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RESEARCH

# Wealth, prevention, and longevity: Integrating health into portfolio decisions

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## Abstract

This paper presents a novel framework for integrating preventive health expenditures into a lifetime portfolio selection model under uncertain lifetimes. Building on financial portfolio optimization and actuarial mortality modeling, we examine how health investments influence individual longevity and financial decisions. In our model, age at death is treated as a random variable, and individuals allocate a fraction of their wealth to prevention in order to reduce mortality risk and extend life expectancy. This endogenous link between health spending and survival allows us to explore the dynamic interplay between wealth accumulation, longevity, and health-related behavior over the life cycle. Through numerical simulations, we illustrate how optimal prevention strategies vary systematically by gender, age, and country-specific mortality profiles. The results highlight how personal characteristics and demographic factors shape the trade-offs between consumption, investment, and health. Our findings offer valuable insights for both individual financial planning and the design of public health policies in an era of increasing longevity and growing economic-health interdependence.

**Keywords** Portfolio choice · Lifetime uncertainty · Health prevention · Epidemiological transition

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## 1 Introduction

Population health and economic development are closely interconnected. Economic development, in fact, affects epidemiological transitions, that is, changing patterns of disease, from acute infectious and deficiency diseases to chronic non-communicable diseases, which are the most prevalent causes of death in high-income countries (Gulliford 2003). In turn, the epidemiological transition has been shown to lead an economy to switch to a modern growth regime (Morand 2004). Although acute diseases are more relevant for early-life mortality, chronic conditions impose a higher burden of disease throughout the life cycle, as discussed in Gulis et al. (2025). Recent evidence shows that communicable and non-communicable diseases interact through biological ageing processes. Acute infections can accelerate inflammatory damage and contribute to the accumulation of health deficits, while higher frailty increases both susceptibility to and the severity of infectious episodes (Strulik and Grossmann 2024). This two-way relationship suggests that the traditional distinction between infectious and chronic conditions may underestimate the extent to which ageing dynamics shape disease risks across the life course.

In fact, ageing and longevity are closely related to health and are cross-cutting topics that intersect, for example, the financial sustainability and financial well-being of individuals. A straightforward but meaningful indicator that illustrates developments in longevity is the average number of years people can expect to live at birth if experiencing the same mortality conditions throughout their lifetime, namely, life expectancy at birth. This indicator has increased by 3.7 years between 2002 and 2019 in the European Union and is estimated to be 81.4 years in 2023[1]. Although a collective achievement, an increase in life expectancy implies greater pressure on social security expenditures and public pension systems and increases the risk of deprivation among older people.

What makes longevity a matter of high concern is its random nature, with uncertain evolution over time. Consequently, longevity risk adds complexity to a wide range of forward-looking economic decisions that fundamentally depend on individuals' random lifetime, from consumption choices to prevention and healthcare decisions. As stressed by Hall (2010), forward-looking behavior is at the core of economics and inherently deals with uncertainty. The dynamic programs proposed in the literature to describe and govern intertemporal choices on consumers' resource allocation typically account for stochastic lifetime. For example, the life-cycle model of savings and consumption, as developed by Fisher (1930) and refined by Modigliani and Brumberg (1954) and Modigliani (1986), has allowed for a stochastic lifetime since the contribution of Yaari (1964, 1965). Yaari (1965) assumed that the length of the time horizon over which to make plans for the future, namely the residual lifetime, is a ran-

[1] https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Mortality_and_life_expectancy_statistics.

dom variable with a known probability distribution, instead of a fixed number, thereby making the individual's utility function itself random. Yaari's seminal contribution paved the way to numerous dynamic allocation models regarding the force of mortality as a determinant of individual choices about optimal consumption, investment and, in many cases, life insurance (Merton 1971; Richard 1975; Cocco and Gomes 2012; Huang et al. 2012; Maggistro et al. 2025), at the intersection with insurance economics and actuarial science. Understanding longevity risk and individuals' behavior in hedging against this risk can involve exploring the role of health, which is a relevant and meaningful variable both in the medical and actuarial literature (Carannante et al. 2024). In addition, there is a wide consensus on the association between health expenses and longevity, from an economic perspective (Zhao 2015).

In this spirit, governments, international and local organizations, academia, the private sector, and civil society are committed to achieving a global goal, aligned with the Sustainable Development Goals and framed within the UN Decade of Healthy Ageing (2021-2030), that is a longer and healthier life for everyone [2]. This involves also health promotion (e.g., people's empowerment through health literacy) and disease prevention (e.g., primary and secondary prevention) to reduce the incidence and the development of ageing-related diseases, including several chronic diseases that, as already mentioned, are progressively representing the leading causes of disability and death among the elderly (Guo et al. 2022; Choi et al. 2019). In 2022 in the EU, women at birth could expect to live, on average, 62.8 years in a healthy condition, namely without any activity limitation, thus 80.1% of their total life expectancy; in the same year the number of healthy life years at birth was estimated to be, for men, 62.4 years, namely 75.4% of their total life expectancy [3]. The question of whether the extra years of life resulting from increased longevity are spent in good or poor health is of crucial relevance across all the domains of economics. An example in this respect is the positive association between health status and income, which is a key measure of the economic and social well-being of individuals, especially as people approach silver ages and become more vulnerable [4]. Investing in healthy ageing and preventive health can sustain the economic and social systems burdened by population ageing, especially in the aftermath of COVID-19 [5].

As shown in the literature, in the era of ageing and global health challenges, health promotion, prevention, and preparedness deserve crucial attention in policymaking, either in relation to pandemic diseases or other types of diseases. For instance, being prepared to manage epidemics by balancing health benefits and economic costs is very important to minimize their social cost (La Torre et al. 2020, 2021) and to assess the impact of policies and strategies on disease dynamics (see, e.g., Anderson et al. (2010);

[2] https://www.who.int/initiatives/decade-of-healthy-ageing

[3] https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Healthy_life_years_statistics

[4] https://ec.europa.eu/eurostat/statistics-explained/index.php?title=Disability_statistics_-_poverty_and_income_inequalities

[5] https://www.oecd.org/en/publications/investing-in-health-systems-to-protect-society-and-boost-the-economy_d0aa9188-en.html

Gersovitz and Hammer (2004); Goldman and Lightwood (2002); Goenka et al. (2014). Likewise, preventing chronic diseases may support social welfare and enhance health equity. Health prevention can deliver long-term benefits, such as work productivity, economic growth, and social equity (Wang and Wang 2021; Wouterse et al. 2025), and thus contribute to enhanced resilience. As stressed in OECD/WHO (2015), adopting an economic perspective on health promotion and disease prevention entails not only quantifying costs, but also understanding how policy strategies can influence health-related choices among individuals and reduce inequalities in their health outcomes. In this context, individuals' choices and behaviors can play a role in the construction of improved health outcomes at the aggregate level.

Likewise, at the microeconomic level, integrating health strategies into life-cycle decision-making may improve individuals' financial well-being in later life. Several economic models of consumer behavior explicitly account for the dynamics of health.

A line of research deals with models of the demand for longevity and health. These intertemporal models are characterized by a planning horizon that is a choice variable and potential lifetime resources that are endogenous outcomes. A pivotal contribution in this respect is that of Grossman (1972), who assumed that individuals hold an initial stock of health that depreciates over time and that can be enhanced by making costly investments in it. In this framework, consumers' choices progressively affect the level of health and the length of life as well, with death occurring when health drops below a given threshold. Furthermore, according to Grossman (1972), individuals' demand for health is driven by consumption and investment motives, since an increase in the health stock leads to a higher utility and to a larger amount of time available to earn income. This work presents some limitations related, for instance, to the implied deterministic nature of death (see Ehrlich and Chuma 1990 for further remarks). Ehrlich (2000) viewed uncertain life expectancy as partially the result of individuals' efforts to self-protect against mortality and morbidity risks; this aspect can partly explain the heterogeneity that can be typically observed in life expectancies among individuals (see also Ehrlich and Yin 2005). Bolin and Caputo (2020) devised a demand-for-health model, where the evolution of the health stock is stochastic, due to the uncertain impact of health investment and the stochasticity of the rate of health depreciation. In this framework, individuals can directly affect the stock of health by making precautionary health investments, thereby indirectly influencing the rate at which health depreciates over time or, in other terms, the rate at which health shocks occur.

