Health Risk and the Value of Life*

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Abstract

We extend the conventional life-cycle framework for valuing health and longevity improvements to a stochastic setting with multiple health states and apply it to data on mortality, quality of life, labor earnings, and medical spending for adults with different comorbidities. We find that sick adults are willing to pay nearly twice as much per quality-adjusted life-year (QALY) to reduce mortality risk as healthy adults, and that reducing the risk of serious illness is valued similarly to reducing the risk of mild illness. Our results provide a rational explanation for why people oppose a single threshold value for rationing care and why they invest less in prevention than in treatment.

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1 Introduction

The economic analysis of risks to life and health has made enormous contributions to academic discussions and public policy. Economists have used the standard tools of lifecycle consumption theory to propose a transparent framework that measures the value of improvements to both health and longevity (Murphy and Topel, 2006). Economic concepts such as the value of statistical life (VSL) play central roles in discussions surrounding public and private investments in medical care, public safety, environmental hazards, and countless other arenas. However, the conventional life-cycle framework used to study the value of life includes only a single health state, with a preordained mortality rate that depends on age alone. As a result, it is ill-equipped to investigate how VSL varies with a person's health status, and it cannot distinguish between illness and death. Likewise, the conventional model's reliance on a single health state hampers its ability to engage in ongoing policy debates regarding whether and how healthcare reimbursements should vary with health shocks of differing severity.

Against this backdrop, this paper studies how a rational individual's marginal value of reducing mortality and illness risk varies with baseline health status and with the severity of those risks.¹ We extend the conventional life-cycle framework for valuing life to a stochastic setting that accommodates multiple health states with distinct mortality, quality of life, and financial risk profiles. As in prior studies, we define VSL as the willingness to pay to reduce an immediate risk of death. Our extension enables us to derive the value of statistical illness (VSI), which measures the willingness to pay to reduce illness risk and includes VSL as the special case where that risk is death. Using a tractable version of our model with isoelastic preferences, as in Samuelson (1969) and Merton (1969), we then derive a closed-form solution for consumption and investigate the effect of health shocks on the value of life using health and earnings data from the Future Elderly Model (FEM). A key result is that the willingness to pay for life-extension is higher for individuals with shorter baseline life expectancy.

We start by using our general theoretical framework to illuminate how VSL responds to health risk, documenting that VSL can fall or rise after a positive shock to the risk of dying. This insight hinges on two countervailing effects. Consider an individual newly diagnosed with cancer. On the one hand, the attendant reduction in her life expectancy reduces VSL by reducing lifetime utility. On the other hand, this unexpected reduction in longevity increases VSL by encouraging her to spend down her wealth more quickly.

¹In the remainder of the paper, "health" refers to both longevity and health-related quality of life. We use the terms "health risk" and "illness risk" interchangeably. A "unit of health" measures both longevity and health-related quality of life.

To clarify when one effect dominates, we derive a sufficient condition for VSL to rise following an adverse shock to longevity. This condition, which depends on how prudence (Kimball, 1990) compares to the elasticity of intertemporal substitution, is satisfied by many standard utility functions—including isoelastic preferences with a minimum viable consumption level—as well as current estimates of those two parameters.

Our theoretical result that adverse shocks to longevity increase VSL under certain risk preferences holds constant other factors such as wealth. However, a health shock is often accompanied by financial shocks such as job loss or recurring medical expenses. We show that these financial effects can reduce the value of life, implying that the net effect of health shocks on the value of life is ambiguous.

To quantify the overall effect of health shocks on the value of life, we apply a tractable version of our model with isoelastic preferences to individual-level data from a representative cohort of US adults ages 50–80. The data are obtained from the FEM, which provides detailed information on how mortality, quality of life, labor earnings, and medical spending evolve over the life cycle for people over age 50 with different comorbidities. The data underlying the FEM include more comprehensive information than any single national survey and have been widely used to study elderly health and medical spending (e.g., Goldman et al., 2010, 2013; Leaf et al., 2021; Reif et al., 2021). We quantify VSL and VSI for individuals across twenty different health states, each characterized by unique profiles of mortality, quality of life, and financial risks. We normalize VSL and VSI by remaining life expectancy, measured in units of quality-adjusted life-years (QALYs)—a conventional metric of longevity that accounts for the quality of remaining life. Presenting results as VSL per QALY and VSI per QALY makes comparisons easier and enhances their relevance to health policy, as the value of a QALY is often used to determine optimal reimbursement for new health interventions (Garber and Phelps, 1997).

We find that VSL per QALY rises on average by \$70,000 (21%) in the year following an adverse health shock, and by over \$150,000 (45%) following the worst five percent of shocks. Among 70-year-olds, those in the sickest health state are willing to pay 1.9 times more per QALY to reduce mortality risk than healthy people, representing a wide gap in the value of a QALY across health states. These differences become even larger if people are fully insulated from the financial risks of the shocks. We conclude that while financial losses associated with health shocks do reduce the value of additional life-years, the effect is generally not large enough to offset the increase resulting from a reduction in longevity.

We also provide novel estimates of VSI by quantifying the risk-reduction value of different illnesses for a healthy consumer in a fixed health state. We find that VSI rises

only a little with disease severity: a healthy 70-year-old is willing to pay up to \$23,000 (10%) more per QALY to reduce extreme risks such as serious cancer or death than to reduce mild risks such as developing hypertension. This result indicates that although the value of reducing health risk does rise significantly for those in worse health, it varies little with the severity of the risk itself.

We perform several sensitivity analyses to assess the robustness of our results. While the absolute values of our estimates are moderately sensitive to alternative assumptions about consumer risk preferences or the presence of a bequest motive, our two qualitative conclusions—that the value per QALY of reducing a health risk increases dramatically with baseline health but only slightly with severity of the risk—hold up across a number of alternative parameterizations. We also confirm that a health state's mortality risk profile is the key factor driving our numerical results: we obtain the same patterns if we omit quality of life from our model.

Our primary contribution is the development and application of a new, more general life-cycle model of the value of life. While an individual's value of health risk reduction in the conventional model depends only on her age and wealth, in our model this value depends also on her current state of health and on the characteristics of the health risks she faces. Our finding that the value of a QALY is up to 1.9 times higher among the sick than the healthy helps explain puzzles such as why end-of-life spending is high (Zeltzer et al., 2023) and why consumers invest less in prevention than treatment (Weisbrod, 1991; Dranove, 1998; Pryor and Volpp, 2018), without needing to resort to alternative explanations such as market inefficiencies or irrational behaviors (Fang and Wang, 2015; Bai et al., 2021; Newhouse, 2021). Moreover, with its (health) states appropriately redefined, our stochastic framework can also be applied to a number of other distinct questions, such as why societies appear to invest less in preventing pandemics than in mitigating them and how to value insurance in a setting with shocks to health, longevity, and spending (Kowalski, 2015; Lakdawalla et al., 2017; Fang and Shephard, 2019; Atal et al., 2020). Finally, while a stochastic framework can be challenging for practitioners to use in policy analysis, we provide a closed-form solution and an accompanying open-source tool to rapidly calculate VSL and VSI.

Our study has important implications for debates surrounding the allocation of healthcare resources. In many countries, a medical treatment is covered by insurance only if its price does not exceed a "cost-effective" threshold, and conventional theory implies that this threshold should not vary with patient health or with the severity of the illness being treated (Hammitt, 2013; Lucarelli et al., 2022).² However, survey research finds scant

²US law prohibits federal agencies such as Medicare from using conventional cost-effectiveness methods

public support for a constant threshold (Nord et al., 1995; Linley and Hughes, 2013), and several countries that rely on cost-effectiveness to allocate healthcare resources including Norway, Sweden, the Netherlands, and the UK—have defied the underlying economic theory by codifying ad hoc approaches that increase the cost-effectiveness threshold for treatments of severe diseases.³ Our findings that the value of a QALY varies significantly with health status helps explain consumer and voter opposition to the use of a single threshold value when making decisions about health resource allocation, and provides a novel source of support for "top-up" insurance policies, which allow patients who value health more highly to pay incremental prices for more expensive treatments (Einav et al., 2016; Shepard et al., 2020; Lucarelli et al., 2022; Baicker et al., 2023). As a related matter, our theory also provides a novel rationale for firms simultaneously offering more and less generous health insurance plans, which are taken up by the sick and the healthy, respectively.

The economic literature on the value of life includes seminal studies by Arthur (1981), Rosen (1988), Murphy and Topel (2006), and Hall and Jones (2007). Shepard and Zeckhauser (1984) and Ehrlich (2000) note the important role played by insurance markets. Aldy and Smyth (2014) use microsimulation to assess heterogeneity in VSL by race and sex. Córdoba and Ripoll (2016) and Bommier et al. (2021) use Epstein-Zin-Weil preferences to study the implications of non-separable utility on the value of life. The models used in these prior studies include only a single health state for living individuals and focus exclusively on the value of preventing death, setting illness aside. Our study increases the scope and relevance of standard economic theory for understanding health risk by, for example, allowing researchers to compare VSL across health states and to quantify the relative values of reducing the risk of different illnesses.⁴

Our model also reconciles the standard life-cycle framework with results from a distinct literature that uses one-period models to study the value of mortality risk-reduction (Raiffa, 1969; Weinstein et al., 1980; Pratt and Zeckhauser, 1996; Hammitt, 2000). The standard life-cycle model, which assumes complete insurance markets, predicts that reductions in life expectancy will reduce VSL. In contrast, the one-period models predict that an increase in baseline mortality risk will raise VSL when insurance markets are

⁽Lakdawalla and Phelps, 2022).

³Norway and Sweden identify disease severity as a core determinant of treatment value (Defechereux et al., 2012; Persson, 2012). The Netherlands relies on "proportional shortfall" methods that increase reimbursements for treatments of diseases that cause greater relative reductions in quality-adjusted life expectancy (Reckers-Droog et al., 2018). The UK has recently formalized similar ad hoc approaches in its latest health technology evaluation manual (NICE, 2022).

⁴Several empirical papers have already demonstrated the relevance of valuing illness-prevention (Cameron and DeShazo, 2013; Hummels et al., 2016).

incomplete, a result often referred to as the "dead-anyway" effect. We show that in a lifecycle setting with incomplete markets, adverse health shocks can increase *or* decrease VSL, depending on risk preferences.

The remainder of this paper is organized as follows. Section 2 presents the general model, derives key theoretical results, and discusses welfare. Section 3 applies a tractable version of the model to data and shows how VSL varies across people with different health histories and how the value of preventing illness varies with the degree of illness risk. Section 4 concludes.

2 Model

Consider an individual who faces a health risk such as illness or death. We are interested in analyzing the value of a marginal reduction in that risk. Section 2.1 solves a lifetime utility model that accommodates multiple health states with varying degrees of financial, illness, and mortality risk. We then present analytic expressions for the value of statistical life (VSL) and the value of statistical illness (VSI). Section 2.2 provides conditions under which VSL falls or rises following a health shock. Section 2.3 discusses welfare.

Like prior studies on the value of life, we focus throughout this paper on the demand for health and longevity. Quantifying optimal health spending requires additionally modeling the supply of health care (Hall and Jones, 2007). In light of all the institutional differences across health care delivery systems, a wide variety of plausible approaches can be taken to this modeling problem, which we leave to future research.

2.1 The value of health and longevity

Let Y_t denote the consumer's health state at time t. We assume Y_t is a continuous-time Markov chain with finite state space $Y = \{1, 2, ..., n, n + 1\}$, where state $i \in \{1, ..., n\}$ represents different possible health states while alive, and state i = n + 1 represents death. We assume Y_t is right-continuous with left limits (RCLL) and denote the transition rates by:

$$\lambda_{ij}(t) = \lim_{h \to 0} \frac{1}{h} \mathbb{P}[Y_{t+h} = j | Y_t = i], \ j \neq i,$$

$$\lambda_{ii}(t) = -\sum_{j \neq i} \lambda_{ij}(t)$$

For analytical convenience and without meaningful loss of generality, we assume that individuals can transition only to higher-numbered states, i.e., $\lambda_{ij}(t) = 0 \quad \forall j < i.^5$ The probability that a consumer in state *i* at time 0 remains in state *i* at time *t* is then equal to:

$$\tilde{S}(i,t) = \exp\left[-\int_0^t \sum_{j>i} \lambda_{ij}(s) \, ds\right]$$

For expositional purposes, we shall often refer to a transition as a "health shock," "falling ill," or "dying," but our model also accommodates transitions from sick states to healthy states. We denote the stochastic mortality rate at time *t* as:

$$\mu(t) = \sum_{i=1}^{n} \lambda_{i,n+1}(t) \, \mathbf{1} \{ Y_t = i \}$$

where $1{Y_t = i}$ is an indicator variable equal to 1 if the individual is in state *i* at time *t* and 0 otherwise. When the number of states is equal to n = 1, we obtain the setting with deterministic health risk studied in prior literature (e.g., Shepard and Zeckhauser, 1984; Rosen, 1988; Murphy and Topel, 2006). The maximum lifespan of an individual is *T*, and we denote her stochastic probability of surviving until $t \le T$ as:

$$S(t) = \exp\left[-\int_0^t \mu(s)\,ds\right]$$

Let c(t) be consumption at time t, W_0 be baseline wealth, and ρ be the rate of time preference. Health-related quality of life at time t, $q_{Y_t}(t)$, is exogenous and depends on the health state, Y_t . Normalizing the utility of death to zero, the consumer's maximization problem for $Y_0 \in \{1, ..., n\}$ is:

$$V(0, W_0, Y_0) = \max_{c(t)} \mathbb{E}\left[\int_0^T e^{-\rho t} S(t) u(c(t), q_{Y_t}(t)) dt \middle| Y_0, W_0 \right]$$
(1)

Let the state variable W(t) represent current wealth at time t. We assume W(t) evolves

⁵That is, a person can transition from state *i* to *j*, *i* < *j*, but not vice-versa. This restriction does not meaningfully limit the generality of our model because one can always define a new state k > j with properties similar to state *i*.

according to:

$$W(0) = W_0,$$

$$W(t) \ge 0,$$

$$dW(t) = \left(r_{Y_t} W(t) - c(t) \right) dt - W(t) h_{Y_{t-}, Y_t} dY_t$$
(2)

The no-debt constraint, $W(t) \ge 0$, means the consumer cannot borrow. We allow state transitions to affect wealth in two ways: via a state-dependent interest rate, r_{Y_t} , and via a one-time, immediate reduction in wealth of proportion $h_{Y_{t-},Y_t} dY_t$.⁶ These two channels let us model different types of financial consequences, including reduced wage growth, recurring health costs, expenses when falling sick, and expected loss of future income. We assume that $|h_{Y_{t-},Y_t} dY_t| < 1$, so that the fraction of wealth following the transition, $\tilde{h}_{ij} \equiv 1 - h_{ij} (j-i)$, is positive.⁷

The utility function, u(c,q), is time-separable and depends on both consumption and health-related quality of life. We assume throughout that $u(\cdot)$ is strictly increasing and concave in its first argument, and twice continuously differentiable. Hence, we must have W(T) = 0, since it cannot be optimal to have wealth remaining at the maximum possible age. We denote the marginal utility of consumption as $u_c(\cdot)$ and assume that this function diverges to positive infinity as consumption approaches zero, so that optimal consumption is always positive.

Define the consumer's objective function at time *t* as:

$$J(t, w, i) = \mathbb{E}\left[\int_{0}^{T-t} e^{-\rho v} \exp\left\{-\int_{0}^{v} \mu(t+s)ds\right\} u\left(c(t+v), q_{Y_{t+v}}(t+v)\right) dv \middle| Y_{t} = i, W(t) = w\right]$$

Define the optimal value function as:

$$V(t, w, i) = \max_{c(s), s \ge t} \{J(t, w, i)\}$$

subject to the wealth dynamics above and V(t, w, n+1) = 0. Under conventional regularity conditions, if V and its partial derivatives are continuous, then V satisfies the

⁶Equation (2) is a stochastic differential equation driven by the RCLL Markov process Y_t . Here, $dY_t = 0$ for all *t* except for the (finite number of) transition times.

⁷It is straightforward to let r and h also depend on age/time. For ease of exposition we do not do so here, but we allow for this dependence in the numerical model we present in Section 3.

following Hamilton-Jacobi-Bellman (HJB) system of equations:

$$\rho V(t, w, i) = \max_{c(t)} \left\{ u(c(t), q_i(t)) + \frac{\partial V(t, w, i)}{\partial w} [r_i w - c(t)] + \frac{\partial V(t, w, i)}{\partial t} + \sum_{j > i} \lambda_{ij}(t) \Big[V\Big(t, w \, \tilde{h}_{ij}, j\Big) - V(t, w, i) \Big] \right\}, \ i = 1, \dots, n \quad (3)$$

where c(t) = c(t, w, i) is the optimal rate of consumption. The first-order condition in the HJB (3) implies that the marginal utility of wealth in state Y_t at time t is equal to:

$$\frac{\partial V(t, W(t), Y_t)}{\partial W(t)} = u_c(c(t), q_{Y_t}(t))$$
(4)

In order to apply our value of life analysis, we exploit recent advances in the systems and control literature. Parpas and Webster (2013) show that one can reformulate a stochastic finite-horizon optimization problem as a deterministic problem that takes V(t, w, j), $j \neq i$, as exogenous. More precisely, we focus on the path of Y_t that begins in state *i* and remains in state *i* until time *T*. We denote optimal consumption and wealth in that path by $c_i(t)$ and $W_i(t)$, respectively.⁸ A key advantage of this method is that it allows us to apply the standard deterministic Pontryagin maximum principle and derive analytic expressions.

Lemma 1. Consider the following deterministic optimization problem for $Y_0 = i$ and $W(0) = W_0$:

$$V(0, W_0, i) = \max_{c_i(t)} \left[\int_0^T e^{-\rho t} \tilde{S}(i, t) \left(u(c_i(t), q_i(t)) + \sum_{j > i} \lambda_{ij}(t) V(t, W_i(t) \tilde{h}_{ij}, j) \right) dt \right]$$
(5)

subject to:

$$W_i(0) = W_0,$$

$$W_i(t) \ge 0,$$

$$\frac{\partial W_i(t)}{\partial t} = r_i W_i(t) - c_i(t)$$

⁸Consumption, c(t), is a stochastic process. We occasionally denote it as $c(t, W(t), Y_t)$ to emphasize that it depends on the states $(t, W(t), Y_t)$. When we reformulate our stochastic problem as a deterministic problem and focus on a single path $Y_t = i$, consumption is no longer stochastic because there is no uncertainty in the development of health states. We emphasize this point in our notation here by writing consumption as $c_i(t)$, and wealth as $W_i(t)$.

where V(t, w, j), $j \neq i$, are taken as exogenous. Then the optimal value function, $V(t, W_i(t), i)$, satisfies the HJB equation given by (3), for all $i \in \{1, ..., n\}$.

