the derivation of ψ_0, ψ_1 and ψ_2 in the following.

In the next step we estimate the impact of the age at the diagnosis, i.e. the parameters ψ_1 and ψ_2, by assuming an adjusted version of (50) with the impact of the cancer stock being replaced by an duration-specific parameter ν_d, i.e.

$$\widetilde{\mu^m}(d, s) = \nu_d \cdot \exp\left\{\psi_1 \cdot \left(\frac{s}{T}\right)^{\psi_2}\right\}$$

Since $\mathcal{N}$ only contains data for age-groups j and not each age at diagnosis s, we define the average mortality for each age-group as

$$\widetilde{\mu^m}(d, j) = \frac{1}{|R(j)|} \sum_{s \in R(j)} \nu_d \cdot \exp\left\{\psi_1 \cdot \left(\frac{s}{T}\right)^{\psi_2}\right\}$$

with $R(j)$ being the set of all ages in age-group j and $|R(j)|$ being the number of discrete ages in group j. Again using a non-linear least-square estimation function we obtain estimates for $\nu_0, \ldots, \nu_{10}$ and ψ_1 and ψ_2. Finally using the observation, that we normalized the initial cancer stock $M(s, s) = 1.0$, we can use ν_0 as an estimate for ψ_0.