The combination of financial and health economics perspectives provides a more comprehensive explanation of the relationship between health and wealth, with allocations and welfare. For example, in this context, Hugonnier et al. (2013) developed a dynamic model to jointly determine consumption, portfolio, health investment, and insurance coverage, consistent with the patterns that could be observed empirically. Guasoni and Huang (2019) analyze a dynamic problem for optimizing investment, consumption, and healthcare spending, under the assumption that individuals make constant investments in healthcare and, in doing so, they directly impact on the natural

growth of mortality, by making it slow down compared to what is prescribed by the endogenous mortality law chosen to govern the mortality phenomenon (Gompertz law). At the same time, individuals indirectly increase the utility of consumption as a result of the gain in life expectancy. A further relevant contribution is that of Ferrari and Zhu (2023).

A complementary line of research shows that mortality risk rises systematically with the accumulation of health deficits, making deficit dynamics an essential component of survival modeling (Strulik and Hansen 2025). Within this framework, preventive health spending becomes economically relevant because it can slow the accumulation of deficits over time, thereby reducing mortality risk and altering life-cycle choices regarding wealth allocation. This mechanism provides a rationale for studying prevention within a unified economic model in which health expenditures influence longevity and the horizon over which financial decisions unfold.

The key question we address in this paper concerns the development of a modeling framework to analyze how health expenditures impact mortality risk, reduce the incidence and spread of diseases, and enhance financial wealth accumulation. As explained above, this mechanism has been theorized and/or empirically verified in previous contributions. The modeling framework we propose aims to build a bridge between financial and actuarial models that are well established in the literature, for instance, because of their proven theoretical suitability, analytical tractability, and clear interpretation. In particular, in this unified framework, we revisit Merton's canonical portfolio selection and the Gompertz law of mortality to study a life-cycle decision problem in which agents choose a constant fraction of their wealth to allocate to preventive healthcare and, at the same time, maximize their bequest by deciding the optimal share of wealth to assign to risky and risk-free assets. Consistent with this mortality specification, we later relate the hazard rate to deficit accumulation, offering a biological interpretation of its sensitivity to preventive efforts. Our aim is not only to construct a rigorous mathematical framework in which to develop the dynamic optimization problem under consideration, but also to shed light on relevant implications through empirical implementation. Indeed, empirical analysis illustrates how optimal prevention expenditure levels can be derived for different populations, by gender, age, and country, due to heterogeneity in the underlying mortality patterns. For example, we show the extent to which optimal prevention expenditure levels are higher for males than for females, under our working hypotheses. Furthermore, the analysis highlights inherent trade-offs, as greater longevity achieved through prevention comes at the cost of lower investment returns, but extends the investment horizon. Although derived within a simplified mathematical description of the relationship between health and mortality outcomes, our numerical outcomes can be interpreted coherently with the existing evidence on some phenomena, such as the male–female health–mortality paradox, and may be considered informative for life course health strategies.

From a mathematical point of view, the model takes the form of a stochastic optimal control problem. The solution is obtained by formulating the Hamilton-Jacobi-Bellman equation (hereafter HJB) and assuming a sufficiently regular form for the

value function, which is then shown to solve this partial differential equation (PDE). The decision to allocate part of one's wealth to preventive healthcare introduces a new source of randomness into the problem (independent of the risky asset's dynamics) and is linked to the decision-maker's remaining lifetime. From a probabilistic perspective, we control a parameter of an exponential distribution that represents this remaining lifetime. By adopting the Gompertz model, we connect to the actuarial literature and assume that investment in preventive healthcare has a direct effect on the hazard rate. We present the results by systematically introducing the assumptions employed. The analytical formulation we propose is sufficiently simple to be understood by researchers working in actuarial mathematics while also being highly versatile and adaptable to different demographic cohorts.

The paper is structured as follows. Section 2 summarizes Merton's model and includes a review of the literature on the extensions of the Merton problem from different points of view. Moreover, it also analyzes the case in which it focuses only on optimizing the bequest function by explicitly determining the optimal investment strategy. In Sect. 3, we present our model in which age at death is a random variable and a cost of health prevention is included to impact the investor's life expectancy and thus age at death. In Sect. 4, we explicitly solve our control problem by characterizing both the optimal investment strategy and the value function by evaluating the corresponding objective functional for each value of the constant fraction of wealth spent on preventive healthcare. Section 5 formalizes the link between mortality and the accumulation of health deficits, clarifying how changes in preventive spending translate into variations in the hazard rate. In Section 6, we carry out an empirical implementation of the model. In particular, the optimal fraction of wealth worth investing in preventive healthcare is calculated for different countries and by gender. Moreover, the impact of preventive healthcare on life expectancy is also analyzed according to the starting age, gender, and country of origin of the investors. Section 7, as usual, presents the concluding remarks and highlights directions for future research.

## 2 Merton portfolio problem

Merton's model is an intertemporal portfolio choice problem. In its classical formulation (Merton 1969, 1971), it is defined as a stochastic optimal control model, and it can be solved in a closed-form solution by means of the Hamilton–Jacobi–Bellman (HJB) equation. Its analytical tractability is due to the following assumptions: a perfect and frictionless market, normally distributed asset returns, continuous trading, and no default risk for the issuer of the financial assets. Moreover, the following conditions hold:

- the investor chooses its portfolio by selecting one risky asset and a risk-free asset;
- the investor can consume any fractional amount of wealth at any time;
- the investor aims at maximizing lifetime utility for consumption and bequest;
- the consumption utility $u : \mathbb{R}^+ \to \mathbb{R}$ is a strictly concave $C^2$ (twice differentiable with continuous derivatives) function, while the bequest function $b(X(T))$ is strictly concave in the wealth $X(T) \in \mathbb{R}^+$, where $T$ is the exogenous finite hori-

zon denoting the age of death of the investor who is $h$ years old at the beginning of the investment period.

The price of the risky asset evolves as

$$\begin{cases} dP(t) = P(t)\left(\mu dt + \sigma dW(t)\right), & t \in [h, T] \\ P(h) > 0 \text{ given}, \end{cases} \tag{1}$$

where $\mu$ is the instantaneous expected return, $\sigma > 0$ is the volatility and $W(t)$ is a Brownian motion defined on $[h, T]$, with $W(h) = 0$ a.s. The price of the risk-free asset is

$$\begin{cases} dB(t) = \rho B(t)dt, & t \in [h, T] \\ B(h) > 0 \text{ given}, \end{cases} \tag{2}$$

where $\rho$ stands for the risk-free interest rate, and we assume that the risk premium condition $\mu > \rho$ holds.

Let $w(t)$ now be the proportion of the portfolio allocated at time $t$ to the risky asset, so that $1 - w(t)$ represents the portion invested in the risk-free asset. Finally, introducing consumption $c(t) \geq 0$, the dynamics of wealth $X(t)$ can be written as

$$\begin{cases} dX(t) = \left[X(t)\left((\mu - \rho)\, w(t) + \rho\right) - c(t)\right] dt + \sigma X(t) w(t) dW(t), \\ X(h) > 0 \text{ given}. \end{cases} \tag{3}$$

Therefore, Merton's portfolio problem is described as follows:

$$\begin{aligned} \max_{\{c,w\} \in \mathcal{A}} \; & J(h, X(h), c(\cdot), w(\cdot)) := \mathbb{E}\left[\int_h^T u(c(t))\, dt + b\left(X(T)\right)\right] \\ & dX(t) = \left[X(t)\left((\mu - \rho)\, w(t) + \rho\right) - c(t)\right]dt + \sigma X(t) w(t) dW(t) \\ & X(h) > 0 \text{ given} \end{aligned} \tag{4}$$

where $\mathbb{E}$ is the expectation operator and $\mathcal{A}$ is the set of all admissible investment and consumption strategies. Hence, Eq. 4 is formulated as an optimal stochastic control problem whose solution is obtained using the HJB equation. Then, let

$$V(h, X(h)) = \max_{\{c,w\} \in \mathcal{A}} J(h, X(h), c(\cdot), w(\cdot)), \tag{5}$$

be the value function; it satisfies the following HJB equation:

$$\begin{cases} \partial_t V + \max\limits_{c(t), w(t)} \left\{ u(c(t)) + \left[X(t)\left((\mu - \rho)\, w(t) + \rho\right) - c(t)\right] \partial_X V + \dfrac{1}{2}\sigma^2 X(t)^2 w(t)^2 \partial_X^2 V \right\} = 0 \\ V(T, X(T)) = b(X(T)), \end{cases}$$

where $\partial_t V$ is the first-order derivative of $V$ with respect to $t$, while $\partial_X V$ and $\partial_X^2 V$ are, respectively, the first-order and the second-order derivatives of $V$ with respect to $X$.