Proof. See Appendix A

Because the value function V(t, w, i) corresponding to (5) satisfies the HJB equation given by (3), it must also be equal to the consumer's optimal value function (Bertsekas, 2005, Proposition 3.2.1). The present value Hamiltonian corresponding to (5) is:

$$H\left(W_{i}(t), c_{i}(t), p_{t}^{(i)}\right) = e^{-\rho t}\tilde{S}(i, t)\left(u(c_{i}(t), q_{i}(t)) + \sum_{j>i}\lambda_{ij}(t)V\left(t, W_{i}(t)\tilde{h}_{ij}, j\right)\right) + p_{t}^{(i)}[r_{i}W_{i}(t) - c_{i}(t)]$$

where $p_t^{(i)}$ is the costate variable for state *i*. The necessary costate equation is:

$$\dot{p}_{t}^{(i)} = -\frac{\partial H}{\partial W_{i}(t)} = -p_{t}^{(i)}r_{i} - e^{-\rho t}\tilde{S}(i,t)\sum_{j>i}\lambda_{ij}(t)\tilde{h}_{ij}\frac{\partial V\left(t,W_{i}(t)\tilde{h}_{ij},j\right)}{\partial w}$$
(6)

The solution to the costate equation can be obtained using the variation of the constant method:

$$p_t^{(i)} = \left[\int_t^T e^{(r_i - \rho)s} \tilde{S}(i, s) \sum_{j > i} \lambda_{ij}(s) \tilde{h}_{ij} \frac{\partial V\left(s, W_i(s) \tilde{h}_{ij}, j\right)}{\partial w} ds\right] e^{-r_i t} + \theta^{(i)} e^{-r_i t}$$

where $\theta^{(i)} > 0$ is a constant. The necessary first-order condition for consumption is:

$$p_t^{(i)} = e^{-\rho t} \tilde{S}(i, t) u_c(c_i(t), q_i(t))$$
(7)

where the marginal utility of wealth at time t = 0 is $\frac{\partial V(0, W_0, i)}{\partial W_0} = p_0^{(i)} = u_c(c_i(0), q_i(0))$. Since the Hamiltonian is concave in $c_i(t)$ and $W_i(t)$, the necessary conditions for optimality are also sufficient (Seierstad and Sydsaeter, 1977).

To analyze the value of health and longevity, we follow Rosen (1988). Let $\delta_{ij}(t)$ be a perturbation on the transition rate, $\lambda_{ij}(t)$, $0 \le t \le T$, where $\sum_{j>i} \int_0^T \delta_{ij}(t) dt = 1$. The impact of a small (ε) perturbation on the likelihood of exiting state *i* is:

$$\tilde{S}^{\varepsilon}(i,t) = \exp\left[-\int_{0}^{t} \sum_{j>i} \left(\lambda_{ij}(s) - \varepsilon \delta_{ij}(s)\right) ds\right], \text{ where } \varepsilon > 0$$
(8)

The marginal value of preventing illness or death is equal to $\frac{\partial V/\partial \varepsilon}{\partial V/\partial w}\Big|_{\varepsilon=0}$, the marginal rate

of substitution between longer life and wealth. The next lemma provides the numerator of this marginal value expression.

Lemma 2. The marginal utility of preventing illness or death in state i is given by:

$$\frac{\partial V(0, W_0, i)}{\partial \varepsilon} \bigg|_{\varepsilon=0} = \int_0^T e^{-\rho t} \tilde{S}(i, t) \left[\left(\int_0^t \sum_{j>i} \delta_{ij}(s) \, ds \right) \left(u(c_i(t), q_i(t)) + \sum_{j>i} \lambda_{ij}(t) V\left(t, W_i(t) \tilde{h}_{ij}, j\right) \right) - \sum_{j>i} \delta_{ij}(t) V\left(t, W_i(t) \tilde{h}_{ij}, j\right) \right] dt$$

Proof. See Appendix A

Lemma 2 pertains to a marginal reduction in transition rates for all states and times. Consider as a special case perturbing only $\lambda_{i,n+1}(t)$, the mortality rate in state *i*, and set the perturbation $\delta(\cdot)$ in Equation (8) equal to the Dirac delta function, so that the mortality rate is perturbed at t = 0 and remains unaffected otherwise (Rosen, 1988). This perturbation yields an expression commonly known as the value of statistical life (VSL).

Proposition 3. Set $\delta_{ij}(t) = 0 \quad \forall j < n+1$ in the marginal utility expression given in Lemma 2 and let $\delta_{i,n+1}(t)$ equal the Dirac delta function. Dividing by the marginal utility of wealth yields:

$$VSL(i) = \mathbb{E}\left[\int_0^T e^{-\rho t} S(t) \frac{u(c(t), q_{Y_t}(t))}{u_c(c(0), q_{Y_0}(0))} dt \middle| Y_0 = i, W(0) = W_0\right] = \frac{V(0, W_0, i)}{u_c(c_i(0), q_i(0))}$$
(9)

Proof. See Appendix A

VSL is the value of a marginal reduction in the risk of death in the current period. Put differently, it is the amount that a large group of individuals are collectively willing to pay to eliminate a current risk that is expected to kill one of them, where the identity of the decedent is unknown *ex ante*. Proposition 3 shows that VSL is proportional to expected lifetime utility, and inversely proportional to the marginal utility of consumption.

We can also value a marginal reduction in the risk of falling ill. As before, it is helpful to choose the Dirac delta function for $\delta(t)$, so that the transition rates are perturbed at t = 0 only. Consider a reduction in the transition rate for a single alternative state, $j \le n+1$, so that $\delta_{ik}(t) = 0 \forall k \ne j$. Dividing the result by the marginal utility of wealth then yields what we term the "value of statistical illness" (VSI):

$$VSI(i,j) = \frac{V(0, W_0, i) - V(0, W_0 \tilde{h}_{ij}, j)}{u_c(c_i(0), q_i(0))}$$
(10)

The interpretation of VSI is analogous to VSL: it is the amount that a large group of individuals are collectively willing to pay in order to eliminate a current disease risk that is expected to befall one of them, where the identity of the sick person is unknown *ex ante*. If health state *j* corresponds to death, so that V(0, w, j) = 0, then VSI(i, j) = VSL(i). Thus, VSI is a generalization of VSL.

The values of statistical life and illness depend on how substitutable consumption is at different ages and states. Intuitively, if present consumption is a good substitute for future consumption, then living a longer life is less valuable. Define the elasticity of intertemporal substitution, σ , as the inverse of relative risk aversion:

$$\frac{1}{\sigma} \equiv -\frac{u_{cc} c}{u_c}$$

In addition, define the elasticity of quality of life with respect to the marginal utility of consumption as:

$$\eta \equiv \frac{u_{cq} \, q}{u_c}$$

When η is positive, the marginal utility of consumption is higher in healthier states, and vice-versa. Taking logarithms of Equation (7), differentiating with respect to *t*, plugging in the result for the costate equation and its solution, and rearranging yields an expression for the life-cycle profile of consumption:

$$\frac{\dot{c}_i}{c_i} = \sigma \left(r_i - \rho \right) + \sigma \eta \frac{\dot{q}_i}{q_i} - \sigma \lambda_{i,n+1}(t) - \sigma \sum_{j=i+1}^n \lambda_{ij}(t) \left[1 - \tilde{h}_{ij} \frac{u_c \left(c \left(t, W_i(t) \tilde{h}_{ij}, j \right), q_j(t) \right)}{u_c \left(c \left(t, W_i(t), i \right), q_i(t) \right)} \right]$$
(11)

The first two terms in Equation (11) relate the growth rate of consumption to the consumer rate of time preference and to life-cycle changes in the quality of life. The third term shows that consumption growth is a declining function of the current mortality rate, $\lambda_{i,n+1}(t)$. Because the consumer cannot purchase annuities to insure against her uncertain lifetime, higher rates of mortality depress the rate of consumption growth over the life-cycle (Yaari, 1965).

The fourth term in Equation (11)—which is absent from the conventional deterministic setting—accounts for the possibility that the consumer might transition to a different health state in the future. The possibility of transitioning to a state with low marginal utility of consumption shifts life-cycle consumption earlier still, whereas transitioning to a state with high marginal utility shifts consumption to later in the life-cycle. The effect on current consumption of potential financial shocks, as captured by \tilde{h}_{ij} , is ambiguous. On the one hand, the possibility of losing wealth in the future increases the incentive to consume today. On the other hand, the possibility of low future wealth—and thus high marginal utility of consumption—increases the incentive to save. The net effect depends on the curvature of the utility function.⁹

Equation (11) describes consumption dynamics conditional on the individual's health state *i*. It is not readily apparent from (11) whether modeling health as stochastic causes consumption to shift forward, *on average* across all states, relative to modeling health as deterministic. We confirmed in numerical exercises that modeling health as stochastic has an ambiguous effect on consumption (and VSL), even when holding quality of life constant across states and time.¹⁰

2.2 The effect of health shocks on VSL

In our model, an adverse health shock can reduce a person's longevity and quality of life; increase her recurring health costs, as captured by the health-related interest rate, r_{Y_t} ; and produce an upfront financial shock, as captured by h_{Y_{t-},Y_t} . This section isolates each of these effects in a series of simple, transparent two-state settings and shows that they can have opposing effects on VSL.¹¹ To quantify their net effect, we turn to a numerical model in Section 3.

We first consider a setting where a transition reduces wealth but otherwise has no effect. Because the willingness-to-pay for life extension increases with wealth, this transition reduces VSL.

Proposition 4. Assume utility is positive. Let there be n = 2 states with identical healthcare spending and quality of life, so that $r_1 = r_2$ and $q_1(s) = q_2(s) \forall s$, and identical probabilities of dying, so that $\lambda_{13}(s) = \lambda_{23}(s) \forall s$. Assume that transitioning from state 1 to state 2 is associated with a one-time financial shock, $h_{12} > 0$. Suppose the consumer transitions from state 1 to state 2 at time t, and that $\lambda_{12}(\tau) = 0 \forall \tau > t$. Then VSL(1,t) > VSL(2,t).

Proof. See Appendix A

⁹This result reflects the classic tradeoff between income and substitution effects, as applied to intertemporal consumption (Merton, 1969). These opposing effects exactly offset each other in the special case where the elasticity of intertemporal substitution, σ , equals 1.

¹⁰Modeling health as stochastic increases lifetime utility because a stochastic environment allows the consumer to adjust consumption after a health shock. Put differently, a deterministic model is equivalent to a stochastic model where the consumer is forced to keep consumption constant across states.

¹¹While we focus here on how VSL changes following a state transition, our results have similar implications for VSI. In a two-state model, the only relevant values to compare are VSI(1,3) = VSL(1) and VSI(2,3) = VSL(2). In a model with more than two states, one could also compare the values of preventing illness.

Next, we consider a transition that reduces the health-related interest rate, r_{Y_t} . Because this transition reduces future wealth, it will reduce expected lifetime utility. At the same time, a reduction in the interest rate may encourage individuals to increase current consumption, which raises their willingness to pay for life-extension, all else equal. The net effect depends on the elasticity of intertemporal substitution, σ . As we discuss later in this section, prior studies generally assume that $\sigma < 1$. Our next proposition shows that this assumption is a sufficient condition for VSL to decrease following a reduction in r_{Y_t} .

Proposition 5. Assume utility is positive and satisfies the condition $\sigma < 1$. Let there be n = 2 states with identical quality of life and identical probabilities of dying, so that $q_1(s) = q_2(s)$ and $\lambda_{13}(s) = \lambda_{23}(s) \forall s$, and assume that there are no one-time financial shocks when transitioning, so that $h_{12} = 0$. Assume that transitioning from state 1 to state 2 is associated with a decrease in the health-related interest rate such that $r_1 > r_2$. Suppose the consumer transitions from state 1 to state 2 at time t, and that $\lambda_{12}(\tau) = 0 \forall \tau > t$. Then VSL(1,t) > VSL(2,t).

Proof. See Appendix A

Finally, we consider transitions that affect quality of life or longevity. An adverse shock to quality of life will lower lifetime utility, $V(\cdot)$. However, inspection of Equation (9) shows that the shock's effect on VSL will also depend on the relationship between quality of life and the marginal utility of consumption, a phenomenon often referred to as "health state dependence." Quantifying this relationship is a longstanding topic in health economics, but there is still no consensus regarding its sign or magnitude.¹² Thus, the effect of a quality of life shock on VSL cannot be signed without making further assumptions.

The effect on VSL of a change in longevity will depend on consumer preferences and expectations of future mortality. Intuitively, adverse longevity shocks have two countervailing effects on VSL. On the one hand, a shorter lifespan reduces the lifetime utility of life-extension. On the other hand, a shorter lifespan increases current consumption, which lowers marginal utility and thus increases the willingness to pay for health and longevity. The net effect will depend on the curvature of the utility function relative to the curvature of the marginal utility function.

¹²Viscusi and Evans (1990), Sloan et al. (1998), and Finkelstein et al. (2013) find evidence of negative state dependence, meaning that the marginal utility of consumption falls when health deteriorates. In contrast, Lillard and Weiss (1997) and Edwards (2008) find evidence of positive state dependence. Evans and Viscusi (1991) find no evidence of state dependence. Murphy and Topel (2006) assume negative state dependence when performing their empirical exercises, while Hall and Jones (2007) assume state independence.

We first show that consumption increases when transitioning to a state where current and future mortality are high, holding wealth constant. If wealth falls following the transition, then the proposition may not hold, depending on the size of the wealth shock. However, in our quantitative analysis—where wealth can fall after a health shock because of medical spending and income loss—we find that non-medical consumption still generally rises. This result is consistent with Smith (1999), who finds that the reduction in wealth following an adverse health shock is larger than its combined effects on out-of-pocket medical spending and income.

Proposition 6. Let there be n = 2 states with identical constant quality of life and healthrelated interest rates, so that $q_1(s) = q_2(s) = q$ and $r_1 = r_2 \forall s$, and no financial shocks upon transition, so that $h_{1,2} = 0$. Assume that the transition rates $\lambda_{12}(s)$ are uniformly bounded (finite), and that the mortality rate is uniformly higher in state 2: $\lambda_{13}(s) < \lambda_{23}(s) \forall s$. Suppose the consumer transitions from state 1 to state 2 at time t. Then $c_1(t, W(t), 1) < c_2(t, W(t), 2)$.

Proof. See Appendix A

To analyze the effect of this transition on VSL, we focus on the case where $\dot{c}_i/c_i \leq 0$, so that consumption does not grow for people who stay in the same health state. Empirical evidence suggests this case accurately describes the typical consumer nearing retirement.¹³ According to Equation (11), and under the assumptions of Proposition 6, a sufficient condition for a declining consumption profile is $r_i \leq \rho$.

Our analysis will compare a persistently healthy individual to someone who suffers an adverse shock to life expectancy but is otherwise identical. To make headway we must introduce the notion of prudence. The elasticity of intertemporal substitution, σ , measures utility curvature (i.e., the change in its slope). Prudence, π , is the analogous measure for the curvature of marginal utility (Kimball, 1990):

$$\pi \equiv -\frac{c \, u_{ccc}(\cdot)}{u_{cc}(\cdot)}$$

It will also be convenient to define the elasticity of the flow utility function:

$$\epsilon \equiv \frac{c \, u_c(\cdot)}{u(\cdot)}$$

¹³A typical consumption profile is constrained by low income at early ages, increasing during middle ages when income is high, and then declines during retirement until consumption equals the consumer's pension. This inverted U-shape for the age profile of consumption has been widely documented across different countries and goods (Carroll and Summers, 1991; Banks et al., 1998; Fernandez-Villaverde and Krueger, 2007).

The utility elasticity, ϵ , is positive when utility is positive. Positive utility ensures wellbehaved preferences, and is often enforced by adding a constant to the utility function. Although adding a constant to the utility function does not affect the solution to the consumer's maximization problem, this constant matters for the value of life.¹⁴

The following proposition provides sufficient conditions for VSL to rise following an adverse shock to longevity.

Proposition 7. Consider a two-state setting with assumptions set out in Proposition 6. Assume that $r_1 = r_2 \le \rho$, and that utility is positive and satisfies the condition:

$$\pi < \frac{2}{\sigma} + \epsilon \tag{12}$$

Suppose that the consumer transitions from state 1 to state 2 at time t, and that $\lambda_{12}(\tau) = 0 \forall \tau > t$. Then, VSL(1,t) < VSL(2,t).

Proof. See Appendix A

Proposition 7 shows that the effect of longevity shocks on VSL depends on both prudence and the elasticity of intertemporal substitution. Consumers with inelastic demand for current consumption (low σ) prefer to smooth consumption over time because consumption expenditures at different ages are poor substitutes. They therefore have a high willingness to pay for life-extension and, all else equal, are more likely to exhibit a rise in VSL following an adverse longevity shock than consumers with more elastic demand. Likewise, consumers with low levels of prudence, π , have marginal utility that decreases rapidly with consumption and produces a high willingness to pay for life-extension following a shock that increases consumption.

Prior studies on the value of life generally assume that 0.5 to 0.8 is a reasonable range for the value of σ (Murphy and Topel, 2006; Hall and Jones, 2007), and recent empirical studies suggest that π is about 2 (Noussair et al., 2013; Christelis et al., 2020). Under these parameterizations, condition (12) will hold whenever utility is positive. Condition (12) is always satisfied by isoelastic utility with a minimum viable consumption level, provided that utility is positive.¹⁵ That said, the condition is not innocuous: one can easily find linear combinations of isoelastic and polynomial utility functions where VSL declines following an illness.

¹⁴Rosen (1988) was the first to point out that the level of utility is an important determinant of the value of life. See also additional discussion on this point in Hall and Jones (2007) and Córdoba and Ripoll (2016).

¹⁵Isoelastic utility with a non-zero constant term, exemplified by Equation (15), is commonly used to quantify the value of life (Murphy and Topel, 2006; Hall and Jones, 2007; Finkelstein et al., 2024).

Thus, VSL can in general rise or fall following an increase in baseline mortality risk. In static models commonly used in prior studies, however, VSL can only rise with baseline risk (Weinstein et al., 1980; Pratt and Zeckhauser, 1996; Hammitt, 2000). This discrepancy arises because these prior studies focus on a one-period setting with two states, alive and dead. In that context, if the marginal utility of consumption is lower in the dead state, then an increase in baseline mortality risk must lower the expected marginal utility of consumption and thus raise the willingness to pay for survival (the "dead-anyway" effect).¹⁶ Proposition 6 confirms that an increase in the risk of death also reduces marginal utility in our dynamic context. However, unlike in the highly simplified static setting, the resulting effect on VSL is ambiguous because of an offsetting decrease in lifetime utility.

Finally, we note that Proposition 7 applies only to the moment of transition. For individuals who survive long enough, an adverse shock to longevity will eventually lower VSL, relative to no shock, because it accelerates the depletion of their wealth over time. This depletion will be further exacerbated if the health shock is accompanied by increased medical expenses.

The value of a health unit

Our results on the effect of health shocks on VSL extend naturally to the value of a health unit, like a life-year or quality-adjusted life-year (QALY). Let $D_i(t)$ denote some measure of remaining lifetime health for an individual in state *i* at time *t*. For example, $D_i(t)$ could be simple life expectancy, quality-adjusted life expectancy, or generalized risk- and quality-adjusted life expectancy (Lakdawalla and Phelps, 2020, 2023). We assume this measure is non-negative, equals 0 only when dead, and is independent of consumption, but otherwise impose no restrictions on its form. When VSL rises following a transition from state *i* to some state *j* with lower health (e.g., as in Proposition 7), the value per unit of health must naturally rise as well:

$$\frac{VSL(i,t)}{D_i(t)} < \frac{VSL(j,t)}{D_j(t)}$$
(13)

This result contrasts with traditional cost-effectiveness analysis, where a QALY is equally valuable regardless of baseline health (Drummond et al., 2015, Chapter 5). In our more general framework, however, a constant value for QALYs emerges only under the assumption of constant consumption, a condition that frequently does not hold (Ble-

¹⁶Let expected utility be equal to EU = p u(0, c) + (1 - p) u(1, c), where $p \in (0, 1)$ is the probability of death and the states $\{0, 1\}$ represent death and life, respectively. The willingness to pay for a marginal reduction in the probability of dying is given by $VSL = \frac{u(1,c)-u(0,c)}{p u_c(0,c)+(1-p)u_c(1,c)}$, which increases with p if $u_c(1,c) > u_c(0,c)$.

ichrodt and Quiggin, 1999). One exception is the special case where financial markets are complete, the rate of time preference matches the interest rate, and quality of life is constant.

The result also sheds light on the value of prevention versus treatment. We define a "preventive" intervention as one that improves health for the healthy, and a "treatment" intervention as one that improves health for the sick. This distinction is qualitative, with interventions classified as "more preventive" if they target health in healthier states, and vice versa. Indeed, the prevention versus treatment distinction is fluid in the real-world, because most disease-modifying "treatments" also *prevent* health deterioration or death. For example, hypertension "treatments" not only manage blood pressure but also help prevent heart disease, stroke, and mortality. Nonetheless, our definitions align with the common notion that vaccines are preventive, whereas drugs administered after an illness has occurred are considered treatments. Within this conceptual framework, our result that VSL may rise after a health shock (Proposition 7) implies that "more preventive" interventions produce less value per health unit gained. This insight helps explain lower demand for preventive investments, even when prevention and treatment produce the same expected health gain, and why preventive interventions frequently fail to improve health (Jones et al., 2019).

While an increase in VSL following an adverse health shock is sufficient to raise the value of a health unit, it is not a necessary condition. Our quantitative analysis reveals that even when VSL decreases after a health shock, the value of a health unit, such as a QALY, typically still increases.

It is worth underscoring that VSL measures the value of reducing an immediate risk to life, not a future risk to life. Notable examples of preventive investments that reduce immediate risks include seatbelt usage, firearm safety measures, and vaccines against rapidly fatal illnesses like the measles or COVID-19. While our model can evaluate reductions in future mortality risks (see Lemma 2), this analysis extends beyond the traditional scope of VSL. We therefore leave this extension for future work.

2.3 Welfare

This paper studies the willingness to pay for health and longevity, shedding light on puzzles such as why sick individuals are often willing to pay more to reduce mortality risk than those in good health. Policymakers, however, frequently face the challenge of allocating limited resources across different people. Who should receive access to a scarce vaccine during a pandemic? Should fixed resources be directed to the elderly or

the young, the sick or the poor?

In such contexts, economists frequently rely on comparisons of aggregate social surplus, that is, the aggregate sum of willingness to pay. For example, Murphy and Topel (2006) employ this approach in the framework of the standard life-cycle VSL model. Garber and Phelps (1997) rely on it to develop the theory of cost-effectiveness for health interventions. Einav et al. (2010) use it to study the welfare effects of health insurance. Industrial organization economists use it, in the form of deadweight loss comparisons, to evaluate the welfare consequences of market power (Martin, 2019).

While popular among applied economists and policymakers, the aggregate surplus approach has been criticized by welfare theorists for several reasons (Boadway, 1974; Blackorby and Donaldson, 1990). Equity concerns arise because each dollar of surplus is weighted equally, regardless of differences in wealth or income across people; this implicitly places more weight on the utility of wealthier individuals. Aggregation can also produce intransitive rankings of alternative allocations. Heterogeneity in marginal utility across consumers can break the link between growth in aggregate surplus and increases in utility (Martin, 2019). These points matter little when valuing the prevention of different illnesses, which can be accomplished from the perspective of a single individual, but it does suggest a need for caution when making welfare inferences across individuals inhabiting different health states.

One alternative solution is to aggregate utilities rather than monetized surplus, but debate persists about how to aggregate in situations involving risk (Fleurbaey, 2010). In a foundational study, Harsanyi (1955) shows that a social welfare function satisfying both rationality and the Pareto principle must be a weighted sum of ex ante individual utilities. However, this utilitarian approach ignores ex post distributional concerns (Diamond, 1967). As a result, one cannot simultaneously satisfy both rationality and the Pareto principles. Diamond (1967) advocates minimizing ex ante inequality, but this violates rationality. Adler and Sanchirico (2006) advocate minimizing ex post inequality, but this violates the Pareto principle. In the specific context of VSL, Pratt and Zeckhauser (1996) advocate maximizing ex ante utility, but this ignores ex post equity concerns in light of Diamond's result. We do not aim to resolve this long-standing debate in welfare economics, but instead note that our stochastic model can be incorporated into these different welfare frameworks as desired.

3 Quantitative Analysis

This section quantifies the value of health improvements. While our model provides useful insights on its own, some of our theoretical results require imposing restrictions on the consumer's setting, such as holding wealth constant. Therefore, we complement our theory with quantitative analysis calculating the value of health improvements for a consumer with standard preferences and whose mortality, quality of life, labor earnings, and medical spending can vary across 20 different health states.¹⁷ We calculate both VSI and VSL but focus more on their normalized values, VSI per QALY and VSL per QALY, which are more easily compared and of great relevance to healthcare reimbursement and pricing (Garber and Phelps, 1997). All of our data and code are publicly available online.¹⁸

3.1 Framework

We employ a discrete time analogue of the model from Section 2. There are *n* health states (excluding death). Denote the transition probabilities between health states by:

$$p_{ij}(t) = \mathbb{P}\left[Y_{t+1} = j | Y_t = i \right]$$

The mortality rate at time t, d(t), depends on the individual's health state:

$$d(t) = \sum_{j=1}^{n} \overline{d}_{j}(t) \mathbf{1} \{Y_{t} = j\}$$

where $\{\overline{d}_j(t)\}\$ are given and $\mathbf{1}\{Y_t = j\}\$ is an indicator equal to 1 if the individual is in state j at time t and 0 otherwise.¹⁹ The maximum lifespan of a consumer is T, so d(T) = 1. We denote the stochastic probability of surviving from time t to time $s \leq T$ as $S_t(s)$, where:

$$S_t(t) = 1,$$

 $S_t(s) = S_t(s-1)(1 - d(s-1)), s > t$

Let c(t) and W(t) denote non-medical consumption and wealth in period *t*, respectively. Quality of life at time *t*, $q_{Y_t}(t)$, depends on the individual's health state, Y_t . Let ρ denote

¹⁷Our quantitative framework is related to a number of papers that study the savings behavior of the elderly (Kotlikoff, 1988; Palumbo, 1999; De Nardi et al., 2010). These prior studies allow health to affect wealth accumulation by including two or three different health states in the model.

¹⁸They are available at: https://julianreif.com/research/reif.wp.healthrisk.replication.zip.

¹⁹Because our mortality data are distinct from our health state transition data, we denote the probability of dying in state *i* as $\overline{d}_i(t)$ rather than $p_{i,n+1}(t)$, which differs slightly from the notation used in Section 2.

the rate of time preference. We measure health in state i and time t using discounted quality-adjusted life expectancy, defined as:

$$D_{i}(t) = \mathbb{E}\left[\sum_{j=t}^{T} e^{-\rho(j-t)} q_{Y_{j}}(j) S_{t}(j) \middle| Y_{t} = i\right]$$
(14)

We assume annuity markets are absent. This simplification allows us to calculate the value of life using an analytical solution to the consumer's problem. It is possible to incorporate partial annuitization in this setting. However, generalization requires numerical optimization, which may necessitate limiting the number of health states included in the model. In our sensitivity analysis, we model the effects of a bequest motive and of decreasing the substitutability of consumption over time, both of which—similar to annuitization—reduce consumption at earlier ages.

The consumer's maximization problem is:

$$\max_{c(t)} \mathbb{E}\left[\sum_{t=0}^{T} e^{-\rho t} S_0(t) u(c(t), q_{Y_t}(t)) \middle| Y_0, W_0\right]$$

subject to:

$$W(0) = W_0,$$

 $W(t) \ge 0,$
 $W(t+1) = (W(t) - c(t))e^{r(t,Y_t,Y_{t+1})}$

We account for out-of-pocket medical spending and other health-related financial shocks by allowing the individual's effective interest rate, $r(t, Y_t, Y_{t+1})$, to depend on her health states at the beginning and end of the period, Y_t and Y_{t+1} , respectively. Our baseline model sets $r(t, Y_t, Y_{t+1}) = r + \ln[1 - m(t, Y_t) + f(t, Y_t, Y_{t+1})]$, where r is the rate of interest, $m(t, Y_t)$ is the average share of an individual's wealth spent on medical and long-term care in health state Y_t at time t, and $f(t, Y_t, Y_{t+1})$ is the average change in the expected present value of future income when transitioning from health state Y_t to Y_{t+1} between time t and t + 1, expressed as a share of current wealth.²⁰ Instead of deducting financial losses from wealth directly, we treat them as modifying the interest rate. This approach allows us to capture the financial effects of changes in health, while preserving the closed-

²⁰We calculate $m(t, Y_t)$ by dividing out-of-pocket spending in health state Y_t at time t by average wealth at time t. We calculate $f(t, Y_t, Y_{t+1})$ by dividing the change in the expected present value of future income between t and t + 1 by wealth at time t.

form solution that facilitates quantitative analysis, similar to Krebs (2003). We assume throughout that $r = \rho = 0.03$ (Siegel, 1992; Moore and Viscusi, 1990).

Finally, we assume that utility takes the following isoelastic form:

$$u(c,q) = q\left(\frac{c^{1-\gamma} - \underline{c}^{1-\gamma}}{1-\gamma}\right) \tag{15}$$

The quality of life measure has non-negative values of $q \le 1$, where q = 1 indexes perfect health. Utility is positive when non-medical consumption, $c \ge 0$, exceeds the minimum viable level, \underline{c} . As discussed previously, there is no consensus regarding the sign or magnitude of health state dependence, $u_{cq}(\cdot)$. Here, we assume a multiplicative relationship where the marginal utility of non-medical consumption increases with the health-related quality of life (negative state dependence), which is consistent with the methods used to measure quality-adjusted life-years (QALYs) in our data, and with prior theoretical literature on VSL (Garber and Phelps, 1997; Bleichrodt and Quiggin, 1999; Murphy and Topel, 2006). The utility function (15) satisfies condition (12) from Proposition 7 when it is positive, specifically when consumption exceeds the minimum viable level, \underline{c} .

The value function for the consumer's maximization problem is defined as:

$$V(t, w, i) = \max_{c(s)} \mathbb{E}\left[\sum_{s=t}^{T} e^{-\rho(s-t)} S_t(s) u(c(s), q_i(s)) \middle| Y_t = i, W(t) = w\right]$$

We reformulate this optimization problem as a recursive Bellman equation:

$$V(t, w, i) = \max_{c(t)} \left[u(c(t), q_i(t)) + \frac{1 - \overline{d}_i(t)}{e^{\rho}} \sum_{j=1}^{N} p_{ij}(t) V(t+1, (w-c(t)) e^{r(t, i, j)}, j) \right]$$

We solve for consumption analytically, following approaches similar to those of Samuelson (1969), Merton (1969), Krebs (2003), and others. We then use the formulas derived in Section 2 to calculate the value of life (see Appendix C for details).

The elasticity of intertemporal substitution, $\sigma = 1/\gamma$, is a key determinant of the value of life. Dating back to Hall (1988), standard estimates of σ are around 0.5 ($\gamma = 2$), or even lower (see Attanasio and Weber (2010) for a literature review). Attanasio and Weber (1993) point out that estimates derived from aggregate data are downward biased and suggest a higher value of $\sigma = 0.8$ ($\gamma = 1.25$) instead. At the opposite end of the spectrum, the macro-finance literature on long-run risk identifies higher σ values in order to match moments of consumption growth and volatility. For instance, Bansal and Yaron (2004) calibrate a model with $\sigma = 1.5$ ($\gamma = 0.66$). Our primary specification adopts $\sigma = 0.8$ ($\gamma =$ 1.25), a value situated midway within this range of estimates (see Table 1). Additionally, we set the minimum viable consumption level, <u>c</u>, equal to \$5,000, consistent with the parameterization used in Murphy and Topel (2006).

The previous paragraph underscores the considerable uncertainty among economists concerning the appropriate values of key parameters in our model, or, equivalently, the potential heterogeneity in these parameters across the population. The goal of the subsequent analyses is to quantify the economic significance of our insights by applying our model to real-world data using reasonable parameterizations. To investigate the sensitivity of our results to the parameterization of our utility function, we consider specifications with alternative assumptions regarding the elasticity of intertemporal substitution, $\sigma = 1/\gamma$. We also consider an alternative specification that includes a bequest motive. Rather than setting the utility of death to zero, our bequest motive specification follows Fischer (1973) and sets it equal to u(W(t) - c(t), b(t)), where $u(\cdot)$ takes the form given in (15), W(t) - c(t) is wealth at death, and the parameter b(t) governs the strength of the bequest motive. We conservatively set b(t) = 1.2, the largest value considered in Fischer (1973), for all t. The baseline parameters in our simulation are summarized in Table 1.

3.2 Data

We obtain individual-level data on mortality, disease incidence, quality of life, labor earnings, and out-of-pocket medical spending from the Future Elderly Model (FEM), a widely published microsimulation model that combines nationally representative information from the Health and Retirement Study (HRS), the Medical Expenditure Panel Survey (MEPS), the Panel Study of Income Dynamics, and the National Health Interview Survey. The FEM provides a uniquely rich set of information about older Americans. For instance, while the HRS provides detailed data on health and wealth, it lacks survey questions that would allow us to calculate quality of life using standard survey instruments. To solve this problem, the FEM weaves together validated quality of life estimates from the MEPS and maps them to the HRS using variables common to both databases.

The FEM, which has been released into the public domain, produces estimates for individuals ages 50–100 with different comorbid conditions (see Appendix B). It accounts for six different chronic conditions (cancer, diabetes, heart disease, hypertension, chronic lung disease, and stroke) and six different impaired activities of daily living (bathing, eating, dressing, walking, getting into or out of bed, and using the toilet). We divide the health space within the FEM into n = 20 states. Each state corresponds to the number (0, 1, 2, 3 or more) of impaired activities of daily living (ADL) and the number (0, 1,

2, 3, 4 or more) of chronic conditions, for a total of $4 \times 5 = 20$ health states. Health states are indexed first by number of ADLs and then by number of chronic diseases, so that state 1 corresponds to 0 ADLs and 0 chronic conditions, state 2 corresponds to 0 ADLs and 1 chronic condition, and so on. This aggregation provides a parsimonious way of incorporating information about functional status and several major diseases. All ADLs and chronic conditions are treated as permanent, so they should be interpreted as the highest number of ADLs and chronic conditions that an individual has experienced. Hence, an individual's transition probabilities are non-zero only for higher-numbered states that have more ADLs and/or more chronic conditions than her current state.

Since health states in this framework vary across two dimensions (ADLs and chronic conditions), the index values do not follow a strict monotonic relationship with health, meaning that a consumer in a higher-numbered state is not necessarily sicker (or healthier) than one in a lower-numbered state. However, holding ADLs fixed, we expect health to worsen as the number of chronic conditions increases; similarly, holding chronic conditions fixed, we expect health to decline with the number of ADLs. Table 2 presents life expectancy at age 70 for each health state, with quality-adjusted life expectancy shown in brackets. Moving down the table corresponds to an increase in the number of ADLs, while moving across from left to right corresponds to an increase in chronic conditions. As expected, both life expectancy and quality-adjusted life expectancy decline as the number of ADLs and chronic conditions increases.

The quality of life data in the FEM are measured using the EuroQol five dimensions questionnaire (EQ-5D), which has been fielded by the MEPS (Stewart et al., 2014). These five dimensions are based on five survey questions that elicit the extent of a respondent's problems with mobility, self-care, daily activities, pain, and anxiety/depression. These questions are then combined using weights derived from stated preference data.²¹ The result is a single quality of life index, the EQ-5D, which is anchored at 0 (equivalent to death) and 1 (perfect health), as is standard in the literature (Garber and Phelps, 1997).