Various extensions of the Merton problem have been proposed in the literature. Some of these yield closed-form solutions, while others rely on numerical methods. Based on the additional assumptions introduced into the model, three main research streams can be identified. The first stream focuses on utility functions within the Constant Relative Risk Aversion (CRRA) class. In this context, notable contributions include Zariphopoulou (1999), who extends the Merton framework to accommodate nonlinear coefficients; Liu (2007), who develops a unified framework that incorporates stochastic interest rates, asset returns, and volatility; Bo and Capponi (2016), who integrate contagion risk, and La Torre and Maggistro (2024), who investigate a multi-agent extension of Merton's model within the framework of dynamic game theory. The second stream abstracts from consumption decisions and concentrates solely on maximizing terminal wealth, with the primary aim of maintaining analytical tractability. Contributions in this direction include Kim and Omberg (1996), where the risk premium is modeled as an Ornstein–Uhlenbeck process; Brennan and Xia (2002), who incorporate stochastic inflation, and Ma et al. (2020), who provide a numerical solution to the Merton problem under general utility functions. Finally, in the last strand of this research, the investment horizon is assumed to be infinite. This assumption reduces the HJB equation to a nonlinear ordinary differential equation, which facilitates the derivation of analytical solutions. See, for instance, Fleming and Pang (2004) and Bo et al. (2010).

More recently, Ma and Zhu (2022) provides a closed-form solution of the Merton problem defined on a finite horizon and with CARA utility function without any assumption mentioned above.

In this paper, we position ourselves within the second research stream; hence, we only focus on maximizing the bequest, which will take logarithmic form. The model is thus reformulated as:

$$\begin{aligned} &\max_{w(t)} J(h, X(h), w(\cdot)) := \mathbb{E}\left[b\left(X(T)\right)\right] \\ &\quad dX(t) = \left[X(t)\left(\left(\mu - \rho\right) w(t) + \rho\right)\right]dt + \sigma X(t) w(t) dW(t) \\ &\quad X(h) > 0 \text{ given} \end{aligned} \tag{6}$$

where $V(h, X(h)) = \max_{w(t)} J(h, X(h), w(\cdot))$, is the corresponding value function. By assuming that

$$\begin{aligned} &V(t, X(t)) = \ln(X(t)) + A(t), \quad A(t) \in \mathbb{R}, \\ &b(X(T)) = \ln(X(T)), \end{aligned} \tag{7}$$

and by substituting Eq. 7 in the corresponding HJB equation

$$\begin{cases} \partial_t V + \max_{w(t)} \left\{ \left[X(t)\left(\left(\mu - \rho\right) w(t) + \rho\right)\right] \partial_X V + \frac{1}{2}\sigma^2 X(t)^2 w(t)^2 \partial_X^2 V \right\} = 0, \\ V(T, X(T)) = b(X(T)), \end{cases}$$

one gets that $A(t)$ satisfies the following

$$\begin{cases} A'(t) = -\left[\dfrac{(\mu-\rho)^2}{2\sigma^2} + \rho\right], \\ A(T) = 0, \end{cases}$$

whose solution is

$$A(t) = \left[\frac{(\mu-\rho)^2}{2\sigma^2} + \rho\right](T-t).$$

Then, the resulting decision rule for portfolio selection $w^*(t)$ is

$$w^*(t) = \frac{(\mu-\rho)}{\sigma^2}.$$

## 3 Uncertain lifetimes and health prevention

In this section, we extend the previous model Eq. 6 by considering the investor's age at death as a random variable $\tau$, rather than an exogenously fixed quantity $T$, and by introducing a cost for health prevention $qX(t) \geq 0, q \in [0, 1]$, incurred by the investor to improve its health. This cost, which is a constant fraction of wealth, does not generate instantaneous utility but influences the investor's life expectancy and consequently its death age $\tau$. Furthermore, as it is common in the literature, we assume the existence of a maximal age $\widetilde{T}$ beyond which no individual will be alive. Accordingly, the support of $\tau$ is the interval $[h, \widetilde{T}]$. The investor's optimization problem is thus formulated over a time horizon that is bounded from below by its initial age $h$. Such an age defines the cohort to which the investor belongs (the birth having occurred $h$ years before). Then, for every $q \in [0, 1]$, the portfolio problem can be written as:

$$\begin{aligned} &\max_{w(t)} \ \mathbb{E}\left[b\left(X(\tau)\right)\right] \\ &\quad dX(t) = \left[X(t)\left(\left(\mu-\rho\right)w(t) + \rho - q\right)\right]dt + \sigma X(t) w(t) dW(t) \\ &\quad X(h) > 0 \text{ given} \end{aligned} \tag{8}$$

where $\tau$ and $W(t)$ are independent random variables.

Preventive healthcare can help improve survival outcomes, for instance, thanks to screening, diagnosis, and early detection of diseases, especially in high-risk individuals. In particular, the importance of early diagnosis is debated in various fields, with objectives that range from raising public awareness towards health promotion and personalized strategies to control modifiable risk factors, to improving current disease detection methods, e.g., through the adoption of informatics and AI-driven techniques (Abbate et al. 2020; Whitaker 2020; Jin et al. 2025). To the best of our knowledge, there is no unique and consistent quantitative measure in the literature of the improving effect of the investment in preventive healthcare on survival.

In our analysis, we adopt the Gompertz law (Gompertz 1825) to model the average human mortality. We then introduce the effect of health prevention on mortality through a reparameterization of the classical Gompertz law. Assuming that $h$ is the initial individual's age, the classical Gompertz force of mortality at any age $t \in [h, \widetilde{T}]$ is given by

$$\lambda(t) = \beta e^{\gamma(t-h)}, \tag{9}$$

where the parameter $\beta > 0$ denotes the mortality level at age $h$ and the parameter $\gamma > 0$ indicates the rate of ageing. As stressed by Milevsky (2020), the Gompertz law of mortality, although more than two centuries have passed since its formulation, is still widely deployed in many fields, including demography and actuarial science. As shown in Eq. 9, the distinctive feature of this law is the exponential increase, at a constant rate, in the force of mortality with age. Such an age pattern of mortality has been empirically verified for adult-age populations in most countries of the world. The choice of the Gompertz law to model standard mortality over age is also consistent with Guasoni and Huang (2019). As we will show in Sect. 5, the Gompertz law of mortality can be expressed in such a way as to explicitly account for the increase in the health deficits, thereby capturing the contribution of frailty to mortality dynamics. In the remainder of this section, we illustrate how, within the mathematical structure of the general Gompertz law of mortality, we formalize the effect of health prevention on mortality, while in Sect. 5, we address in more detail the link between health deficits and mortality, and contextualize it in our modeling framework.

To reparameterize the force of mortality Eq. 9, we introduce a function of the initial mortality level $\beta$ and of the fraction of wealth invested in health prevention that is devised to describe the mechanism by which health investing may trigger downward shifts in the mortality curve. We point out that we propose a stylized mathematical relationship, which simplifies the much more complex and non-obvious links between genetic and mortality risk factors with mortality outcomes. Therefore, we consider the following reparameterized version of the Gompertz force of mortality at any age $t \in [h, \widetilde{T}]$:

$$\lambda(t, q) = g(\beta, q) e^{\gamma(t-h)}, \tag{10}$$

where the function $g(\beta, q)$ represents the mortality level at age $h$. If $q = 0$, that is, when there is no health prevention, Eq. 10 results in the classical Gompertz force of mortality, with $\lambda(t, 0) = \beta e^{\gamma(t-h)}$; this implies that $g(\beta, 0) = \beta$. Moreover, we expect that as $q$ increases, $g(\beta, q)$ will decrease, thus $\frac{\partial g}{\partial q}(\beta, q) < 0$.