We calculate population-weighted averages by health state and age, and then use those means as inputs for our model. Table 3 provides the full set of summary means for ages 50 and 70, by health state. In reading the table, it helps to recall from Table 2 that health does not worsen monotonically with the health state index, because health states encompass two distinct dimensions of health. Noting that point, we can see that at age 50, life expectancy ranges from 30.9 years to 9.1 years (column 2) or 16.3 to 4.1 QALYs (column 4), quality of life ranges from 0.88 to 0.54 (column 6), out-of-pocket medical spending

²¹The five dimensions of the EQ-5D are weighted using estimates from Shaw et al. (2005). Additional technical details about the FEM are available in our Supplementary Materials.

ranges from \$686 to \$2,759 per year (column 8), and labor earnings range from \$36,111 to \$8,375 per year (column 10). Columns (12) and (13) report the probability that an individual exits her health state but remains alive, i.e., acquires at least one new ADL or chronic condition within the following year. The permanence of ADLs and chronic conditions imposes some natural restrictions on state transitions. For example, an individual in state 14 has 2 ADLs and 3 chronic conditions. She can only transition to states 19 and 20, because all others involve fewer ADLs and/or fewer chronic conditions. Health states are relatively persistent, with empirical exit rates never exceeding 15 percent at ages 50 or 70. State 20 is an absorbing state with an exit rate of 0 percent.

Figure 1a plots average out-of-pocket medical spending by age for three selected health states. These data include all inpatient, outpatient, prescription drug, and long-term care spending not paid for by insurance. Spending is higher in sicker health states and, consistent with De Nardi et al. (2010), increases greatly at older ages, when long-term care expenses arise. The effect of sickness on out-of-pocket spending is modest in comparison to long-term care costs, causing the overall gap in spending across states to shrink with age.²²

Figure 1b plots corresponding estimates for labor earnings. For healthy individuals in state 1, earnings peak at age 55 and then fall to zero by age 80. By contrast, earnings decline continuously for individuals in state 20, the sickest possible health state. When calculating the health-related interest rate, r(t, i, j), we assume income is equal to labor earnings plus a Social Security payment equal to \$15,100 per year beginning at age 65.²³

We evaluate our life-cycle model using FEM data for ages 50–100 but focus our discussion below on ages 50–80, where the FEM estimates are more precise and labor earnings are still quite relevant for consumption decisions. We assume that the distribution of initial wealth across health states is proportional to labor earnings at age 50. Finally, we calibrate the level of initial wealth by assuming that the population-weighted average VSL at age 50 is \$6 million, which matches the value from Murphy and Topel (2006) and is within the range estimated by empirical studies of VSL for working-age individuals (O'Brien, 2018). Our calibration implies that a healthy 50-year-old in state 1 has a VSL of \$6.8 million.

²²FEM medical spending estimates have been validated by comparing them to estimates from the National Health Expenditure Accounts (see Section 8.2, Appendix B of National Academies of Sciences, Engineering, and Medicine, 2015).

²³The FEM earnings data are based on HRS data from 2000–2012. In 2010, the average Social Security benefit received by retired workers was \$15,100.

3.3 Explaining variation in the value of life

We begin with a simple example. The solid red and dashed blue lines in Figure 2 report VSL and non-medical consumption for a healthy individual who experiences a modest health shock at age 60 that reduces her life expectancy by 3.0 years, suffers a severe health shock at age 70 that reduces life expectancy by 6.8 years, and then dies at age 75. Each shock also produces sudden changes to quality of life, medical spending, and labor earnings, as estimated by the FEM. The black X at age 60 reports what this person's VSL would have been absent the first health shock; the second X reports what her VSL at age 70 would have been absent the second shock. The vertical difference between the X and the red VSL line thus represents the effect of the health shock on VSL.

Consumption increases sharply following the two health shocks depicted in Figure 2. While the shock at age 60 does not affect VSL, the shock at age 70 leads to a 5 percent increase. This example demonstrates that our results from Propositions 6 and 7—which predict that consumption and VSL rise following an adverse shock to life expectancy— can hold in a more general setting where health shocks also reduce quality of life and wealth. This contrasts with the conventional complete markets life-cycle model, such as Murphy and Topel (2006), in which VSL always declines after an adverse shock to life expectancy. Finally, we note that the steeper decline in VSL following the age-70 shock indicates that the initial rise in VSL is only temporary.

To characterize the effects of health shocks among the US elderly population more generally, we conduct a Monte Carlo exercise that begins with 50,000 nationally representative individuals at age 50. Each person's health path then evolves at random according to the nationally representative health transition probabilities estimated by the FEM. At age 50, VSL ranges from \$0.8 million for individuals in poor health to \$6.8 million for those in excellent health. This dispersion results from differences in both initial health and wealth. Traditional theory accounts for variation in wealth, but it is not configured to analyze variation in baseline health states or future health risks. In order to abstract away from the effects of differences in initial wealth on VSL, the remainder of this section focuses on the 22,214 healthy individuals who were initially in health state 1 at age 50. For analyses comparing differences across health states, we use age 70 as a representative benchmark.²⁴

Figure 3a illustrates how the distribution of VSL varies over the life cycle for these 22,214 individuals. The figure plots all nineteen ventiles and the bottom and top five percentiles of the distribution in light blue, with the mean in bold. Figure 3b provides a

²⁴Some of our theoretical results, such as Proposition 7, assume consumption growth is weakly declining—a condition more likely to hold in retirement.

fuller picture of the distribution at age 70. There is a long left-tail of sick individuals who, expecting an imminent death, have spent down their wealth. Individuals with aboveaverage VSL are a mix of healthy individuals and newly diagnosed sick individuals who have begun rapidly spending down their wealth. By age 70, the VSL inter-vigintile range spans \$1.7 to \$2.9 million, with the 95th percentile consumer willing to pay 70% more for life-extension than the 5th.

This cohort of 22,214 individuals experiences about 58,000 health shocks between the ages of 50 and 80. Figure 4a displays the distribution of the change in VSL following each of those shocks.²⁵ On average, a health shock increases VSL by \$16,000 (< 1% of VSL prior to the shock), though there is considerable heterogeneity: 3 percent of shocks increase VSL by over \$50,000, while 5 percent cause VSL to fall. Figure 4b normalizes these results by the individual's quality-adjusted life expectancy. This second plot shows that health shocks increase VSL per QALY by \$70,000 (21%), on average. The distribution is skewed to the right, with the value of a QALY rising by over \$150,000 (45%) in 5 percent of cases. Although some shocks do reduce VSL, none of them reduce the value of a QALY. This rise in the willingness to pay for a QALY helps explain why people state a preference for prioritizing the health of the severely ill (Linley and Hughes, 2013; Nord et al., 1995).

Figure 5 illustrates variation in the value of life for individuals at age 70.²⁶ The positive slope of the blue dashed line indicates that, on average, VSL rises with life expectancy, consistent with recent work finding that VSL is higher for healthier people (Ketcham et al., 2022). However, the solid red line in Figure 5 indicates that VSL per QALY typically falls rapidly with quality-adjusted life expectancy. One notable deviation from this trend occurs between the second and third points (reading from left to right on the graph); this exception arises because, although quality-adjusted life expectancy increases slightly between these points, quality of life actually declines.²⁷ Individuals in the worst health state have an average VSL per QALY of \$500,000, about 1.9 times higher than individuals

²⁵The change is equal to the difference between actual VSL and a counterfactual VSL, where the counterfactual assumes the individual did not experience the health shock. See Figure 2 for a visual example.

²⁶Figure 4a described changes in VSL for people who had just experienced a shock. Because health states are persistent (see Columns (10)–(11) of Table 3), the typical 70-year-old in Figure 5 has not experienced a recent health shock. For individuals who survive sufficiently long, an adverse shock to longevity must eventually reduce VSL, relative to no shock, because it causes them to spend down their wealth more quickly.

²⁷Reading from left to right, the first four points on the solid red line correspond to states 20, 15, 19, and 10, respectively. According to Table 3, these states have life expectancies of 2.6, 3.5, 3.5, and 4.2 QALYs respectively, with a slight increase in QALYs at the third point compared to the second. However, the corresponding quality of life values are 0.54, 0.61, 0.58, and 0.66, indicating a slight decline in quality of life from the second to the third point despite the increase in life expectancy. This reduction in quality of life lowers the marginal utility of consumption, thereby increasing the value of life. With the exception of these two points, the red line generally follows a smooth, monotonic decline.

in the healthiest state, where VSL per QALY is \$260,000. These results contradict conventional cost-effectiveness theory—which implies that the value of a QALY is constant—but are consistent with generalized approaches that predict cost-effectiveness thresholds that vary with income and disease severity (Lakdawalla and Phelps, 2021; Phelps and Cinatl, 2021).

Next, we quantify how the value of reducing illness risk varies with the severity of the illness. The dashed blue line in Figure 6 reports VSI's for different illnesses from the perspective of a healthy 70-year-old with a quality-adjusted life expectancy of 11.0 QALYs. Each value represents the healthy individual's marginal willingness to pay for a reduction in the risk of death or of transitioning to one of the 19 other health states in our model. The values are inversely related to life expectancy in the sick state because it is more valuable in absolute terms to reduce the risk of a severe illness than a mild one. A marginal reduction in the risk of transitioning to the worst health state, where quality-adjusted life expectancy is 2.6 QALYs, is worth about \$2.1 million. VSL, which is a special case of VSI where life expectancy is 0 years in the sick state, is \$2.8 million.

The solid red line in Figure 6 reports VSI per QALY. The negative slope indicates that the values assigned to reducing the risk of more severe diseases are higher. For instance, reducing the risk of death (\$260,000 per QALY) is worth 10 percent more per QALY than reducing the risk of transitioning to health state 2 (\$237,000 per QALY), the mildest possible illness in our model (life expectancy of 9.7 QALYs). Notably, the solid red line in Figure 6 is positioned lower and features a flatter slope than the solid red line in Figure 5. This difference indicates that while consumers place significantly less value on QALYs gained in healthier states, the value of a QALY is relatively insensitive to the severity of the illness prevented.

Figure 7 shows how varying our utility function parameters and incorporating a bequest motive affect our estimates. Setting $\gamma = 1.5$ ($\sigma = 2/3$), which reduces the elasticity of demand for current consumption, flattens the life-cycle consumption profile and increases the value of a QALY. Setting $\gamma = 0.8$ ($\sigma = 1.25$), by contrast, pulls consumption forward in time and reduces the value of life-extension because consumption at early ages becomes a better substitute for consumption at later ages. Introducing a bequest motive encourages individuals to delay consumption, because money saved for consumption in old age has the added benefit of increasing bequests in the event of death (Figure 7a). Likewise, it reduces the value of life-extension because death becomes less costly (Figure 7b). It also dampens the increase in consumption following health shocks, eliminating the increase in VSL that followed the health shock at age 70 in the baseline specification. Thus, the likelihood of health shocks boosting VSL depends on the intertemporal elasticity of substitution and the strength of bequest motives. Varying these parameters can increase the proportion of shocks that reduce VSL beyond the 5 percent observed in our baseline specification (Figure 4a). However, the shape of the VSL per QALY curve remains stable across our sensitivity analyses, demonstrating the robustness of our finding that VSL per QALY is higher for individuals in poorer health.

Finally, Figure 8 shows what happens to our estimates when we omit quality of life or health-related financial shocks from our model.²⁸ Omitting financial shocks accentuates the increase in consumption and VSL following an adverse health shock (Panels a–b), and widens the gap in the value of QALY between the sickest and healthiest states (Panel c), but these effects are relatively small in magnitude. Omitting quality of life heightens the sensitivity of the value of a QALY to the severity of the illness (Panel d), but has little effect on other outcomes.

Overall, our results demonstrate that, while VSL can rise or fall following an adverse health shock, the value of a QALY is consistently higher for sicker individuals (Figures 7c and 8c) and rises with the severity of illness risk (Figures 7d and 8d).

4 Conclusion

The economic theory surrounding the value of life has many important applications. Yet, a number of limitations have surfaced over time. The conventional model does not distinguish between illness and death, and fails to explain several empirical facts, such as the apparent preferences of consumers to pay more for life-extension when survival prospects are bleaker.

Our model overcomes these limitations by allowing for multiple health states. Given that patient health varies widely across diseases and that many health investments aim to prevent deterioration of health rather than reduce immediate mortality risk, our framework aligns closely with real-world needs. By providing a closed-form solution, we simplify calculations, enabling practitioners to easily calculate VSL and VSI in applied settings. Indeed, our model has meaningful practical implications: using nationally representative data, we find that the value of a QALY is considerably higher for people in poorer health states.

These findings provide a rational explanation for why many people state preferences for prioritizing the health of the severely ill over other patients and for why it has proven so difficult for policymakers to encourage investments in preventive care (Linley and

²⁸Omitting quality of life is equivalent to setting q = 1 in Equation (15). Omitting financial losses is equivalent to setting the effective interest rate $r(t, Y_t, Y_{t+1}) = r = 0.03$.

Hughes, 2013; Reif et al., 2020). Kremer and Snyder (2015) show that heterogeneity in consumer valuations distorts R&D incentives by allowing firms to extract more consumer surplus from treatments than preventives. Our results suggest that differences in private VSL may reinforce this result and further disadvantage incentives to develop preventives. While we do not dismiss the potential role of behavioral biases or other non-rational factors in explaining the low demand for preventive care, we highlight how rational behavior itself contributes to this phenomenon.

Regarding healthcare resource allocation, our results provide support for the notion of a "severity premium" that increases reimbursements for treatments of more severe diseases (Skedgel et al., 2022). They also provide an additional justification for "top-up" insurance policies: because the ex post willingness to pay for treatment when sick can significantly exceed the ex ante willingness to pay for health insurance coverage when healthy, consumers benefit from a mechanism that permits them to make supplemental purchases in the sick state. Likewise, our results provide a novel rationale for insurers offering health care plans that are more generous alongside those that are less generous. Because US private insurance contracts are typically short-term arrangements, consumers' health status varies at the time of purchasing insurance. Our results imply that sicker consumers are inclined to buy more generous (and expensive) policies that cover costly treatments, whereas healthier consumers are not, even if the treatments are equally effective for both groups.

Our theoretical work points toward a number of extensions and related conjectures. First, our model does not account for the effect of health shocks on productivity, hours worked, or labor force participation—channels we expect to lead to a decrease in wealth. While our quantitative analysis incorporates data on the effect of health shocks on earnings to address these factors, a deeper theoretical investigation could yield additional insights. We conjecture that these additional channels would dampen the tendency of adverse health shocks to increase VSL.

Our analysis opens up several important avenues for further research. First, what does our model imply for the value of health insurance? While it is clear that willingness to pay for medical treatments may increase following a health shock, its effect on ex ante insurance valuations remains less clear. Second, what are the most practical strategies for incorporating our insights into the practice of cost-effectiveness? Although practitioners have traditionally assumed that a QALY possesses a constant value, many people indicate otherwise on surveys (Linley and Hughes, 2013; Sullivan, 2023). While this conventional approach is easier to implement, it overlooks variations in value that depend on health histories and illness severity. Translational research should prioritize practical strategies severity.

gies for aligning cost-effectiveness practice with the generalized theory of the value of life. Finally, what are the implications for empirical research on VSL? Prior studies have assumed that health histories can be ignored when estimating VSL (Hirth et al., 2000; Mrozek and Taylor, 2002; Viscusi and Aldy, 2003), but recent research suggests otherwise (Ketcham et al., 2022). This oversight may help explain the significant variation in empirical VSL estimates.

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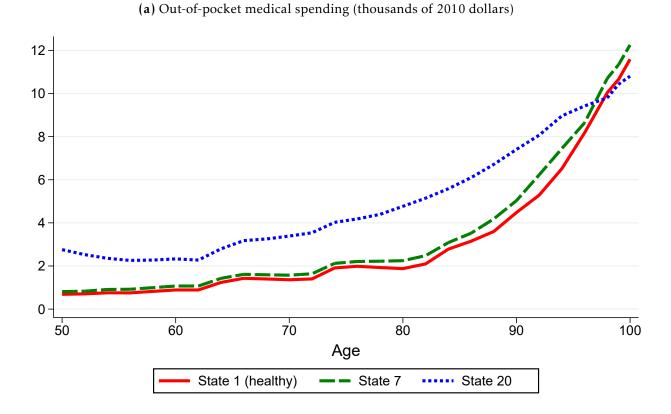
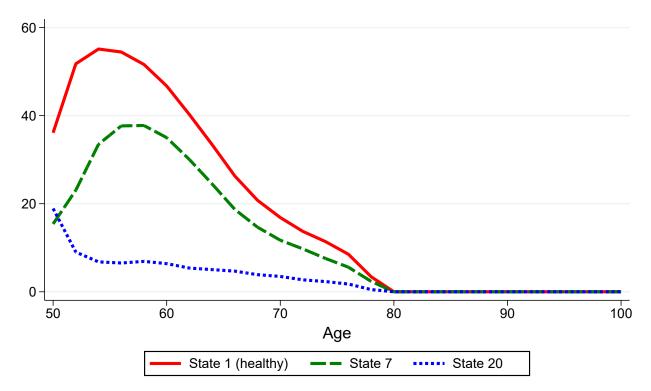


Figure 1: Average annual medical spending and labor earnings, by age

(b) Labor earnings (thousands of 2010 dollars)



Notes: All estimates are produced by the Future Elderly Model (FEM). Out-of-pocket medical spending includes all inpatient, outpatient, prescription drug, and long-term care spending not paid for by insurance. Health state 1 describes healthy individuals with no impaired activities of daily living (ADL) and no chronic conditions. State 7 describes individuals with 1 impaired activity of daily living and 1 chronic condition. State 20 describes very ill individuals with three or more impaired ADLs and four or more chronic conditions. Additional characteristics for these health states are provided in Table 3.

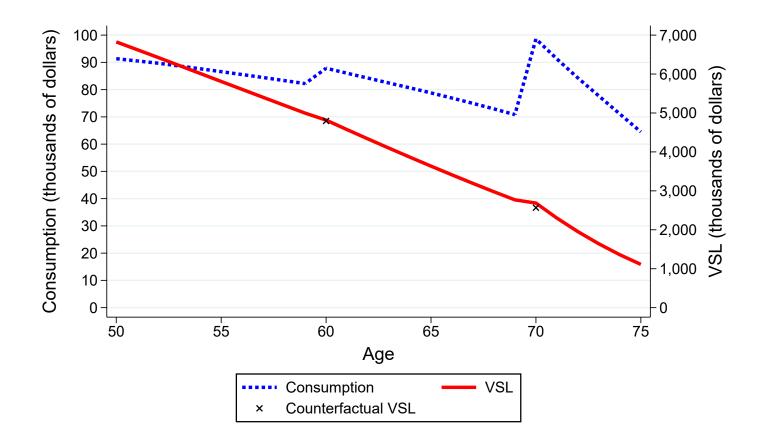


Figure 2: Consumption and VSL for an individual who suffers two health shocks and dies at age 75

Notes: This figure plots an individual's non-medical consumption (dashed blue line) and value of statistical life (VSL, solid red line) for a randomly chosen health trajectory. The individual is healthy at age 50, but then falls ill twice, once at age 60 and then again at age 70. At age 60, the illness impairs one activity of daily living (ADL). At age 70, she is diagnosed with three chronic conditions and one additional impaired ADL. Equivalently, she transitions from state 1 to state 6 at age 60, and then from state 6 to state 14 at age 70 (see Table 3). The individual dies at age 75. The two black X's report VSL for the counterfactual where the individual did not suffer a health shock at age 60 or age 70, respectively. The vertical difference between the black X and the solid red VSL line equals the effect of the health shock on VSL.

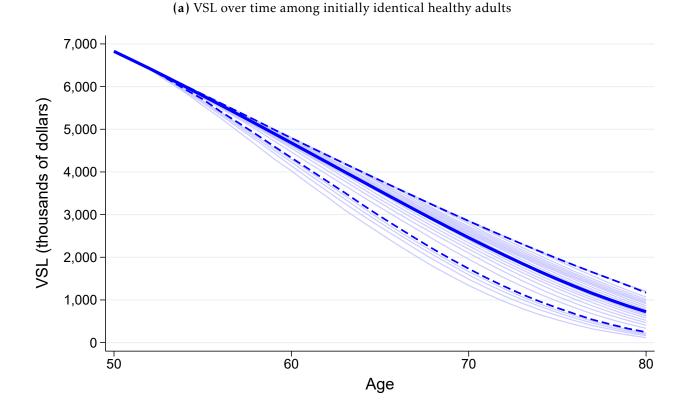
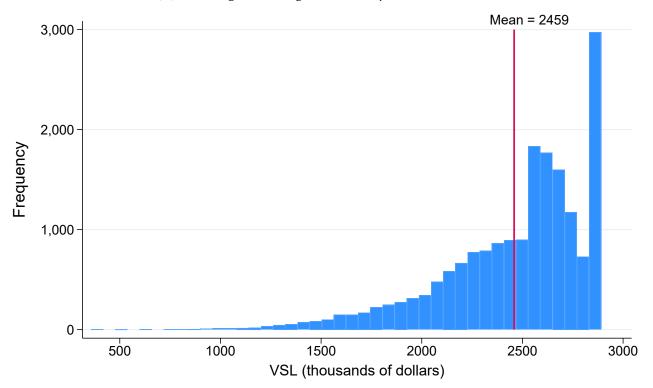
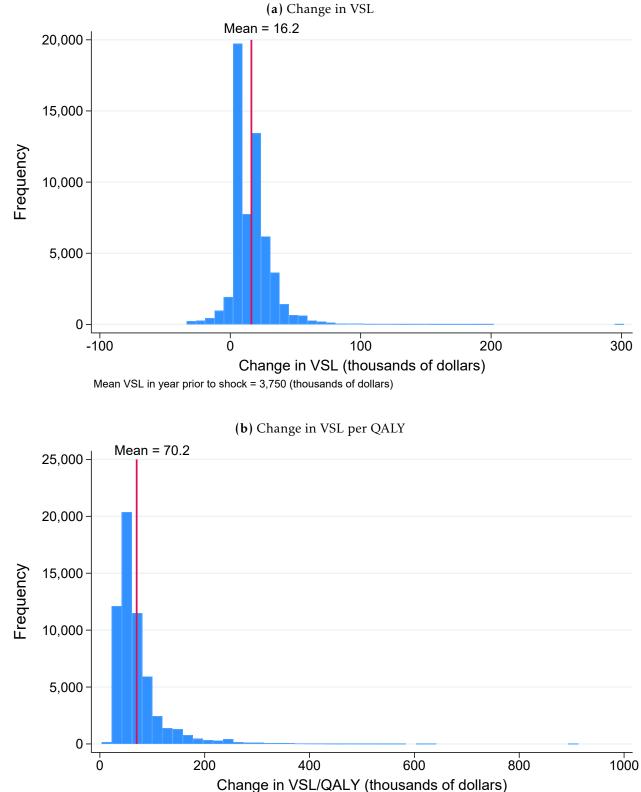


Figure 3: Health risk produces substantial heterogeneity in the value of statistical life

(b) VSL at age 70 among these initially identical adults



Notes: This figure reports VSL statistics for 22,214 initially identical individuals who at age 50 were all healthy and had the same wealth. These individuals then suffer health shocks as they age. Panel (a) plots the nineteen ventiles and the bottom and top five percentiles of VSL for this population in light blue, and the mean in dark blue. The 5th and 95th percentiles are plotted as dark blue dashed lines. Panel (b) plots the VSL distribution at age 70. Probabilities and characteristics of the health shocks are estimated by the Future Elderly Model (FEM). FEM summary statistics are available in Table 3.



Mean VSL/QALY in year prior to shock = 337 (thousands of dollars)

Notes: This figure illustrates the change in VSL following a health shock, relative to a counterfactual with no shock, among the sample of 22,214 initially healthy adults from the Future Elderly Model. These individuals experience 58,008 shocks between the ages of 50-80. Panel (a) plots the distribution of the change in VSL following a health shock. Panel (b) plots the distribution of the change in VSL per quality-adjusted life year (QALY). The vertical red lines report the means of the distributions. QALYs are discounted at a rate of 3 percent. Figure 3a reports how average VSL evolves over the life cycle for this cohort of individuals. 42

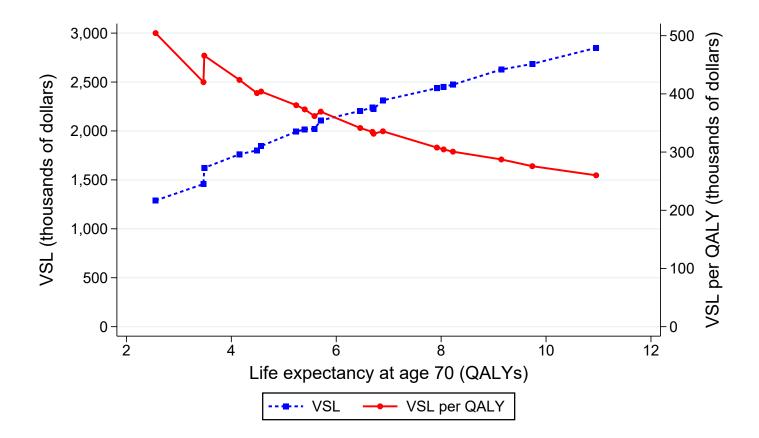
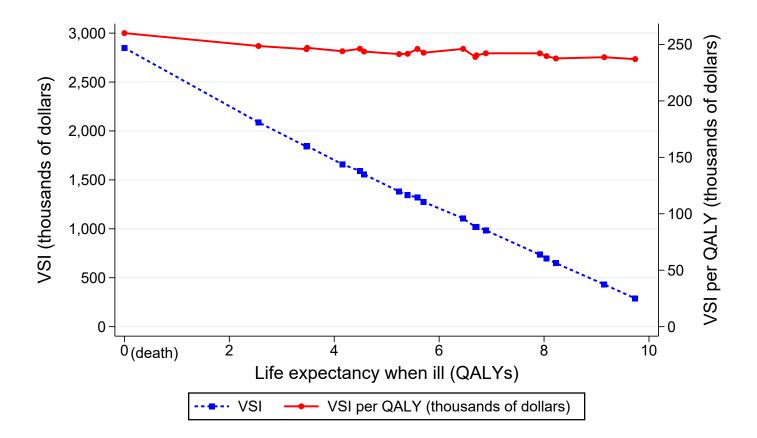
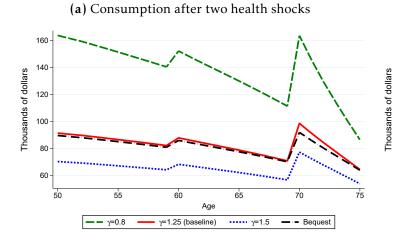


Figure 5: VSL per QALY declines with remaining life expectancy

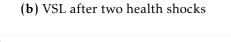
Notes: This figure presents VSL calculations among the sample of 22,214 initially healthy adults from the Future Elderly Model. The dashed blue line reports average VSL at age 70 for each of the 20 health states described in Table 3. The solid red line normalizes that value by the life expectancy for a person in that health state. Life expectancy is measured in units of quality-adjusted life-years (QALYs) and discounted at a rate of 3 percent. The negative slope of the VSL per QALY line indicates that, on average, sick individuals have a higher willingness to pay for a fixed health gain than healthier individuals.

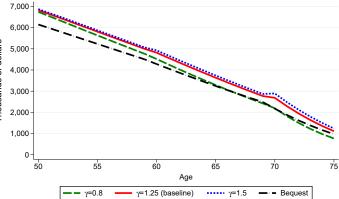


Notes: This figure presents values of statistical illness (VSI) among the sample of 22,214 initially healthy adults from the Future Elderly Model. The dashed blue line reports a healthy (health state 1) 70-year-old's marginal willingness to pay to reduce the risk of different illness, including death (value 0 on the x-axis). The solid red line normalizes that value by the change in life expectancy caused by the illness. Life expectancy is measured in units of quality-adjusted life-years (QALYs) and discounted at a rate of 3 percent. Life expectancy for a 70-year-old in health state 1 is equal to 11.0 QALYs (see Table 3). The negative slope of the VSI per QALY line indicates that individuals are willing to pay more per QALY to reduce severe illness risks than mild illness risks.

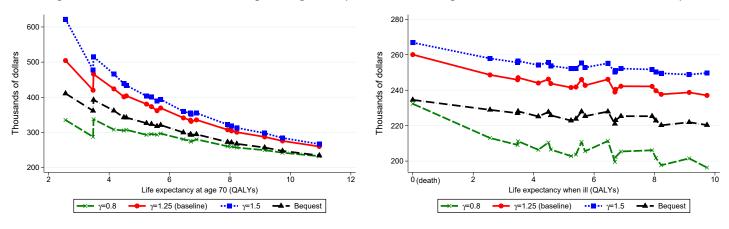


(c) VSL per QALY declines with remaining life expectancy

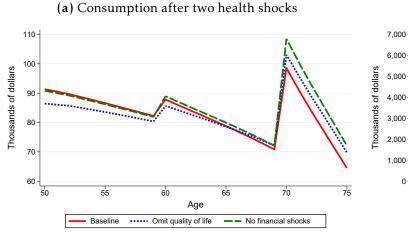




(d) VSI per QALY increases with illness severity



Notes: The solid red lines in panels (a), (b), (c), and (d) replicate the baseline results from Figure 2 (consumption and VSL), Figure 5, and Figure 6, respectively. The dashed green and dashed blue lines present results under the alternative parameter assumptions $\gamma = 0.8$ and $\gamma = 1.5$, respectively, for the utility function (15). The bequest motive specification, depicted by the black dashed line, is based on Fischer (1973) and sets the bequest motive parameter b(t) = 1.2 (see Appendix C). Life expectancy is measured in quality-adjusted life-years (QALYs) and discounted at a rate of 3 percent.



(c) VSL per life-year declines with remaining life expectancy



Age

60

····· Omit quality of life

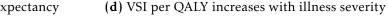
65

70

- No financial shocks

75

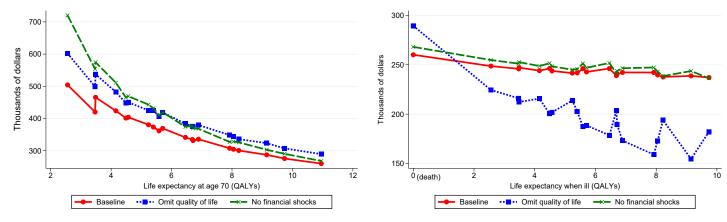
(b) VSL after two health shocks



55

Baseline

50



Notes: The solid red lines in panels (a), (b), (c), and (d) replicate the baseline results from Figure 2 (consumption and VSL), Figure 5, and Figure 6, respectively. The dashed blue lines present results when omitting quality of life from the model, which is equivalent to setting $q_{Y_t} = q = 1$. The dashed green lines present results when omitting financial losses from health transitions, which is equivalent to setting the effective interest rate $r(t, Y_t, Y_{t+1}) = r = .03$. Life expectancy is measured in units of quality-adjusted life-years (QALYs) and discounted at a rate of 3 percent.

| (1) | (2) | (3) | (4) | (5) |
|---|---|--------------------------|--|---|
| | | P | arameter values | |
| Parameter | Description | Baseline | Alternatives | Literature/data sources |
| σ | Elasticity of intertemporal substitution | 0.8 | 2/3-1.25 | Estimates from prior studies range from 0.5–1.5 (Hall, 1988; Attanasio and Weber, 2010, 1993; Bansal and Yaron, 2004; Murphy and Topel, 2006). |
| <u>c</u> | Minimum viable consumption level | \$5,000 | \$5,000 | Murphy and Topel (2006) consider minimum viable consumption ranges of 5–20 percent of full income. |
| b(t) | Bequest motive | 0 | 1.2 | Fischer (1973) considers values ranging from 0.42 to 1.20. |
| ρ | Rate of time preference | 0.03 | 0.03 | See Siegel (1992) and Moore and Viscusi (1990). |
| $r(t, Y_t, Y_{t+1})$ Mean Std. dev. | Age- and state-dependent interest rate | 0.0256 0.0093 | 0.03 0 | Based on a baseline interest rate of $r = 0.03$ (Siegel, 1992; Moore and Viscusi, 1990); FEM medical spending and earnings data; and average Social Security benefits. |
| $q_{Y_t}(t)$ Mean Std. dev. | Age- and state-dependent quality of life | $0.8194 \\ 0.0688$ | 1 0 | Future Elderly Model |
| $p_{Y_t,Y_{t+1}}(t)$ Mean Std. dev. | Age- and state-dependent transition probability | 0.8157 0.2546 | 0.8157 0.2546 | Future Elderly Model |
| $ar{d}_{Y_t}(t)$ Mean Std. dev. | Age- and state-dependent mortality probability | 0.0040 0.0528 | 0.0040 0.0528 | Future Elderly Model |
| $W_0(Y_t)$ Mean Std. dev. | State-dependent initial wealth at age 50 | \$1,600,000 \$350,000 | \$1,290,000–\$2,560,000 \$280,000–\$560,000 | Calibrated to yield an average VSL of \$6 million (Murphy and Topel, 2006; O'Brien, 2018). |

Table 1: Parameter values and sources for the numerical model calibration

Notes: This table displays the parameter values used in the numerical model described in Section 3. Column (3) lists the parameter values for the baseline specification, while Column (4) provides the values for the alternative specifications shown in Figures 7 and 8. Means and standard deviations are weighted by the prevalence of different age and state realizations over the life cycle.

| | (1) | (2) | (3) | (4) | (5) | | | |
|----------------|------------------------------|------------|------------|------------|------------|--|--|--|
| | Number of chronic conditions | | | | | | | |
| Number of ADLs | 0 | 1 | 2 | 3 | 4+ | | | |
| 0 | 17.6 [11.0] | 15.8 [9.7] | 13.6 [8.2] | 11.2 [6.7] | 9.0 [5.2] | | | |
| | (State 1) | (State 2) | (State 3) | (State 4) | (State 5) | | | |
| 1 | 15.3 [9.1] | 13.7 [8.0] | 11.6 [6.7] | 9.5 [5.4] | 7.5 [4.2] | | | |
| | (State 6) | (State 7) | (State 8) | (State 9) | (State 10) | | | |
| 2 | 13.8 [7.9] | 12.3 [6.9] | 10.4 [5.7] | 8.5 [4.6] | 6.7 [3.5] | | | |
| | (State 11) | (State 12) | (State 13) | (State 14) | (State 15) | | | |
| 3+ | 11.8 [6.5] | 10.4 [5.6] | 8.6 [4.5] | 6.9 [3.5] | 5.3 [2.6] | | | |
| | (State 16) | (State 17) | (State 18) | (State 19) | (State 20) | | | |

Table 2: Life expectancy (years and QALYs) at age 70, by health status

Notes: This table reports life expectancy in years at age 70, by number of impaired activities of daily living (ADLs) and chronic conditions. Life expectancy in QALYs, reported in brackets, is calculated using Equation (14) with a 3% discount rate. Data are obtained from the Future Elderly Model (FEM). Additional details about the FEM are available in Appendix B.