Furthermore, we can characterize the survival function as

$$\begin{aligned} S(t, q) &= \exp\left[ - \int_h^t \lambda(s, q)\, ds \right] \\ &= \exp\left[ - \int_h^t g(\beta, q)\, e^{\gamma(s-h)} ds \right] = \exp\left[ -g(\beta, q)\, \frac{(e^{\gamma(t-h)} - 1)}{\gamma} \right]. \end{aligned} \tag{11}$$

By definition, $S(h,q)=1$. We can also assume $S(\widetilde{T},q)=0$, since, if $\widetilde{T}$ is the maximal age beyond which no one will be alive, then $\int_{\widetilde{T}}^{x}\lambda(s,q)ds=o(1/x),\forall x\geq\widetilde{T}$ (Olivieri and Pitacco 2011). Finally, we recall that the death age of the investor, i.e., the random variable $\tau$, and we introduce its probability density function, $f(t,q)$, which describes the curve of deaths at age $t$ as a consequence of the health prevention,

$$\begin{aligned} f(t,q) &= S(t,q)\lambda(t,q) \\ &= g(\beta,q)\exp\left[\gamma(t-h)-g(\beta,q)\,\frac{(e^{\gamma(t-h)}-1)}{\gamma}\right]. \end{aligned} \tag{12}$$

Within this framework, where the force of mortality Eq. 10 is deterministic, it is possible to rewrite the control problem in Eq. 8 computing the expected value with respect to the probability density function of $\tau$, $f(t,q)=S(t,q)\lambda(t,q)$,

$$\begin{aligned} &\max_{w(t)}\ \mathbb{E}\left\{\int_h^{\widetilde{T}} S(t,q)\lambda(t,q)b\left(X(t)\right)dt\right\} \\ &\quad dX(t)=\left[X(t)\left(\left(\mu-\rho\right)w(t)+\rho-q\right)\right]dt+\sigma X(t)w(t)dW(t) \\ &\quad X(h)>0 \text{ given.} \end{aligned} \tag{13}$$

Note that the bequest function is no longer considered only at the age of death, but over the entire age range, $[h,\widetilde{T}]$, and its value depends on the probability of death mitigated by investment in preventive medical care.

## 4 Equilibrium outcomes

In the previous section, we introduced the stochastic optimal control problem associated with the optimal investment strategy in the presence of health prevention expenditure. Note that the control problem Eq. 13, derived at the end of the previous section, does not depend on the specific reparameterization of the hazard rate given in Eq. 10. Nonetheless, this particular form is of analytical interest, as it allows for an explicit calculation of the density of the random variable $\tau$.

In the first part of this section, we retain the more general expression for the density of $\tau$, namely $S(t,q)\lambda(t,q)$. Subsequently, we will specify when the form chosen in Eq. 10 for the hazard rate becomes essential.

The main assumption that allows us to prove the next result is that the bequest function $b$ is logarithmic. This allows the derivation of a tractable HJB equation that can be solved in closed form.

**Proposition 1** *Assume that the function b in problem Eq. 13 takes the form:*

$$b(X(t))=\ln(X(t)). \tag{14}$$

*If we denote by $V(t, X(t))$ the value function associated with the problem Eq. 13, then the HJB equation for this stochastic optimal control problem is the following:*

$$\begin{cases} \partial_t V + \max\limits_{w(t)} \Big\{ S(t,q)\lambda(t,q)\ln(X(t)) + [X(t)((\mu-\rho)w(t)+\rho-q)]\partial_X V \\ \qquad\qquad + \frac{1}{2}\sigma^2 X(t)^2 w(t)^2 \partial_X^2 V \Big\} = 0 \\ V(\widetilde{T}, X(\widetilde{T})) = 0. \end{cases} \tag{15}$$

*Under these assumptions, $V$ is a classical solution of the HJB Eq. 15 (i.e. $V \in C^{1,2}([h, \widetilde{T}] \times \mathbb{R}^+)$) and then the optimal share of wealth invested in the risky asset that solves the problem Eq. 13 is given by*

$$w^*(t) = \frac{(\mu-\rho)}{\sigma^2}. \tag{16}$$

***Proof*** The Hamiltonian $H$ associated with the problem Eq. 13 is:

$$H(X(t), w(t)) = S(t,q)\lambda(t,q)\ln(X(t)) + [X(t)((\mu-\rho)w(t)+\rho-q)]\,\partial_X V + \frac{1}{2}\sigma^2 X(t)^2 w(t)^2 \partial_X^2 V.$$

This gives the HJB Eq. 15. Now, by assuming that the value function has the following form

$$V(t, X(t)) = S(t,q)\,[C(t)\ln(X(t)) + F(t)]\,, \tag{17}$$

we apply the first-order condition, i.e., $\partial H/\partial w = 0$, from which we get the optimal control

$$w^*(t) = \frac{(\mu-\rho)}{\sigma^2}. \tag{18}$$

Now, by inserting the expressions of Eqs. 17–18 into Eq. 15, we get that $C(t)$ and $F(t)$ must satisfy the following system

$$\begin{cases} C'(t) = -\lambda(t,q)\,(C(t)+1)\,, \\ F'(t) = -\lambda(t,q)F(t) - \left(\dfrac{(\mu-\rho)^2}{2\sigma^2} + \rho - q\right) C(t), \end{cases} \tag{19}$$

with $C(t) > 0$, $\forall\, t \in [h, \widetilde{T})$, and with terminal conditions $C(\widetilde{T})$, $F(\widetilde{T})$ obtained by evaluating the value function $V$ in Eq. 17 at $\widetilde{T}$ and by imposing it equal to 0. Since $S(\widetilde{T}, q) = 0$, $C(\widetilde{T})$ and $F(\widetilde{T})$ could assume every real values. We then consider $C(\widetilde{T}) = k > 0$ and $F(\widetilde{T}) = \widetilde{k}$ for $k, \widetilde{k} \in \mathbb{R}$. Since the system Eq. 19 admits a unique

continuously differentiable solution for all $k > 0, \widetilde{k} \in \mathbb{R}$, then function $V$ in Eq. 17 stays in the space $C^{1,2}([h, \widetilde{T}] \times \mathbb{R}^+)$, hence it solves the HJB Eq. 15 in the classical way. Then it is the optimal value function for the control problem Eq. 13 with the bequest function as defined in Eq. 14 (see Theorem 25.7, p. 343 in Björk 2005). □

Note that, since the wealth dynamics in the problem Eq. 13 is governed by a geometric Brownian motion, there is a closed-form solution, allowing the optimal value function $V$ to be calculated directly by evaluating the corresponding functional objective. Let denote by $X^*(t)$ the optimal wealth, i.e., the wealth solving the problem Eq. 13 with logarithmic bequest Eq. 14 and with $w^*(t)$, the optimal proportion of the portfolio allocated at time $t$ to the risky asset given in Eq. 16. Then,

$$X^*(t) = X(h) \exp\left\{\left(\frac{(\mu-\rho)^2}{2\sigma^2} + \rho - q\right)(t-h) + \frac{\mu-\rho}{\sigma}(W(t) - W(h))\right\}, \tag{20}$$

where $W(h) = 0$. By inserting Eq. 20 into the bequest function $b(X^*(t)) = \ln(X^*(t))$ and the latter in the functional

$$\mathbb{E}\left\{\int_h^{\widetilde{T}} S(t,q)\lambda(t,q)\, b\left(X^*(t)\right) dt\right\},$$

of the control problem Eq. 13, we get

$$\mathbb{E}\left\{\int_h^{\widetilde{T}} S(t,q)\lambda(t,q)\left[\ln(X(h)) + \left(\frac{(\mu-\rho)^2}{2\sigma^2} + \rho - q\right)(t-h) + \frac{\mu-\rho}{\sigma}W(t)\right] dt\right\}. \tag{21}$$

By applying Fubini's theorem, it is possible to average under the sign of the integral in Eq. 21, hence the value function $V$ takes the following expression:

$$\begin{aligned} V(h, X(h)) &= \int_h^{\widetilde{T}} S(t,q)\lambda(t,q)\left[\ln(X(h)) + \left(\frac{(\mu-\rho)^2}{2\sigma^2} + \rho - q\right)(t-h)\right] dt \\ &= \ln(X(h)) + \int_h^{\widetilde{T}} S(t,q)\lambda(t,q)\left[\left(\frac{(\mu-\rho)^2}{2\sigma^2} + \rho - q\right)(t-h)\right] dt, \end{aligned} \tag{22}$$

since $\mathbb{E}(W(t)) = 0$ and $\int_h^{\widetilde{T}} S(t,q)\lambda(t,q)dt = \int_h^{\widetilde{T}} f(t,q)dt = 1$.

We observe that Eq. 22 does not depend on the specific form chosen for the function $\lambda(t, q)$, and can be derived in full generality by representing the density of $\tau$ as the product $S(t, q)\lambda(t, q)$. It depends only on the choice of the bequest function. In the next result, we rely crucially on assumption Eq. 10, which enables us to derive an explicit and tractable analytical form for the density of $\tau$, as previously presented in Eq. 12.

The following proposition shows how to explicitly compute the integral that appears in Eq. 22.

**Proposition 2** *Let the hazard function in the model be defined as in Eq. 10, i.e.,*

$$\lambda(t, q) = g(\beta, q)e^{\gamma(t-h)}. \tag{23}$$

*Then, the value function in Eq. 22 can be written as follows:*

$$V(h, X(h)) = \ln(X(h)) + \frac{(r-q)}{\gamma} \exp\left[\frac{g(\beta, q)}{\gamma}\right]\left\{-\exp\left[-\frac{g(\beta, q)}{\gamma}e^{\gamma(\widetilde{T}-h)}\right]\gamma\,(\widetilde{T}-h)\right.$$
$$\left. - E_1\left(\frac{g(\beta, q)}{\gamma}e^{\gamma(\widetilde{T}-h)}\right) + E_1\left(\frac{g(\beta, q)}{\gamma}\right)\right\}, \tag{24}$$

*where* $E_1(x) = \int_x^{+\infty}(e^{-z}/z)\,dz$ *is the exponential integral.*

***Proof*** Let us compute the integral in Eq. 22. Set $r = ((\mu-\rho)^2/2\sigma^2)+\rho$ and substitute the expressions of $\lambda(t, q)$ and $S(t, q)$, respectively, given in Eqs. 10 and 11.

$$\int_h^{\widetilde{T}} S(t, q)\lambda(t, q)\left[\left(\frac{(\mu-\rho)^2}{2\sigma^2}+\rho-q\right)(t-h)\right]dt = \int_h^{\widetilde{T}} S(t, q)\lambda(t, q)\,(r-q)\,(t-h)dt$$
$$= g(\beta, q)(r-q)\int_h^{\widetilde{T}} \exp\left[\gamma(t-h) - g(\beta, q)\,\frac{(e^{\gamma(t-h)}-1)}{\gamma}\right](t-h)\,dt.$$

Now, let us consider the following change of variable with $u = t - h$. It yields

$$= g(\beta, q)(r-q)\int_0^{\widetilde{T}-h} \exp\left[\gamma u - g(\beta, q)\,\frac{(e^{\gamma u}-1)}{\gamma}\right]u\,du$$
$$= g(\beta, q)(r-q)\exp\left[\frac{g(\beta, q)}{\gamma}\right]\int_0^{\widetilde{T}-h} e^{\gamma u}\exp\left[-g(\beta, q)\,\frac{e^{\gamma u}}{\gamma}\right]u\,du.$$

We apply a second change of variable by setting $z = g(\beta, q)e^{\gamma u}/\gamma$; then

$$dz = g(\beta, q)e^{\gamma u}du, \quad du = \frac{dz}{\gamma z}, \quad \gamma u = \ln\left(\frac{\gamma z}{g(\beta, q)}\right).$$

This implies that the integral in Eq. 22 reads

$$g(\beta,q)(r-q)\exp\left[\frac{g(\beta,q)}{\gamma}\right]\int_{\frac{g(\beta,q)}{\gamma}}^{\frac{g(\beta,q)}{\gamma}e^{\gamma(\widetilde{T}-h)}}\frac{\gamma z}{g(\beta,q)}\frac{e^{-z}}{\gamma}\ln\left(\frac{\gamma z}{g(\beta,q)}\right)\frac{dz}{\gamma z}$$
$$=\frac{(r-q)}{\gamma}\exp\left[\frac{g(\beta,q)}{\gamma}\right]\int_{\frac{g(\beta,q)}{\gamma}}^{\frac{g(\beta,q)}{\gamma}e^{\gamma(\widetilde{T}-h)}}e^{-z}\ln\left(\frac{\gamma z}{g(\beta,q)}\right)dz.$$

Integrating by parts gives

$$=\frac{(r-q)}{\gamma}\exp\left[\frac{g(\beta,q)}{\gamma}\right]\left\{-\exp\left[-\frac{g(\beta,q)}{\gamma}e^{\gamma(\widetilde{T}-h)}\right]\gamma\,(\widetilde{T}-h)+\int_{\frac{g(\beta,q)}{\gamma}}^{\frac{g(\beta,q)}{\gamma}e^{\gamma(\widetilde{T}-h)}}\frac{e^{-z}}{z}\,dz\right\}.$$

Finally, recall that $\int_x^{+\infty}(e^{-z}/z)\,dz=\mathrm{E}_1(x)$ is the exponential integral, the integral becomes

$$\frac{(r-q)}{\gamma}\exp\left[\frac{g(\beta,q)}{\gamma}\right]\left\{-\exp\left[-\frac{g(\beta,q)}{\gamma}e^{\gamma(\widetilde{T}-h)}\right]\gamma\,(\widetilde{T}-h)+\mathrm{E}_1\left(\frac{g(\beta,q)}{\gamma}\right)-\mathrm{E}_1\left(\frac{g(\beta,q)}{\gamma}e^{\gamma(\widetilde{T}-h)}\right)\right\},$$

and this concludes the proof. □

## 5 Capturing the effect of frailty and health prevention under the Gompertz law of mortality

As stressed by the World Health Organization[6], health conditions strongly affect mortality. Studying the relationship between health conditions and mortality is essential for enhancing health services and reducing preventable deaths worldwide, particularly by enabling effective responses to epidemiological change. Both communicable diseases (CDs) and noncommunicable diseases (NCDs), along with disability, are relevant causes of death, with noncommunicable diseases being prominent (e.g., due to the higher prevalence of Alzheimer's disease and diabetes among the population). Frailty entails the accumulation of health deficits and the connected greater burden on mortality risk. A substantial multidisciplinary literature—spanning medicine, epidemiology, and health economics—has established frailty as a key predictor of mortality. Strulik and Hansen (2025) propose a decomposition of the Gompertz law of mortality to enhance its explanatory power; the proposed decomposition relies on two laws: an exponential increase in health deficits estimated through a frailty index and a power law association between the frailty index and the mortality rate. We adopt the approach

[6] https://www.who.int/data/gho/data/themes/mortality-and-global-health-estimates/ghe-leading-causes-of-death

of Strulik and Hansen (2025) to integrate the contribution of frailty into our modeling framework, which, likewise, is based on the Gompertz law of mortality. We thus explain how the decomposition of the Gompertz law can be formalized and interpreted within our model.

In Strulik and Hansen (2025), the (average) stock of health deficits at age $t$, $D(t)$, evolves as follows:

$$\frac{d}{dt}D(t) = \zeta D(t), \qquad D(h) = D_h > 0, \tag{25}$$

where $\zeta > 0$ represents the rate of physiological ageing while $D_h$ represents the initial stock of health deficits at age $h$. The solution of Eq. 25 is given by

$$D(t) = D_h\, e^{\zeta(t-h)}, \tag{26}$$

which describes the exponential increase of health benefits with chronological age, as a result of the progressive accumulation process. Such health deficit accumulation is measured at the country level and for every age $t$ by the frailty index, which consists of the average of the prevalence rates of all potential deficits. Referring to the empirical disease categories documented in Strulik and Hansen (2025), the frailty index employed there aggregates 32 conditions encompassing both CDs — mainly infectious and nutritional causes such as diarrheal diseases and malnutrition — and a much broader set of noncommunicable diseases, including cardiovascular, metabolic, neurological, musculoskeletal, renal, and sensory disorders.