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) | (12) | (13) |
|--------------|------------|--------------|--------------|--------------|-------------|------------|-------------|-------------|------------------|-------------|---------------|-----------|-------------|
| | | Life expecta | ancy (years) | Life expecta | ncy (QALYs) | Quality of | ife (EQ-5D) | Medical spe | ending (2010 \$) | Labor earni | ngs (2010 \$) | Exit prob | ability (%) |
| Health state | ADLs / CCs | Age 50 | Age 70 | Age 50 | Age 70 | Age 50 | Age 70 | Age 50 | Age 70 | Age 50 | Age 70 | Age 50 | Age 70 |
| 1 | 0 / 0 | 30.9 | 17.6 | 16.3 | 11.0 | 0.88 | 0.87 | 686 | 1,361 | 36,111 | 16,858 | 4.2 | 12.6 |
| 2 | 0/1 | 28.2 | 15.8 | 14.8 | 9.7 | 0.85 | 0.84 | 866 | 1,578 | 34,517 | 13,309 | 3.6 | 10.8 |
| 3 | 0 / 2 | 24.6 | 13.6 | 12.8 | 8.2 | 0.81 | 0.80 | 1,145 | 1,925 | 32,483 | 10,875 | 3.6 | 10.2 |
| 4 | 0/3 | 20.5 | 11.2 | 10.7 | 6.7 | 0.77 | 0.76 | 1,487 | 2,366 | 28,718 | 8,537 | 3.9 | 10.2 |
| 5 | 0 / 4+ | 16.1 | 9.0 | 8.3 | 5.2 | 0.73 | 0.72 | 2,318 | 3,193 | 25,689 | 6,503 | 3.9 | 7.9 |
| 6 | 1 / 0 | 26.6 | 15.3 | 13.5 | 9.1 | 0.83 | 0.82 | 598 | 1,378 | 15,216 | 15,576 | 6.3 | 14.7 |
| 7 | 1/1 | 24.0 | 13.7 | 12.1 | 8.0 | 0.80 | 0.78 | 812 | 1,573 | 15,410 | 11,711 | 5.7 | 12.7 |
| 8 | 1 / 2 | 20.5 | 11.6 | 10.2 | 6.7 | 0.75 | 0.75 | 1,129 | 1,940 | 15,766 | 9,398 | 6.1 | 12.2 |
| 9 | 1/3 | 16.8 | 9.5 | 8.3 | 5.4 | 0.72 | 0.71 | 1,394 | 2,439 | 22,850 | 7,033 | 6.4 | 11.7 |
| 10 | 1 / 4+ | 13.2 | 7.5 | 6.5 | 4.2 | 0.67 | 0.66 | 2,098 | 3,287 | 11,009 | 4,904 | 6.1 | 8.6 |
| 11 | 2 / 0 | 24.3 | 13.8 | 11.9 | 7.9 | 0.78 | 0.77 | 585 | 1,314 | 14,853 | 15,383 | 7.3 | 14.3 |
| 12 | 2 / 1 | 21.5 | 12.3 | 10.4 | 6.9 | 0.75 | 0.73 | 797 | 1,600 | 12,282 | 11,488 | 7.5 | 14.3 |
| 13 | 2/2 | 18.1 | 10.4 | 8.7 | 5.7 | 0.71 | 0.69 | 1,043 | 1,934 | 17,616 | 8,915 | 7.5 | 13.8 |
| 14 | 2/3 | 15.0 | 8.5 | 7.1 | 4.6 | 0.67 | 0.66 | 1,348 | 2,412 | 10,848 | 6,507 | 7.5 | 13.1 |
| 15 | 2 / 4+ | 11.5 | 6.7 | 5.4 | 3.5 | 0.63 | 0.61 | 1,997 | 3,322 | 8,375 | 4,111 | 7.3 | 10.6 |
| 16 | 3+/0 | 21.9 | 11.8 | 10.3 | 6.5 | 0.70 | 0.69 | 693 | 1,358 | 9,369 | 13,618 | 3.4 | 11.1 |
| 17 | 3+/1 | 19.0 | 10.4 | 8.9 | 5.6 | 0.66 | 0.66 | 948 | 1,567 | 12,421 | 10,821 | 2.8 | 8.5 |
| 18 | 3+/2 | 15.7 | 8.6 | 7.3 | 4.5 | 0.62 | 0.62 | 1,105 | 1,965 | 10,130 | 7,604 | 2.3 | 7.1 |
| 19 | 3+/3 | 12.7 | 6.9 | 5.8 | 3.5 | 0.58 | 0.58 | 1,671 | 2,472 | 9,044 | 5,376 | 1.4 | 5.3 |
| 20 | 3+ / 4+ | 9.1 | 5.3 | 4.1 | 2.6 | 0.54 | 0.54 | 2,759 | 3,388 | 18,917 | 3,481 | 0.0 | 0.0 |

Table 3: Summary means for the Future Elderly Model data, by health state

Notes: This table reports selected means for the health data obtained from the Future Elderly Model (FEM). Column (1) reports the number of impaired activities of daily living (ADLs) and the number of chronic conditions (CCs), which together define a health state. Columns (2)–(3) report life expectancy in years. Columns (4)–(5) report life expectancy in QALYs, which is calculated using Equation (14) with a 3% discount rate. Columns (6)–(7) report average quality of life as measured by the EQ-5D, where 1 indexes perfect health. Columns (8)–(9) report average annual out-of-pocket medical spending, which includes all inpatient, outpatient, prescription, and long-term care spending not covered by insurance. Columns (10)–(11) report average labor earnings. Columns (12)–(13) report the percentage probability that an individual transitions to a different health state in the following year (excluding death). All impaired ADLs and chronic conditions are permanent, i.e., individuals can transition only to higher-numbered health states. Additional details about the FEM are available in Appendix B.

Online Appendix

"Health Risk and the Value of Life"

Daniel Bauer, University of Wisconsin-Madison Darius Lakdawalla, University of Southern California and NBER Julian Reif, University of Illinois and NBER

Appendix A: Mathematical Proofs

Appendix **B**: Future Elderly Model

Appendix C: Supporting Calculations for Quantitative Analysis

A Mathematical Proofs

Proof of Lemma 1. Recall that the transition rates $\lambda_{ij}(t) = 0 \quad \forall j < i$. The optimization problem in state *n* is therefore the standard problem with a single health state. We can contemplate a successive solution strategy by starting in state *n* and then moving sequentially to state n - 1, n - 2, etc. Thus, we can consider the deterministic optimization problem for an arbitrary state *i* by taking V(t, w, j), j > i, as given (exogenous):

$$V(0, W_0, i) = \max_{c_i(t)} \left\{ \int_0^T e^{-\rho t} \tilde{S}(i, t) \left(u(c_i(t), q_i(t)) + \sum_{j > i} \lambda_{ij}(t) V(t, W_i(t) \tilde{h}_{ij}, j) \right) dt \right\}$$

subject to:

$$\frac{\partial W_i(t)}{\partial t} = r_i W_i(t) - c_i(t),$$
$$W_i(0) = W_0$$

Optimal consumption and wealth in state *i* are denoted by $c_i(t)$ and $W_i(t)$, respectively. Denote the optimal value-to-go function as:

$$\tilde{V}(u, W_i(u), i) = \max_{c_i(t)} \left\{ \int_u^T e^{-\rho t} \tilde{S}(i, t) \left(u(c_i(t), q_i(t)) + \sum_{j > i} \lambda_{ij}(t) V\left(t, W_i(t) \tilde{h}_{ij}, j\right) \right) dt \right\}$$

Setting $\tilde{V}(t, W_i(t), i) = e^{-\rho t} \tilde{S}(i, t) V(t, W_i(t), i)$ then demonstrates that $V(\cdot)$ satisfies the HJB (3) for *i*. See Theorem 1 and the proof of Theorem 2 in Parpas and Webster (2013) for additional details and intuition behind this result.

Proof of Lemma 2. From (5), the marginal utility of preventing an illness or death is:

$$\begin{split} \frac{\partial V}{\partial \varepsilon}\Big|_{\varepsilon=0} &= \left. \frac{\partial}{\partial \varepsilon} \int_0^T e^{-\rho t} \exp\left\{-\int_0^t \sum_{j>i} \left(\lambda_{ij}(s) - \varepsilon \delta_{ij}(s)\right) ds\right\} \left[u\left(c_i^\varepsilon(t), q_i(t)\right) + \sum_{j>i} \left(\lambda_{ij}(t) - \varepsilon \delta_{ij}(t)\right) V(t, W_i^\varepsilon(t) \tilde{h}_{ij}, j) \right] dt \Big|_{\varepsilon=0} \\ &= \left. \int_0^T e^{-\rho t} \tilde{S}(i, t) \left[\left(\int_0^t \sum_{j>i} \delta_{ij}(s) ds \right) \left(u(c_i(t), q_i(t)) + \sum_{j>i} \lambda_{ij}(t) V(t, W_i(t) \tilde{h}_{ij}, j) \right) - \sum_{j>i} \delta_{ij}(t) V(t, W_i(t) \tilde{h}_{ij}, j) \right] dt \\ &+ \int_0^T e^{-\rho t} \tilde{S}(i, t) \left[u_c \left(c_i^\varepsilon(t), q_i(t)\right) \frac{\partial c_i^\varepsilon(t)}{\partial \varepsilon} + \sum_{j>i} \lambda_{ij}(t) \tilde{h}_{ij} \frac{\partial V(t, W_i(t) \tilde{h}_{ij}, j)}{\partial w} \frac{\partial W_i^\varepsilon(t)}{\partial \varepsilon} \right] dt \Big|_{\varepsilon=0} \end{split}$$

where $c_i^{\varepsilon}(t)$ and $W_i^{\varepsilon}(t)$ represent the equilibrium variations in $c_i(t)$ and $W_i(t)$ caused by this perturbation.

We conclude the proof by showing that the second term in the last equality is equal to 0. Note that along this path, wealth at time *t* is equal to:

$$W_i(t) = W_0 e^{r_i t} - \int_0^t e^{r_i(t-s)} c_i(s) ds$$

which implies $\frac{\partial W_i^{\varepsilon}(t)}{\partial \varepsilon} = -\int_0^t e^{r_i(t-s)} \frac{\partial c_i^{\varepsilon}(s)}{\partial \varepsilon} ds$. From the solution to the costate equation, we know that:

$$e^{-\rho t} \tilde{S}(i,t) u_c(c_i(t), q_i(t)) = \left[\int_t^T e^{(r_i - \rho)s} \tilde{S}(i,s) \sum_{j > i} \lambda_{ij}(s) \tilde{h}_{ij} \frac{\partial V\left(s, W_i(s) \tilde{h}_{ij}, j\right)}{\partial w} ds \right] e^{-r_i t} + \theta^{(i)} e^{-r_i t}$$

Thus, we can rewrite the second term in the expression for $\frac{\partial V}{\partial \varepsilon}\Big|_{\varepsilon=0}$ above as:

$$\int_{0}^{T} \left[\int_{t}^{T} e^{\left(r_{i}-\rho\right)s} \tilde{s}\left(i,s\right) \sum_{j>i} \lambda_{ij}(s)\tilde{h}_{ij} \frac{\partial V\left(s,W_{i}(s)\tilde{h}_{ij},j\right)}{\partial w} ds + \theta^{(i)} \right] e^{-r_{i}t} \frac{\partial e^{\varepsilon}_{i}(t)}{\partial \varepsilon} dt - \int_{0}^{T} e^{-\rho t} \tilde{s}\left(i,s\right) \sum_{j>i} \lambda_{ij}(t)\tilde{h}_{ij} \frac{\partial V(t,W_{i}(t)\tilde{h}_{ij},j)}{\partial w} \int_{0}^{t} e^{r_{i}(t-s)} \frac{\partial e^{\varepsilon}_{i}(s)}{\partial \varepsilon} ds dt \right|_{\varepsilon=0}$$

$$= \int_{0}^{T} \left[\int_{t}^{T} e^{\left(r_{i}-\rho\right)s} \tilde{s}\left(i,s\right) \sum_{j>i} \lambda_{ij}(s)\tilde{h}_{ij} \frac{\partial V\left(s,W_{i}(s)\tilde{h}_{ij},j\right)}{\partial w} ds \right] e^{-r_{i}t} \frac{\partial e^{\varepsilon}_{i}(t)}{\partial \varepsilon} dt - \int_{0}^{T} \left[\int_{t}^{T} e^{\left(r_{i}-\rho\right)s} \tilde{s}\left(i,s\right) \sum_{j>i} \lambda_{ij}(s)\tilde{h}_{ij} \frac{\partial V\left(s,W_{i}(s)\tilde{h}_{ij},j\right)}{\partial w} ds \right] e^{-r_{i}t} \frac{\partial e^{\varepsilon}_{i}(t)}{\partial \varepsilon} dt \right|_{\varepsilon=0}$$

$$= \theta^{(i)} \frac{\partial}{\partial \varepsilon} \underbrace{\int_{0}^{T} e^{-r_{i}t} e^{\varepsilon}_{i}(t) dt}_{W_{0}} \Big|_{\varepsilon=0}$$

where the last equality follows from application of the budget constraint.

Proof of Proposition 3. Choosing the Dirac delta function for $\delta(t)$ in Lemma 2 yields:

$$\frac{\partial V}{\partial \varepsilon}\Big|_{\varepsilon=0} = \int_0^T \left| e^{-\rho t} \tilde{S}(i,t) \left(u(c_i(t), q_i(t)) + \sum_{j>i} \lambda_{ij}(t) V(t, W_i(t), j) \right) \right| dt$$
$$= \mathbb{E} \left[\int_0^T e^{-\rho t} S(t) u(c(t), q_{Y_t}(t)) dt \middle| Y_0 = i, W(0) = W_0 \right]$$

Dividing the result by the marginal utility of wealth at time t = 0, given by Equation (4), then yields the expression for VSL given by Equation (9):

$$VSL(i) = \mathbb{E}\left[\int_0^T e^{-\rho t} S(t) \frac{u(c(t), q_{Y_t}(t))}{u_c(c(0), q_{Y_0}(0))} dt \middle| Y_0 = i, W(0) = W_0\right]$$

Proof of Proposition 4. Since the characteristics of state 1 and state 2 are identical and a transition after time *t* is no longer possible, the value functions and consumption levels for these two states are identical at time τ , holding wealth constant: $V(\tau, w, 1) = V(\tau, w, 2), c(\tau, w, 1) = c(\tau, w, 2) \forall w, \tau \ge t$. Since a transition from state 1 to state 2 at time *t* decreases wealth:

$$W(t) = W(t-)(1-h_{1,2}) < W(t-)$$

it suffices to show that VSL at time *t*:

$$\frac{V(t,W,1)}{u_c(c(t,W,1),q_1(t))}$$

is increasing in wealth. Taking derivatives, we obtain:

$$\partial/\partial W \frac{V(t,W,1)}{u_c(c(t,W,1),q_1(t))} = \frac{V_w(t,W,1)u_c(c(t,W,1),q_1(t)) - V(t,W,1)u_{cc}(c(t,W,1),q_1(t))\frac{d}{dw}c(t,W,1)}{\left(u_c(c(t,W,1),q_1(t))\right)^2} > 0$$

where the inequality follows since $V_w, u_c, \frac{d}{dw}c(t, W, 1) > 0$ and $u_{cc} < 0$.

Proof of Proposition 5. Without loss of generality, we will prove the proposition for the case where the consumer transitions from state 1 to state 2 at time t = 0. Let $c_1(t)$ denote consumption in state 1 and $c_2(t)$ denote consumption in state 2 after the transition. The interest rates in states 1 and 2 are denoted as r_1 and r_2 , respectively. By assumption, we have $r_1 > r_2$.

Suppose that initial consumption in state 1 is not lower than consumption in state 2: $c_1(0) \ge c_2(0)$. By assumption, the two states have identical probabilities of dying ($\lambda_{13}(s) = \lambda_{23}(s) \forall s$), and there are no transitions from state 1 to state 2 after

time t > 0 ($\lambda_{12}(t) = 0 \forall t > 0$). Thus, the first-order condition (7) implies:

$$\frac{p_t^{(1)}}{p_t^{(2)}} = \frac{u_c(c_1(t), q_1(t))}{u_c(c_2(t), q_2(t))} = \frac{\theta^{(1)}}{\theta^{(2)}} e^{(r_2 - r_1)t} = \frac{u_c(c_1(0), q_1(0))}{u_c(c_2(0), q_2(0))} e^{(r_2 - r_1)t} < 1$$

where $\theta^{(i)} > 0$ is a constant obtained from the solution to the costate equation in a two-state setting with $\lambda_{12}(t) = 0 \forall t > 0$. The final inequality follows because $u_{cc}(\cdot) < 0$, $q_1(0) = q_2(0)$, and $r_1 > r_2$. Thus, since $q_1(t) = q_2(t)$, we have that $c_1(t) > c_2(t) \forall t > 0$. Because utility is increasing in consumption, that implies:

$$VSL(1) = \frac{V(0, W_0, 1)}{u_c(c_1(0), q_1(0))} > \frac{V(0, W_0, 2)}{u_c(c_2(0), q_2(0))} = VSL(2)$$

Therefore, it suffices to prove that $c_1(0) \ge c_2(0)$.

Suppose by way of contradiction that $c_1(0) < c_2(0)$, so that $\theta^{(2)} < \theta^{(1)}$, where $\theta^{(i)} > 0$ is again a constant obtained from the solution to the costate equation. Consider the following consumption plan:

$$c_1^*(t) = u_c^{-1} \left[\theta^{(2)} e^{-r_1 t} e^{\rho t} e^{\int_0^t \lambda_{13}(s) ds}, q_1(t) \right] > u_c^{-1} \left[\theta^{(1)} e^{-r_1 t} e^{\rho t} e^{\int_0^t \lambda_{13}(s) ds}, q_1(t) \right] = c_1(t)$$

where the inequality follows because $u_{cc}(\cdot) < 0$, and hence $u_{cc}^{-1}(\cdot) < 0$. We will show that $\{c_1^*(t)\}$ satisfies the budget constraint, which contradicts the optimality of the plan $\{c_1(t)\}$.

Consider the function:

$$g(x) = x \cdot u_c^{-1} \left[\theta^{(2)} x e^{\rho t} e^{\int_0^t \lambda_{13}(s) ds}, q(t) \right]$$

ct

Then:

$$g'(x) = \underbrace{u_c^{-1} \left[\theta^{(2)} x e^{\rho t} e^{\int_0^t \lambda_{13}(s) ds}, q(t) \right]}_{y} - \frac{x \cdot \theta^{(2)} e^{\rho t} e^{\int_0^t \lambda_{13}(s) ds}}{-u_{cc} \left[u_c^{-1} \left[\theta^{(2)} x e^{\rho t} e^{\int_0^t \lambda_{13}(s) ds}, q(t) \right], q(t) \right]}_{y}$$

= $y - \frac{u_c[y, q(t)]}{-u_{cc}[y, q(t)]y} y = y(1 - \sigma)$
 ≥ 0

where the last inequality follows because we have assumed utility satisfies the condition $\sigma < 1$. Thus:

$$\int_0^T e^{-r_1 t} c_1^*(t) dt = \int_0^T g(e^{-r_1 t}) dt \le \int_0^T g(e^{-r_2 t}) dt = \int_0^T e^{-r_2 t} c_2(t) dt = W_0$$

where the last equality follows because $\{c_2(t)\}$ satisfies the budget constraint. Thus, $\{c_1^*(t)\}$ also satisfies the budget constraint, which yields the contradiction.

Proof of Proposition 6. Without loss of generality, we will prove the proposition for the case where the consumer transitions from state 1 to state 2 at time t = 0. Because we hold quality of life constant, we omit $q_i(t)$ in the notation below in order to keep the presentation concise.