This classification highlights an important feature implicit in the deficit accumulation dynamics: different families of diseases contribute to the growth of $D(t)$ through different biological, behavioral, and environmental channels. CDs typically impact $D(t)$ through episodes of acute health deterioration, often sudden and concentrated in early and mid-life. Their impact on deficits tends to be episodic but potentially severe, and in many cases, they allow a *restitutio ad integrum*, meaning that individuals may return to their pre-shock deficit level once the infection resolves. For this reason, CDs are less suitable for a persistent-deficit framework. By contrast, the progression of NCDs exerts persistent, cumulative, and largely age-progressive effects on $D(t)$. These conditions represent the bulk of frailty accumulation at older ages, and they are particularly relevant given that our analysis focuses on cohorts starting at ages 50 and 60, that is, at ages where NCDs become prominent while CDs become relatively infrequent or less consequential for long-term deficit trajectories. Prevention of CDs often involves public or semi-public interventions — such as vaccination, sanitation, and environmental health policies — which tend to reduce the frequency and intensity of acute deficit spikes. For NCDs, instead, prevention operates mainly through individual behavioral and medical choices, such as diet, exercise, screening, adherence to medication, or early interventions in physiotherapy and physiatry. These investments reduce the baseline drift of deficits, slowing down the long-term increase in $D(t)$. In our framework, a reduction in the deficit growth parameter can be read as a composite effect of preventive investments acting simultaneously on acute shocks (related to CDs) and chronic ones (related to NCDs). This interpretation is fully consistent with the empirical classification of diseases presented in Strulik and Hansen (2025)

and strengthens the link between the microeconomic structure of our model and the epidemiological and demographic evidence underlying the regularities described by the Gompertz law of mortality. If the instantaneous hazard depends on health deficits through a power law,

$$\lambda(t) = \eta D(t)^{\theta}, \tag{27}$$

with scale parameter $\eta > 0$ and elasticity $\theta > 0$, then substituting Eqs. 26 into 27 yields the familiar Gompertz form

$$\lambda(t) = \eta [D_h]^{\theta} e^{(\theta\zeta)(t-h)}. \tag{28}$$

In Eq. 28, $\eta$ represents the baseline mortality level that remains after accounting for health deficits, and is shaped by multiple structural factors unrelated to biological aging itself. It is influenced by health-system quality, epidemiological environment, and socioeconomic conditions.

We now introduce a constant effort to prevent disease $q \in [0, 1]$, representing the fraction of individual wealth allocated to preventive and rehabilitative activities. This investment affects the relationship between deficits and mortality; hence $\eta$ should be treated as an endogenous function $\eta(q)$ that reflects how the physiological sensitivity of mortality to the deficit stock varies with the level of prevention. A higher value of $q$ corresponds to a lower value of $\eta(q)$, implying that, for the same level of deficit $D$, the associated hazard rate is reduced. Therefore, health investment does not change the accumulation of deficits *per se*, but weakens their translation into mortality, operationally captured by a lower effective $\eta$. From an economic point of view, $\eta(q)$ introduces a meaningful feedback between consumption, savings, and longevity. Higher prevention expenditures reduce mortality and extend the expected lifetime. This mechanism parallels the health-capital framework of Grossman (1972), but here operates through a biologically grounded channel, the physiological translation of deficits into mortality risk. The structure also aligns with the dynamic trade-off between health and longevity formalized in Dalgaard and Strulik (2014), where investment in prevention increases the efficiency of health maintenance and shifts the mortality curve. Furthermore, integrating insights from public health economics and geriatric medicine allows the model to account for heterogeneity in $\eta(q)$ in socioeconomic and institutional contexts, as highlighted in Gianfredi et al. (2025): prevention and early intervention are not only medical strategies but also key determinants of economic sustainability under population ageing. We thus assume that mortality follows the modified Gompertz specification:

$$\lambda(t, q) = \eta(q) [D_h]^{\theta} e^{(\theta\zeta)(t-h)}, \tag{29}$$

with $\eta'(q) < 0$. This representation integrates the behavioral, physiological, and economic dimensions of ageing within a unified analytical framework. It enables the model to account for preventive health spending as a direct determinant of mortality deficit mapping, offering a tractable yet realistic way to connect clinical and macroeconomic evidence. Future empirical work could calibrate $\eta(q)$ using clinical indicators of intrinsic capacity as proposed by the WHO ICOPE framework. Such an approach

would link the abstract mortality function with observable health trajectories, allowing the quantitative integration of biomedical and economic data. In this sense, $\eta(q)$ becomes not just a scaling parameter in the Gompertz law but a measurable function of preventive effort, embodying the transition from reactive medicine to proactive health economics.

From a mathematical perspective, comparing Eqs. 23 and 29 reveals that they are formally equivalent. By defining $g(\beta, q) = \eta(q)\,[D_h]^{\theta}$ and $\gamma = \theta\zeta$, we obtain exactly the same analytical structure. Moreover, the function $g(\beta, q)$ gathers the information contained in the parameters $\eta, D_h, \theta$, while $\gamma$ depends solely on the parameters $\zeta, \theta$. Consequently, all theoretical results presented in Sects. 3 and 4 continue to be valid.

## 6 Empirical implementation

We recall that the main key idea in this paper is to understand how allocating a portion of personal wealth to preventive activities can reduce mortality risk and increase life expectancy. This endogenous link between health spending and survival allows us to examine the dynamic interaction between wealth accumulation, longevity, and health-related behaviors throughout the life cycle. In this section, using numerical simulations, we will show how optimal prevention strategies systematically differ according to gender, age, and country-specific mortality patterns. The findings will underscore the role of personal characteristics and demographic factors in shaping the trade-offs between consumption, investment, and health. In the preceding sections, we demonstrated that, assuming the hazard rate follows the specification given in Eq. 10, the value function can be expressed in closed form. The results obtained up to this point do not depend on the particular specification of the function $g(\beta, q)$. Nonetheless, to perform numerical simulations, we must first estimate the Gompertz parameters $\beta$ and $\gamma$ and then choose an explicit analytical expression for $g(\beta, q)$. Section 6.1 is devoted to parameter estimation; in Section 6.2, we introduce the analytical functional form adopted as an example for $g(\beta, q)$; and in Section 6.3, we present the empirical application based on the simulations.

### 6.1 Mortality modeling

We calibrate the Gompertz law of mortality on historical data on exposure-to-risk and the number of deaths of Italian and Polish male and female populations, available in the Human Mortality Database (HMD 2025).

For our numerical application, by way of example, we focus on the cohort of individuals whose age, at the beginning of the investment horizon, is either 50 ($h = 50$) or 60 ($h = 60$). We fix the initial year of observation at 1969 and employ a cohort-based approach using period life tables to model mortality patterns. Specifically, we track the cohort aged 50 in 1969 through to age 100, and the cohort aged 60 through to age 110, with the observation period ending in 2019, that is, the last calendar year

**Table 1** Gompertz model's parameters age $h = 50$

| Italy | Female | Male |
|---|---|---|
| $\beta$ | 0.002634644 | 0.007462202 |
| $\gamma$ | 0.099187 | 0.080488 |
| Poland | Female | Male |
| $\beta$ | 0.004039403 | 0.009701139 |
| $\gamma$ | 0.094524 | 0.079009 |

preceding the most severe spread of the COVID-19 pandemic. Adopting a cohort approach allows us to select mortality information as age and calendar year move forward simultaneously, thereby mimicking the average mortality profile of people as they age over time.

For both initial ages, we use 50 years of data to perform the calibration of the Gompertz law of mortality and limit ourselves to ages up to 100 or 110. This choice relates to the fact that the mortality data on people who reach age 110 or those who are supercentenarians are, in most cases, of poor quality and potentially biased, and do not convey statistically relevant information within the calibration process. Furthermore, this choice is consistent with the availability of data in the HMD.

To perform the calibration, we define a generalized linear model whose dependent variable is the observed number of deaths and with the log exposure-to-risk as an offset in the model. We assume Poisson errors; moreover, we set a log-link function and a linear predictor, according to Currie (2016). The implementation in the **R** software (https://www.r-project.org) is consistent with the procedure described in Currie (2016). Mortality experiences can be heterogeneous across different populations (Milevsky 2020). This is also the case for Italy and Poland, which we choose as examples of different mortality experiences. Italy has historically been characterized by lower mortality (and, thus, higher longevity) than Poland. This can be inferred by the calibrated parameters of the Gompertz law of mortality reported in Tables 1 and 2. For instance, gender being equal, the initial natural mortality, as expressed by the parameter $\beta$, is greater for Poland than for Italy. Furthermore, the country being fixed, females are characterized by lower values of the parameter $\beta$ and higher values of the parameter $\gamma$, compared with men, consistent with the fact that, worldwide, women experience, on average, higher longevity than men, a phenomenon known as the gender mortality gap.

**Table 2** Gompertz model's parameters age $h = 60$

| Italy | Female | Male |
|---|---|---|
| $\beta$ | 0.008564956 | 0.01941307 |
| $\gamma$ | 0.097040912 | 0.07956336 |
| Poland | Female | Male |
| $\beta$ | 0.01078279 | 0.02303999 |
| $\gamma$ | 0.10049617 | 0.08238654 |

### 6.2 Choice of $g(\beta, q)$ function

The function $g(\beta, q)$ is specified in a form that prioritizes modeling flexibility, although its structure may not lend itself to immediate interpretation. In this study, we adopt an exponential specification for two main reasons. First, its structure closely mirrors that of the density Eq. 12 of the random variable $\tau$. Second, this choice ensures that the value function Eq. 24 is concave with respect to $q$, thus guaranteeing the existence of a global maximum for the static optimization problem analyzed in Section 6.3. We recall that function $g(\beta, q)$ must satisfy certain characteristics to be used in the reparameterization of the Gompertz model introduced in Eq. 10. Specifically,

1. for all $q \in [0, 1]$, it must be $g(\beta, q) > 0$;
2. if $q = 0$, then $g(\beta, 0) = \beta$;
3. for all $q \in [0, 1]$, it must be $\frac{\partial g}{\partial q}(\beta, q) < 0$.

These assumptions have an explicit meaning related to the Gompertz model. The first assumption requires that function $g(\beta, q)$ is strictly positive for every admissible value of $q$, since it replaces the parameter $\beta$ in the original Gompertz model, which is inherently positive. The second assumption asserts that when $q = 0$, corresponding to no expenditure on health prevention, the hazard rate to be used is the one whose parameters are estimated in Sect. 6.1. The third assumption states that the hazard rate decreases as the share of the portfolio allocated to health prevention increases; in other words, the greater the health prevention, the lower the hazard rate. Graphically, it is as if the hazard rate is shifted downward following a decrease in the parameter $\beta$.

The analytical form of the function $g$ that we will use in the numerical simulations is the following:

$$g(\beta, q) = \beta \exp(-\varepsilon q), \tag{30}$$

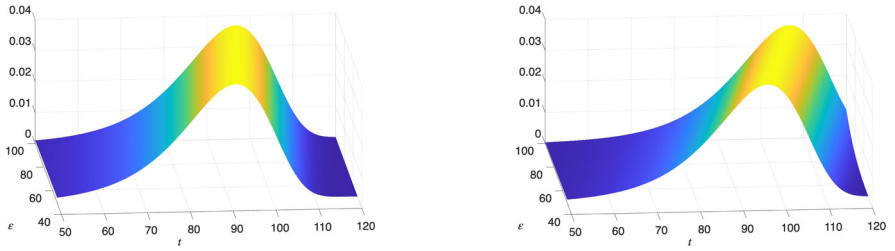
where $\varepsilon > 0$ is a parameter that describes the effectiveness of health prevention spending on the cohort under analysis. Note that the proposed form Eq. 30 is mainly intended as a useful example to shed light on the lifetime financial effects that health expenditures may produce through mortality rate reduction, rather than to precisely capture complex real-world interactions. It is important to interpret this definition in the context of Strulik and Hansen (2025). In that work, the value of $\beta$ is expressed as

$$\beta = \eta[D_h]^{\theta}. \tag{31}$$

Here, we propose a revised formulation of Eq. 31 as

$$g(\beta, q) = \eta e^{-\varepsilon q}[D_h]^{\theta}. \tag{32}$$

What we are doing is modifying the value of the parameter $\eta$ by reducing it more intensely as the percentage of investment in health prevention increases. In other words, we specify the analytical form of $\eta(q)$ that appears in Eq. 29 as $\eta(q) = \eta e^{-\varepsilon q}$, $\varepsilon > 0$.




(a) $q = 0.01$; $\varepsilon \in [50, 100]$, $t \in [50, 120]$ (b) $q = 0.02$, $\varepsilon \in [50, 100]$, $t \in [50, 120]$

**Fig. 1** Sensitivity of the density $f(t, q)$ of $\tau$ with respect to $\varepsilon$ for two different values of $q$

Most likely, the best choice would be to identify different functions $g(\beta, q)$ for each cohort considered. However, this would prevent a meaningful comparison of the results presented in the next section, so we prefer to use this simple structure, which, although less precise, yields results that are easier to interpret. To illustrate the impact of the chosen function $g(\beta, q)$ on the density of the random variable $\tau$, we derive its explicit form and represent it for two different values of $q$. More specifically, by using Eq. 30 the density of $\tau$ takes the following analytical form:

$$f(t, q) = \beta \exp\left[\gamma(t - h) - \varepsilon q - \beta \frac{(e^{\gamma(t-h)-\varepsilon q} - e^{-\varepsilon q})}{\gamma}\right], \tag{33}$$

for all $t \in [h, \widetilde{T}]$, $q \in [0, 1]$ and $\varepsilon \in [0, +\infty)$.

In Fig. 1, we show the results of a sensitivity analysis aimed at showing how the density of $\tau$ varies with respect to different values of the parameter $\varepsilon$. By way of an example, we use the values for Gompertz parameters $\beta$ and $\gamma$ estimated in Section 6.1 and reported in Table 1, corresponding to a 50-year-old woman residing in Italy. We fix two values for the decision variable $q$: $q = 0.01$ in the figure displayed in the left panel and $q = 0.02$ in the figure on the right. As $\varepsilon \in [50, 100]$ varies, it can be observed that the support of the density shifts towards higher ages. The higher the value of the parameter $\varepsilon$, the greater the impact of health prevention on the density of $\tau$, which tends to shift upward.

### 6.3 Optimal health prevention

In this section, we will use the data and ideas presented in the previous sections to numerically solve the optimization problem being developed in this study, namely, to determine the optimal share of investment, $q^*$, in health prevention that the investor should choose. We will use the objective function Eq. 24, where the function $g(\beta, q)$ takes the analytical form defined in Eq. 30. For comparison purposes, we assume that all decision makers are characterized by the same initial bequest $\ln(X(h))$. We omit

this term since it does not depend on $q$. Accordingly, we maximize the following function with respect to the variable $q$. The next expression describes the function with all parameters included:

$$\begin{aligned}\pi(q) : [0,1] &\to \mathbb{R} \\ q &\mapsto \frac{((\mu-\rho)^2/2\sigma^2+\rho-q)}{\gamma}\exp\left[\frac{\beta e^{-\varepsilon q}}{\gamma}\right]\Bigg\{-\exp\left[-\frac{\beta e^{\gamma(\widetilde{T}-h)-\varepsilon q}}{\gamma}\right]\gamma\,(\widetilde{T}-h) \\ &\quad +\mathrm{E}_1\left(\frac{\beta e^{-\varepsilon q}}{\gamma}\right)-\mathrm{E}_1\left(\frac{\beta e^{\gamma(\widetilde{T}-h)-\varepsilon q}}{\gamma}\right)\Bigg\}\end{aligned} \tag{34}$$

We now proceed to define all the parameters that appear in this formula, distinguishing between financial and actuarial ones. For the financial parameters, we set:

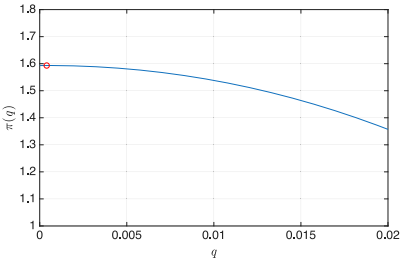
- $\rho = 3\%$, which is in line with the EUR EIOPA term structure of risk-free interest rates for very long maturities [7],
- $\sigma = 15\%$, which is quite in line with the current market situation,
- $\mu = 6\%$, which is obtained by considering the market price of financial risk $\xi = 20\%$ and by using the relation $\mu = \rho + \xi\sigma$.

For the actuarial parameters, we adopt the following assumptions.

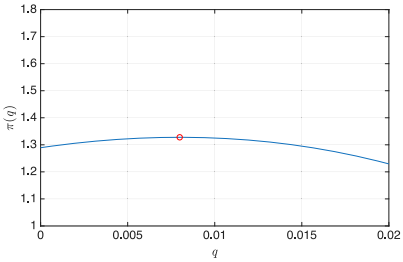
- $h = 50, 60$, as explained in Sect. 6.1, in the case of females and males residing in Italy or Poland. Thus, the parameters $\beta$ and $\gamma$ take the corresponding values reported in Tables 1 and 2;
- $\widetilde{T} = 120$. To capture the effects of health prevention on the right tail of the density Eq. 33, we have chosen 120 years as the maximum attainable age, consistent with standard actuarial practice, e.g., ANIA (2015);
- $\varepsilon = 70, 60$. This parameter represents the effectiveness of health prevention on the force of mortality; it is a parameter that is difficult to estimate and should ideally be calibrated for each cohort. In this work, we will keep it constant for all cohorts in order to compare the results obtained. It will be set $\varepsilon = 70$ for the simulation of the cohort of $h = 50$ years and $\varepsilon = 60$ for the simulation of the cohort of $h = 60$.