We want to prove that $c_2(0) > c_1(0)$. Assume by way of contradiction that $c_2(0) \le c_1(0)$. We will show that this assumption implies $c_2(t) < c_1(t)$ for all t > 0, which is a contradiction since the feasible consumption plan $c_1(\cdot)$ dominates $c_2(\cdot)$.

We proceed by inductively constructing a sequence $0 < t_1 < t_2 \dots$ where for each element in the sequence:

$$c_{2}(t_{i}) < c_{1}(t_{i}) W_{1}(t_{i}) < W_{2}(t_{i}) p_{t_{i}}^{(1)} \le \exp\left\{-\int_{0}^{t_{i}} \lambda_{12}(s) ds\right\} p_{t_{i}}^{(2)}$$

To construct the sequence, for the base case i = 1, we first note that from the first-order condition (7), we obtain:

$$p_0^{(1)} = u_c(c_1(0)) \le u_c(c_2(0)) = p_0^{(2)}$$

The costate equation (6) then implies:

$$\begin{split} \dot{p}_{0}^{(1)} &= -p_{0}^{(1)}r - \lambda_{12}(0)u_{c}(c_{2}(0)) \\ &= -p_{0}^{(1)} \left[r + \lambda_{12}(0) \underbrace{\frac{u_{c}(c_{2}(0))}{u_{c}(c_{1}(0))}}_{\geq 1} \right] \\ &\leq -p_{0}^{(1)} \left[r + \lambda_{12}(0) \right] = \left. \frac{\partial g(t)}{\partial t} \right|_{t=0} \end{split}$$

where $g(t) = p_0^{(1)} \exp\left\{-\int_0^t r + \lambda_{12}(s) ds\right\}$. Hence, there exists a $t_1 > t_0 = 0$ such that:

$$p_t^{(1)} \le g(t) \le p_0^{(2)} \exp\left\{-\int_0^t (r+\lambda_{12}(s))\,ds\right\} = p_t^{(2)} \exp\left\{-\int_0^t \lambda_{12}(s)\,ds\right\}, \ 0 \le t \le t_1$$

which together with the first-order condition (7) implies:

$$e^{-\rho t} \exp\left\{-\int_0^t \left(\lambda_{12}(s) + \lambda_{13}(s)\right) ds\right\} u_c(c_1(t)) \le e^{-\rho t} \exp\left\{-\int_0^t \left(\lambda_{12}(s) + \lambda_{23}(s)\right) ds\right\} u_c(c_2(t)), \ 0 \le t \le t_1$$

so that $c_1(t) > c_2(t)$, $0 < t \le t_1$. The first inequality in turn implies $W_1(t_1) < W_2(t_1)$.

For the induction step, suppose that the following properties also hold for $i \ge 1$:

$$c_{2}(t_{i}) < c_{1}(t_{i})$$

$$W_{1}(t_{i}) < W_{2}(t_{i})$$

$$p_{t_{i}}^{(1)} \le \exp\left\{-\int_{0}^{t_{i}} \lambda_{12}(s)ds\right\} p_{t_{i}}^{(2)}$$

The induction hypothesis implies:

$$c(t_i, W_1(t_i), 2) \le c(t_i, W_2(t_i), 2) = c_2(t_i) < c_1(t_i)$$

so that:

$$\begin{split} \dot{p}_{t_i}^{(1)} &= -p_{t_i}^{(1)} r - e^{-\rho t_i} \tilde{S}(1, t_i) \,\lambda_{12}(t_i) \,u_c\left(c(t_i, W_1(t_i), 2)\right) \\ &= -p_{t_i}^{(1)} \left[r + \lambda_{12}(t_i) \underbrace{\frac{u_c\left(c(t_i, W_1(t_i), 2)\right)}{u_c\left(c_1(t_i)\right)}}_{>1} \right] \\ &< -p_{t_i}^{(1)} \left[r + \lambda_{12}(t_i) \right] = \frac{\partial \tilde{g}(t)}{\partial t} \bigg|_{t=0} \end{split}$$

with $\tilde{g}(t) = p_{t_i}^{(1)} \exp\left\{-\int_{t_i}^t (r + \lambda_{12}(s)) ds\right\}$. Hence, there exists a $t_{i+1} > t_i$ such that:

$$p_t^{(1)} \le \tilde{g}(t)$$

$$\le \exp\left\{-\int_0^{t_i} \lambda_{12}(s) \, ds\right\} \, p_{t_i}^{(2)} \exp\left\{-\int_{t_i}^t (r + \lambda_{12}(s)) \, ds\right\}$$

$$= p_t^{(2)} \exp\left\{-\int_0^t \lambda_{12}(s) \, ds\right\}, \ t_i \le t \le t_{i+1}$$

Applying again the first-order condition (7) for all $t_i \le t \le t_{i+1}$ yields:

$$\exp\left\{-\int_{0}^{t} (\lambda_{12}(s) + \lambda_{13}(s)) \, ds\right\} \, u_{c}(c_{1}(t)) \le \exp\left\{-\int_{0}^{t} (\lambda_{12}(s) + \lambda_{23}(s)) \, ds\right\} \, u_{c}(c_{2}(t))$$

which in turn implies $u_c(c_1(t)) < u_c(c_2(t))$ and $c_2(t) < c_1(t)$. Once again, this inequality implies $W_1(t_{i+1}) < W_2(t_{i+1})$.

Thus, we have proven the existence of the sequence. We then obtain $c_2(t) < c_1(t)$, t > 0, by noting that $\{t_i\}_{i \ge 0}$ strictly increases due to the uniformly boundedness condition on $\lambda_{12}(t)$, which is the desired contradiction.

We note that this proof implies that the consumption paths $c_1(t)$ and $c_2(t)$ cross (at most) once. As soon as $c_1(t)$ exceeds $c_2(t)$ for some time t_0 , $c_1(t)$ will exceed $c_2(t)$ for $t > t_0$. However, we have that $c_2(t)$ exceeds $c_1(t)$ prior to t_0 . In particular, consumption jumps up upon transition at time zero.

For the proof of Proposition 7, we need the following two lemmas (Lemma A.1 and A.2). Since these lemmas hold in more general situations than the assumptions of Proposition 7 and may be of independent interest, we present them separately.

Lemma A.1. Consider a setting without health shocks, i.e., $h_{ij} \equiv 0 \forall i, j$. Then the marginal utility of wealth in state *i* is equal to:

$$\frac{\partial V(0, W_0, i)}{\partial W_0} = u_c(c_i(0), q_i(0))$$

= $\mathbb{E}\left[e^{\int_0^t (r_{Y_s} - \rho)ds} \exp\left\{-\int_0^t \mu(s)ds\right\} u_c(c(t, W(t), Y_t), q_{Y_t}(t)) \middle| Y_0 = i, W(0) = W_0\right], \forall t > 0$

Proof of Lemma A.1. The first equality in Lemma A.1 follows immediately from the first-order condition in state i in the HJB (3) (see Equation (4)). Our proof derives the second equality, which shows that the consumer sets the expected discounted marginal utility of consumption at time t equal to the current marginal utility of wealth. This result is the stochastic analogue of the first-order condition from a conventional (deterministic health risk) model.

We show the result at an arbitrary time *t* and a future time $\tau > t$:

$$\frac{\partial V(t, W_i(t), i)}{\partial W_i(t)} = u_c(c_i(t), q_i(t)) = \mathbb{E}\left[e^{\int_t^\tau (r_{Y_s} - \rho)ds} \exp\left\{-\int_t^\tau \mu(s)ds\right\} u_c\left(c(\tau, W(\tau), Y_\tau), q_{Y_\tau}(\tau)\right) \middle| Y_t = i, W(t) = W_i(t)\right], \forall \tau > t$$

The proof proceeds by induction on $i \le n$. For the base case i = n, in which no state transitions are possible, the solution to the costate equation (6) simplifies to:

$$p_{\tau}^{(n)} = \theta^{(n)} e^{-r_n \tau}$$

= $\exp\left\{-\int_0^{\tau} \rho + \lambda_{n,n+1}(s) ds\right\} u_c(c_n(\tau), q_n(\tau))$
= $\theta^{(n)} e^{-r_n t} e^{-r_n(\tau-t)}$
= $p_t^{(n)} e^{-r_n(\tau-t)}$
= $\exp\left\{-\int_0^t \rho + \lambda_{n,n+1}(s) ds\right\} u_c(c_n(t), q_n(t)) e^{-r_n(\tau-t)}$

where the second equality makes use of the first-order condition (7). Using the expressions in the second and the last lines then gives:

$$u_{c}(c_{n}(t), q_{n}(t)) = e^{r_{n}(\tau-t)} e^{-\rho(\tau-t)} \exp\left\{-\int_{t}^{\tau} \lambda_{n,n+1}(s) ds\right\} u_{c}(c_{n}(\tau), q_{n}(\tau))$$

which shows that the result holds for i = n.

For the induction step, suppose the lemma is true for j > i, $1 \le i \le n - 1$. For any subinterval $[0, \tau]$, the solution of the costate equation can be written as:

$$p_t^{(i)} = \left[\int_t^\tau e^{(r_i - \rho)s} \exp\left\{-\int_0^s \sum_{j > i} \lambda_{ij}(u) du\right\} \sum_{j > i} \lambda_{ij}(s) \frac{\partial V(s, W_i(s), j)}{\partial W_i(s)} ds\right] e^{-r_i t} + \theta(\tau, i) e^{-r_i t}$$
(A.1)

where $\theta(\tau, i)$ is a constant that depends on the choice of τ and i. (Take the derivative of $p_t^{(i)}$ with respect to t to verify.) Evaluating Equation (A.1) at $t = \tau$ and combining with Equation (7) from the main text yields:

$$p_{\tau}^{(i)} = \theta(\tau, i) e^{-r_i \tau} = \exp\left\{-\int_0^{\tau} \rho + \sum_{j>i} \lambda_{ij}(s) ds\right\} u_c(c_i(\tau), q_i(\tau))$$

which implies:

$$\theta(\tau, i) = e^{(r_i - \rho)\tau} \exp\left\{-\int_0^\tau \sum_{j > i} \lambda_{ij}(s) ds\right\} u_c(c_i(\tau), q_i(\tau))$$
(A.2)

Plugging Equations (7) and (A.2) into Equation (A.1) yields:

$$u_{c}(c_{i}(t),q_{i}(t))\exp\left\{-\int_{0}^{t}\rho+\sum_{j>i}\lambda_{ij}(s)\,ds\right\} = \left[\int_{t}^{\tau}e^{(r_{i}-\rho)s}\exp\left\{-\int_{0}^{s}\sum_{j>i}\lambda_{ij}(u)\,du\right\}\sum_{j>i}\lambda_{ij}(s)\frac{\partial V(s,W_{i}(s),j)}{\partial W_{i}(s)}\,ds\right]e^{-r_{i}t} + e^{-r_{i}t}e^{(r_{i}-\rho)\tau}\exp\left\{-\int_{0}^{\tau}\sum_{j>i}\lambda_{ij}(s)ds\right\}u_{c}(c_{i}(\tau),q_{i}(\tau))$$

Since $\frac{\partial V(s, W_i(s), j)}{\partial W_i(s)} = u_c(c(s, W_i(s), j), q_j(s))$ from the first-order condition in the HJB for state *j*, we obtain:

$$\begin{split} u_{c}(c_{i}(t),q_{i}(t)) &= \int_{t}^{\tau} e^{(r_{i}-\rho)(s-t)} \exp\left\{-\int_{t}^{s} \sum_{j>i} \lambda_{ij}(u) \, du\right\} \sum_{j>i} \lambda_{ij}(s) \, u_{c}(c(s,W_{i}(s),j),q_{j}(s)) \, ds + e^{(r_{i}-\rho)(\tau-t)} \exp\left\{-\int_{t}^{\tau} \sum_{j>i} \lambda_{ij}(s) \, ds\right\} \, u_{c}(c_{i}(\tau),q_{i}(\tau)) \\ &= \int_{t}^{\tau} e^{(r_{i}-\rho)(s-t)} \exp\left\{-\int_{t}^{s} \sum_{j>i} \lambda_{ij}(u) \, du\right\} \sum_{j>i} \lambda_{ij}(s) \mathbb{E}\left[e^{(r_{i}-\rho)(\tau-s)} \exp\left\{-\int_{s}^{\tau} \mu(s) ds\right\} \, u_{c}\left(c(\tau,W(\tau),Y_{\tau}),q_{Y_{\tau}}(\tau)\right)\right| Y_{s} = j, W(s) = W_{i}(s)\right] ds \\ &+ e^{(r_{i}-\rho)(\tau-t)} \exp\left\{-\int_{t}^{\tau} \sum_{j>i} \lambda_{ij}(s) \, ds\right\} \, u_{c}(c_{i}(\tau),q_{i}(\tau)) \\ &= \mathbb{E}\left[e^{\int_{t}^{\tau} (r_{Y_{s}}-\rho) ds} \exp\left\{-\int_{t}^{\tau} \mu(s) \, ds\right\} \, u_{c}\left(c(\tau,W(\tau),Y_{\tau}),q_{y_{\tau}}(\tau)\right)\right| Y_{t} = i, \, W(t) = W_{i}(t)\right] \end{split}$$

where the second equality follows from the induction hypothesis.

We can use Lemma A.1 to obtain the following expression for VSL:

Lemma A.2. Applying the second equality given in Lemma A.1 to Equation (9) and rearranging yields the following, equivalent expression for VSL in state i:

$$VSL(i) = \int_0^T \tilde{v}(i,t) dt$$

where $\tilde{v}(i, t)$ represents the discounted value of a one-period change in survival from the perspective of current time:

$$\tilde{v}(i,t) = \frac{\mathbb{E}\left[S(t) \ u(c(t), q_{Y_t}(t)) \middle| Y_0 = i, W(0) = W_0\right]}{\mathbb{E}\left[e^{\int_0^t r_{Y_s} ds} S(t) \ u_c(c(t), q_{Y_t}(t)) \middle| Y_0 = i, W(0) = W_0\right]}$$

Proof of Lemma A.2. Applying Lemma A.1 for t = 0 allows us to rewrite VSL as:

$$VSL(i) = \mathbb{E}\left[\int_{0}^{T} e^{-\rho t} \frac{S(t) u(c(t), q_{Y_{t}}(t))}{\mathbb{E}\left[e^{\int_{0}^{t} (r_{Y_{s}}-\rho)ds} \exp\left\{-\int_{0}^{t} \mu(s)ds\right\} u_{c}\left(c(t), q_{Y_{t}}(t)\right) \middle| Y_{0} = i, W(0) = W_{0}\right]} dt \middle| Y_{0} = i, W(0) = W_{0}\right]$$
$$= \mathbb{E}\left[\int_{0}^{T} \frac{S(t) u(c(t), q_{Y_{t}}(t))}{\mathbb{E}\left[e^{\int_{0}^{t} r_{Y_{s}}ds} \exp\left\{-\int_{0}^{t} \mu(s)ds\right\} u_{c}\left(c(t), q_{Y_{t}}(t)\right) \middle| Y_{0} = i, W(0) = W_{0}\right]} dt \middle| Y_{0} = i, W(0) = W_{0}\right]$$

Exchanging expectation and integration then yields:

$$VSL(i) = \int_0^T \tilde{v}(i,t) \, dt$$

Proof of Proposition 7. Without loss of generality, consider the case t = 0. Applying our assumptions that $r_1 = r_2 \le \rho$, $q_1(s) = q_2(s) = q \forall s$, $\lambda_{13}(s) < \lambda_{23}(s) \forall s$, and no financial shocks to Equation (11) implies that $c_1(t)$ and $c_2(t)$ are decreasing in t. In addition, from Proposition 6 we have that $c_1(0) < c_2(0)$, $c_1(t) < c_2(t)$ for $t \le t_0$, and $c_1(t) > c_2(t)$ for $t > t_0$, where t_0 was defined in the proof of Proposition 6.

Applying our assumption that there are no state transitions for t > 0 to Lemma A.2 yields:

$$VSL(2,0) = \int_0^T \frac{S_2(t)u(c_2(t))}{e^{rt}S_2(t)u_c(c_2(t))}dt = \int_0^T e^{-rt}\frac{u(c_2(t))}{u_c(c_2(t))}dt$$

and:

$$VSL(1,0) = \int_0^T e^{-rt} \frac{u(c_1(t))}{u_c(c_1(t))} dt$$

Let $Y(x) \equiv \frac{u(x)}{u_c(x)}$. Under the stated assumptions on preferences, we have that:

$$Y'(x) = 1 - \frac{u(x)u_{cc}(x)}{(u_c(x))^2} > 0,$$

$$Y''(x) = \frac{2(u_{cc}(x))^2 u(x) - u_c^2(x)u_{cc}(x) - u_c(x)u(x)u_{ccc}(x)}{(u_c(x))^3} > 0$$

Employing Taylor's theorem and making use of the assumption that c(t) is weakly declining in t then implies that for some $\xi(t)$ that lies in-between $c_1(t)$ and $c_2(t)$:

$$\begin{aligned} VSL(2,0) &= \int_{0}^{T} e^{-rt} Y(c_{2}(t)) dt \\ &= \int_{0}^{T} e^{-rt} \left[Y(c_{1}(t)) + [c_{2}(t) - c_{1}(t)] Y'(c_{1}(t)) + \underbrace{\frac{1}{2} [c_{2}(t) - c_{1}(t)]^{2} Y''(\xi(t))]}_{>0} \right] dt \\ &> \int_{0}^{T} e^{-rt} Y(c_{1}(t)) dt + \int_{0}^{t_{0}} e^{-rt} Y'(c_{1}(t)) \underbrace{[c_{2}(t) - c_{1}(t)]}_{\geq 0} dt + \int_{t_{0}}^{T} e^{-rt} Y'(c_{1}(t)) \underbrace{[c_{2}(t) - c_{1}(t)]}_{\leq 0} dt + \int_{t_{0}}^{T} e^{-rt} Y'(c_{1}(t)) \underbrace{[c_{2}(t) - c_{1}(t)]}_{\leq 0} dt \\ &\ge \int_{0}^{T} e^{-rt} Y(c_{1}(t)) dt + \int_{0}^{t_{0}} e^{-rt} Y'(c_{1}(t_{0})) [c_{2}(t) - c_{1}(t)] dt + \int_{t_{0}}^{T} e^{-rt} Y'(c_{1}(t_{0})) [c_{2}(t) - c_{1}(t)] dt \\ &= \int_{0}^{T} e^{-rt} Y(c_{1}(t)) dt + Y'(c_{1}(t_{0})) \underbrace{\left[\int_{0}^{T} e^{-rt} c_{2}(t) dt - \int_{0}^{T} e^{-rt} c_{1}(t) dt\right]}_{=0} \\ &= \int_{0}^{T} e^{-rt} Y(c_{1}(t)) dt \\ &= VSL(1,0) \end{aligned}$$

where the final step follows from the budget constraint.

B Future Elderly Model

The empirical exercises presented in Section 3 employ data obtained from the Future Elderly Model (FEM). The FEM is a microsimulation model that projects future health and medical spending for Americans ages 50 and over. It has been used by a variety of researchers and policy analysts to understand the implications of population aging, health trends, new medical technologies, pandemics, and possible health policy interventions in the US, Europe, and Asia (Goldman et al., 2005; Lakdawalla et al., 2005, 2008; Goldman et al., 2009, 2010; Michaud et al., 2011, 2012; Goldman et al., 2013; Goldman and Orszag, 2014; National Academies of Sciences, Engineering, and Medicine, 2015; Chen et al., 2016; Gonzalez-Gonzalez et al., 2017; Leaf et al., 2021; Reif et al., 2021). Detailed technical information about its data sources and methods is included in our Supplementary Materials, and is also available online at:

https://healthpolicy.usc.edu/future-elderly-model/fem-technical-specifications/

The FEM has three core modules. The first is the Replenishing Cohorts module, which predicts economic and health outcomes of new cohorts of 50-year-olds using data from the Panel Study of Income Dynamics, and incorporates trends in disease and other outcomes based on data from external sources, such as the National Health Interview Survey and the American Community Survey. This module generates new cohorts as the simulation proceeds, so that we can measure outcomes for the age 50+ population in any given year.

The second component is the Health Transition module, which uses the longitudinal structure of the Health and Retirement Study (HRS) to calculate transition probabilities across various health states, including chronic conditions, functional status, body-mass index, and mortality. These transition probabilities depend on a battery of predictors: age, sex, education, race, ethnicity, smoking behavior, marital status, employment and health conditions. FEM transitions produce a large set of simulated outcomes, including diabetes, high-blood pressure, heart disease, cancer (except skin cancer), stroke or transient ischemic attack, and lung disease (either or both chronic bronchitis and emphysema), disability, and body-mass index. Disability is measured by limitations in instrumental activities of daily living, activities of daily living, and residence in a nursing home.

Finally, the Policy Outcomes module estimates medical spending, including payments made by insurers (Medicare, Medicaid and Private) and out-of-pocket payments made by individuals. Medical spending for an individual is predicted as a function of health status (chronic conditions and functional status), demographics (age, sex, race, ethnicity, and education), nursing home status, and mortality. Estimates are based on spending data from the Medical Expenditure Panel Survey for individuals ages 64 and younger and the Medicare Current Beneficiary Survey for individuals ages 65 and older.

The following example illustrates how the three modules interact. For the year 2014, the model begins with the population of Americans ages 50 and over based on nationally representative data from the HRS. Individual-level health and economic outcomes for the next two years are predicted using the Policy Outcomes module. The cohort is then aged two years using the Health Transition Module. Aggregate health and functional status outcomes for those years are then calculated. At that point, a new cohort of 50-year-olds is introduced into the 2016 population using the Replenishing Cohort module, and they join those who survived from 2014 to 2016. This forms the age 50+ population for 2016. The transition model is then applied to this population. The same process is repeated until reaching the last year of the simulation. For our study, we ran the simulation until the year 2064, which gives us complete life-cycle data for ages 50–100 for all people who were ages 50 and over as of 2014.

The projections produced by the FEM have been extensively validated. Mortality forecasts line up closely with published death counts and achieve lower error rates than alternative forecasts used by the Social Security Administration (Leaf et al., 2021). Population, smoking behavior, cancer, diabetes, heart disease, hypertension, lung disease, and stroke forecasts perform well in cross-validation exercises. Medical spending data have been comprehensively tested against national aggregates.

C Supporting Calculations for Quantitative Analysis

This appendix provides the solution to the discrete-time dynamic programming problem described in Section 3.1. This model is solved analytically and provides exact solutions for optimal consumption.

The consumer's problem is:

$$\max_{c(t)} \mathbb{E}\left[\sum_{t=0}^{T} e^{-\rho t} S_0(t) u(c(t), q_{Y_t}(t)) + e^{-\rho t} \left((S_0(t) - S_0(t+1)) u(W(t) - c(t), b(t)) \right) \middle| Y_0, W_0 \right]$$

subject to:

$$W(0) = W_0,$$

 $W(t) \ge 0,$
 $W(t+1) = (W(t) - c(t))e^{r(t, Y_t, Y_{t+1})}$

where all variables are defined as in the main text. The strength of the bequest motive is governed by the parameter b(t). We set b(t) = 0 in our baseline specification, which assumes no bequest motive (and normalizes utility of death to zero). The utility function is given by Equation (15) from the main text:

$$u(c,q) = q\left(\frac{c^{1-\gamma}-\underline{c}^{1-\gamma}}{1-\gamma}\right)$$

where \underline{c} is the minimum viable level of consumption for a healthy person with no bequest motive. Because optimal consumption is unaffected by affine transformations of utility, we shall initially assume $u(c,q) = q c^{1-\gamma}/(1-\gamma)$ when solving the model for consumption.

Define the value function as:

$$V(t, W(t), Y_t) = \max_{c(s)} \mathbb{E}\left[\sum_{s=t}^{T} e^{-\rho(s-t)} S_t(s) u(c(s), q_{Y_s}(s)) + e^{-\rho(s-t)} (S_t(s) - S_t(s+1)) u(W(s) - c(s), b(s))\right| Y_t, W(t)\right]$$

subject to:

$$W(s+1) = (W(s) - c(s))e^{r(s, Y_s, Y_{s+1})}, s > t, W(s) \ge 0$$

We reformulate this optimization problem as a recursive Bellman equation:

$$V(t, w, i) = \max_{c(t)} \left\{ u(c(t), q_i(t)) + \overline{d}_i(t) u((w - c(t)), b(t)) + e^{-\rho} \left(1 - \overline{d}_i(t)\right) \sum_{j=1}^n p_{ij}(t) V\left(t + 1, (w - c(t)) e^{r(t, i, j)}, j\right) \right\}$$

Proposition C.1. *The value function and the optimal consumption level satisfy:*

$$V(t, w, i) = \frac{w^{1-\gamma}}{1-\gamma} K_{t,i},$$
$$c^*(t, w, i) = w \times c_{t,i}$$

where $K_{t,i}$ satisfies the recursion:

$$\begin{split} K_{t,i} &= \left[q_i(t)^{\frac{1}{\gamma}} + \left[\overline{d}_i(t) b(t) + e^{-\rho} \left(1 - \overline{d}_i(t) \right) \left(\sum_{j=1}^n p_{ij}(t) e^{r(t,i,j)(1-\gamma)} K_{t+1,j} \right) \right]^{\frac{1}{\gamma}} \right]^{\gamma}, \ t < T, \\ K_{T,i} &= \left[q_i(T)^{\frac{1}{\gamma}} + b(T)^{\frac{1}{\gamma}} \right]^{\gamma} \end{split}$$

and:

$$\begin{split} c_{t,i} &= \left[1 + \left(\frac{\left[\overline{d}_i(t) \, b(t) + e^{-\rho} \left(1 - \overline{d}_i(t) \right) \left(\sum_{j=1}^n p_{ij}(t) e^{r(t,i,j)(1-\gamma)} \, K_{t+1,j} \right) \right]}{q_i(t)} \right]^{\frac{1}{\gamma}} \right]^{-1} \\ &= \left[1 + \left(\overline{d}_i(t) \frac{b(t)}{q_i(t)} + e^{-\rho} \left(1 - \overline{d}_i(t) \right) \left(\sum_{j=1}^n p_{ij}(t) e^{r(t,i,j)(1-\gamma)} \frac{q_j(t+1)}{q_i(t)} \frac{1}{c_{t+1,j}^{\gamma}} \right) \right)^{\frac{1}{\gamma}} \right]^{-1}, t < T, \\ c_{T,i} &= \left[1 + \left(\frac{b(t)}{q_i(t)} \right)^{\frac{1}{\gamma}} \right]^{-1} \end{split}$$

Proof. See Appendix C.1

When calculating VSL, we incorporate minimum viable consumption back into the utility function. In this case, the value function is:

$$V(0, w, i) = \sum_{t=0}^{T} e^{-\rho t} \mathbb{E} \left[\exp \left\{ -\int_{0}^{t} \mu(s) ds \right\} \left(q_{Y_{t}}(t) \frac{c(t)^{1-\gamma} - \underline{c}^{1-\gamma}}{1-\gamma} \right) \middle| Y_{0} = i, W(0) = w \right] \\ + e^{-\rho t} \mathbb{E} \left[\left(\exp \left\{ -\int_{0}^{t} \mu(s) ds \right\} - \exp \left\{ -\int_{0}^{t+1} \mu(s) ds \right\} \right) \left(b(t) \frac{(W(t) - c(t))^{1-\gamma} - \underline{c}^{1-\gamma}}{1-\gamma} \right) \middle| Y_{0} = i, W(0) = w \right]$$
(C.1)

Rearranging yields:

$$\begin{split} V(0,w,i) &= \sum_{t=0}^{T} e^{-\rho t} \mathbb{E} \left[\exp\left\{ -\int_{0}^{t} \mu(s) ds \right\} q_{Y_{t}}(t) \frac{c(t)^{1-\gamma}}{1-\gamma} \middle| Y_{0} = i, W(0) = w \right] \\ &+ e^{-\rho t} b(t) \mathbb{E} \left[\left(\exp\left\{ -\int_{0}^{t} \mu(s) ds \right\} - \exp\left\{ -\int_{0}^{t+1} \mu(s) ds \right\} \right) \frac{(W(t) - c(t))^{1-\gamma}}{1-\gamma} \middle| Y_{0} = i, W(0) = w \right] \\ &- \frac{c^{1-\gamma}}{1-\gamma} \left[q_{Y_{0}}(0) + b(0) + \sum_{t=1}^{T} e^{-\rho t} \mathbb{E} \left[\exp\left\{ -\int_{0}^{t} \mu(s) ds \right\} \left(q_{Y_{t}}(t) + b(t) - e^{\rho} b(t-1) \right) \middle| Y_{0} = i \right] \right] \\ &= \frac{1}{1-\gamma} \left[w^{1-\gamma} K_{0,i} - \underline{c}^{1-\gamma} \left[q_{Y_{0}}(0) + b(0) + \sum_{t=1}^{T} e^{-\rho t} \mathbb{E} \left[\exp\left\{ -\int_{0}^{t} \mu(s) ds \right\} \left(q_{Y_{t}}(t) + b(t) - e^{\rho} b(t-1) \right) \middle| Y_{0} = i \right] \right] \right] \end{split}$$

We can then calculate VSL in state *i* using the following formula:

$$VSL(i) = \frac{V(0, w, i) - b(0) \left(\frac{w^{1-\gamma} - \underline{c}^{1-\gamma}}{1-\gamma}\right)}{u_c (w c_{0,i}, q_i(0))}$$
(C.2)

The second term in the numerator of (C.2) is the utility at death (the bequest function). When the bequest motive is absent $(b(t) \equiv 0)$, the value function simplifies to:

$$V(0, w, i) = \frac{1}{1 - \gamma} \left[w^{1 - \gamma} K_{0, i} - \underline{c}^{1 - \gamma} \sum_{t=0}^{T} e^{-\rho t} \mathbb{E} \left[\exp \left\{ -\int_{0}^{t} \mu(s) ds \right\} q_{Y_{t}}(t) \middle| Y_{0} = i \right] \right]$$

discounted quality-adjusted life expectancy in state i

and the expression for VSL simplifies to Equation (9) from the main text.

Once one has calculated VSL, it is straightforward to calculate VSI:

Corollary C.2. The value of a marginal reduction in the probability of transitioning from state *i* to state *j* is equal to:

$$VSI(i,j) = VSL(i) - VSL(j)\frac{q_j(0)c_{0,j}^{-\gamma}}{q_i(0)c_{0,i}^{-\gamma}} = VSL(i) - \left(\frac{q_j(0)}{q_i(0)}\right) \left(\frac{c_{0,i}}{c_{0,j}}\right)^{\gamma} VSL(j)$$

Proof. See Appendix C.1

C.1 Proofs

Proof of Proposition C.1. The proof proceeds by induction on $t \le T$. For the base case t = T, note that $\overline{d}_i(t) = 1$, so that the first-order condition from the Bellman equation gives:

$$q_i(T)c(T)^{-\gamma} = b(T)(w - c(T))^{-\gamma}$$

Rearranging this first-order condition yields:

$$c(T) = \frac{w\left(\frac{q_i(T)}{b(T)}\right)^{\frac{1}{\gamma}}}{1 + \left(\frac{q_i(T)}{b(T)}\right)^{\frac{1}{\gamma}}} = w\left[\underbrace{1 + \left(\frac{b(T)}{q_i(T)}\right)^{\frac{1}{\gamma}}}_{c_{T,i}}\right]^{-1}$$

Hence, we obtain:

$$V(T, w, i) = \frac{w^{1-\gamma}}{1-\gamma} \left(q_i(T) c_{T,i}^{1-\gamma} + b(T) (1-c_{T,i})^{1-\gamma} \right)$$
$$= \frac{w^{1-\gamma}}{1-\gamma} \left[q_i(T)^{\frac{1}{\gamma}} + b(T)^{\frac{1}{\gamma}} \right]^{\gamma}$$

For the induction step, suppose the proposition is true for case t + 1. We have:

$$V(t, w, i) = \max_{c} \left\{ q_i(t) \frac{c^{1-\gamma}}{1-\gamma} + b(t) \overline{d}_i(t) \frac{(w-c)^{1-\gamma}}{1-\gamma} + e^{-\rho} \left(1 - \overline{d}_i(t)\right) \sum_{j=1}^{n} p_{ij}(t) \frac{K_{t+1,j}}{1-\gamma} \left[(w-c) e^{r(t,i,j)} \right]^{1-\gamma} \right\}$$

From the first-order condition we obtain:

$$q_{i}(t)c^{-\gamma} = b(t)\overline{d}_{i}(t)(w-c)^{-\gamma} + e^{-\rho}\left(1 - \overline{d}_{i}(t)\right)(w-c)^{-\gamma}\sum_{j=i}^{n} p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}$$

Rearranging yields:

$$q_{i}(t)c^{-\gamma} = (w-c)^{-\gamma} \left[\overline{d}_{i}(t)b(t) + (1-\overline{d}_{i}(t))e^{-\rho} \sum_{j=i}^{n} p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)} \right]$$

which implies:

$$q_{i}(t)^{-1/\gamma}c = (w-c)\left[\overline{d}_{i}(t)b(t) + (1-\overline{d}_{i}(t))e^{-\rho}\sum_{j=i}^{n}p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{-1/\gamma}$$

Rearranging further yields:

$$c = w \underbrace{\left[1 + \left(\frac{\left[\overline{d}_{i}(t) b(t) + \left(1 - \overline{d}_{i}(t)\right)e^{-\rho}\sum_{j=i}^{n} p_{ij}(t) K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]}{q_{i}(t)}\right)^{\frac{1}{\gamma}}_{c_{t,i}}\right]^{-1}_{c_{t,i}}}$$

Thus we obtain:

$$\begin{split} V(t,w,i) &= q_i(t) c_{t,i}^{1-\gamma} \frac{w^{1-\gamma}}{1-\gamma} + b(t) \overline{d}_i(t) \frac{w^{1-\gamma}}{1-\gamma} (1-c_{t,i})^{1-\gamma} + e^{-\rho} \left(1-\overline{d}_i(t)\right) \frac{w^{1-\gamma}}{1-\gamma} (1-c_{t,i})^{1-\gamma} \sum_{j=i}^n p_{ij}(t) K_{t+1,j} e^{r(t,i,j)(1-\gamma)} \\ &= \frac{w^{1-\gamma}}{1-\gamma} \left[q_i(t) c_{t,i}^{1-\gamma} + (1-c_{t,i})^{1-\gamma} \left[\overline{d}_i(t) b(t) + e^{-\rho} \left(1-\overline{d}_i(t)\right) \sum_{j=i}^n p_{ij}(t) K_{t+1,j} e^{r(t,i,j)(1-\gamma)} \right] \right] \end{split}$$

Note that we can write:

$$c_{t,i} = \frac{q_i(t)^{1/\gamma}}{q_i(t)^{1/\gamma} + \left[\overline{d}_i(t)b(t) + (1 - \overline{d}_i(t))e^{-\rho}\sum_{j=i}^n p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{1/\gamma}}$$

which implies:

$$1 - c_{t,i} = \frac{\left[\overline{d}_i(t)b(t) + (1 - \overline{d}_i(t))e^{-\rho}\sum_{j=i}^n p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{1/\gamma}}{q_i(t)^{1/\gamma} + \left[\overline{d}_i(t)b(t) + (1 - \overline{d}_i(t))e^{-\rho}\sum_{j=i}^n p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{1/\gamma}}$$

Plugging this into the expression for V(t, w, i) from above yields:

$$V(t,w,i) = \frac{w^{1-\gamma}}{1-\gamma} \frac{q_i(t)^{1/\gamma} + \left[\overline{d}_i(t)b(t) + (1-\overline{d}_i(t))e^{-\rho}\sum_{j=i}^n p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{1/\gamma}}{\left[q_i(t)^{1/\gamma} + \left[\overline{d}_i(t)b(t) + (1-\overline{d}_i(t))e^{-\rho}\sum_{j=i}^n p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{1/\gamma}\right]^{1-\gamma}} = \frac{w^{1-\gamma}}{1-\gamma} \left[q_i(t)^{1/\gamma} + \left[\overline{d}_i(t)b(t) + (1-\overline{d}_i(t))e^{-\rho}\sum_{j=i}^n p_{ij}(t)K_{t+1,j}e^{r(t,i,j)(1-\gamma)}\right]^{1/\gamma}\right]^{\gamma}$$

Proof of Corollary C.2. The proof follows immediately from the expression for VSI, given by Equation (10), and from noting that $u_c(c_i(0), q_i(0)) = q_i(0)c_{i,0}^{-\gamma}w^{-\gamma}$.