In Figs. 2 and 3, we show the value function against the fraction of investment in health prevention $q$, for female (left panels) and male (right panels) investors belonging to the Italian population or the Polish one (top and bottom panels, respectively). In all graphs, the red dot identifies the maximum value of $V$, and the corresponding value on the $x$-axis indicates the optimal fraction of health investment, $q^*$. We notice that, with the initial available wealth being equal across genders and countries, $q^*$ varies in magnitude due to heterogeneity in the underlying mortality patterns. Among the four cases considered, the Italian female population is characterized by the lowest baseline mortality curve, and thus by the longest expected residual lifetime.

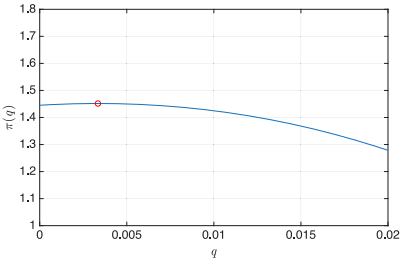
[7] https://www.eiopa.europa.eu/tools-and-data/risk-free-interest-rate-term-structures_en



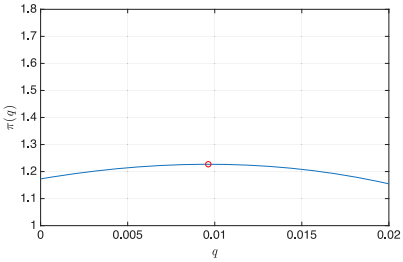

(a) Italy, female, age $h = 50$, $q^* = 0.0004$




(b) Italy, male, age $h = 50$, $q^* = 0.0080$

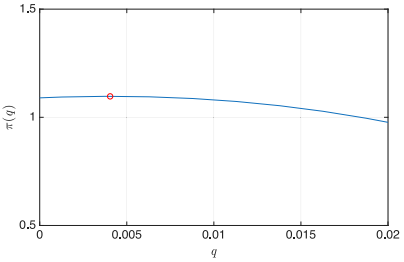



(c) Poland, female, age $h = 50$, $q^* = 0.0033$

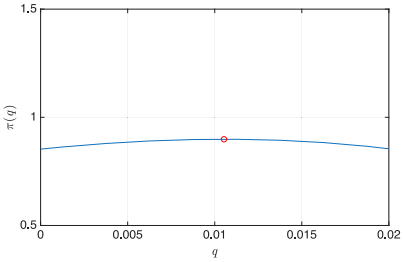



(d) Poland, male, age $h = 50$, $q^* = 0.0096$

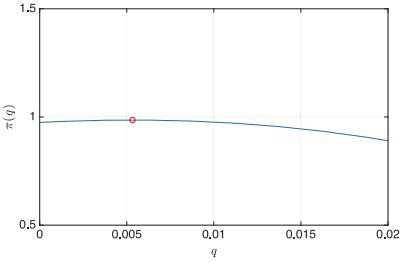
**Fig. 2** Numerical results for 50 years old



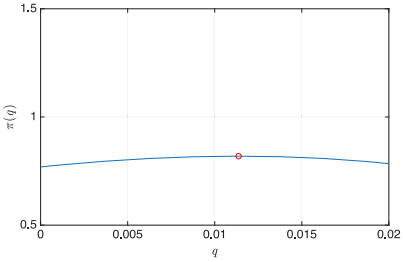

(a) Italy, female, age $h = 60$, $q^* = 0.0040$




(b) Italy, male, age $h = 60$, $q^* = 0.0105$




(c) Poland, female, age $h = 60$, $q^* = 0.0053$




(d) Poland, male, age $h = 60$, $q^* = 0.0114$

**Fig. 3** Numerical results for 60 years old

Let us denote by $e_h(q)$ the expected residual lifetime of people aged $h$, obtained under a level of health prevention equal to $q$; then,

$$e_h(q) := \int_h^{\widetilde{T}} (t-h)\, S(t,q)\, \lambda(t,q)\, dt. \tag{35}$$

In Table 3, we compare the value of $e_h(q)$ without health prevention, $q = 0$, and with optimal health prevention, $q^* > 0$. We observe that the increase is greater for men, while it is less significant for women, being characterized by a lower value of the Gompertz parameter expressing the initial level of mortality, $\beta$.

From a financial perspective, the expected residual lifetime determines how long an investor can accumulate wealth; the longer the expected time horizon, the greater the wealth-compounding effect. At the same time, the longer the expected time horizon ahead, the lower the percentage of health investment that maximizes the value function. The intuition behind this relation is that the higher the baseline residual lifetime, the weaker the contribution of health expenditures in extending the accumulation time horizon. Indeed, when focusing on the initial age $h = 50$, as shown in Fig. 2, we find the lowest value of $q^*$ for Italian females ($q^* = 0.04\%$), that is, the population with the highest baseline residual lifetime $e_h(0) = 31.86$ (see Table 3, line 1). Contextually, we find that the effect of optimal health investing on the residual lifetime, $\Delta e_h = e_h(q^*) - e_h(0)$, is very mild and approximately equal to 0.26 years of life. Based on the same intuition, among the considered cases, Polish males are characterized by the highest value of $q^*$, roughly 0.96% and by the largest spread $\Delta e_h = e_h(q^*) - e_h(0) = 6.93$ years of life, for people whose initial age is $h = 50$.

These numerical outcomes, although derived within a simplified mathematical description of the relationship between health and mortality outcomes, can be interpreted coherently with the existing evidence on the *male–female health–mortality paradox* or, equivalently, the *gender and health paradox*, that is the phenomenon by which women, on average, live longer than men but also experience higher morbidity rates, and spend a larger proportion of their total life in poor health, compared to men (Luy and Minagawa 2014). In particular, some literature points to the fact that there exist differences between females and males in health conditions globally and that women are mostly concerned with diseases that are not closely related to

**Table 3** Expected life $e_h(q)$ at age $h = 50, 60$ under different mortality rates and percentage decrease in $\beta$

| Country | Age $h$ | Gender | $e_h(0)$ | $q^*$ | $e_h(q^*)$ | $\lvert\Delta\beta\rvert$ |
|---|---|---|---|---|---|---|
| Italy | 50 | Female | 31.86 | 0.0004 | 32.12 | 0.000072747 |
| Italy | 50 | Male | 25.79 | 0.0799 | 31.61 | 0.007434414 |
| Italy | 60 | Female | 21.80 | 0.0040 | 23.87 | 0,000986488 |
| Italy | 60 | Male | 17.06 | 0.0105 | 22.76 | 0.005049390 |
| Poland | 50 | Female | 28.90 | 0.0033 | 31.11 | 0.001538524 |
| Poland | 50 | Male | 23.46 | 0.0096 | 30.39 | 0.008500159 |
| Poland | 60 | Female | 19.49 | 0.0053 | 22.06 | 0.002937202 |
| Poland | 60 | Male | 15.38 | 0.0114 | 21.19 | 0.011414136 |

mortality, such as chronic conditions. As stressed in Patwardhan et al. (2024), women tend to have a higher burden of morbidity-driven conditions (e.g., mental disorders and musculoskeletal disorders), while men tend to experience a higher burden of mortality-driven conditions (e.g., ischemic heart disease). In light of the different health burdens that challenge men and women, a strand of literature advocates gender-informed life course health strategies. The key issue for women, which may inform their choice to make their health investments, is to enhance the proportion of total life spent in good health. While health investments targeting the treatment of the most prevalent life-threatening health conditions in men may have a greater potential to add extra years of life as long as possible.

Some literature addresses healthy ageing within a life-course framework, and studies how dynamically lifetime affects health and disease risk (Kuh et al. 2014; Foster and Walker 2021). Cocchi et al. (2025) emphasize the importance of interventions to promote healthy ageing that, although sharing core strategies, account for age-specific needs, for instance, lifestyle interventions to prevent decline in old age. Health promotion and health education can have an impact on the health behavior of older adults (Kim et al. 2024; Mansyur et al. 2022).

In the context of our study, we consider the portfolio selection problem faced by older adults, in particular, of individuals aged 60 years. Older adults are typically exposed to a higher risk of chronic disease morbidity and face more complex healthcare needs. In the simplified framework where we work, the impact of the life course (e.g., the dynamic interaction between genetics, lifestyle, and environmental factors) on the frailty experience in old age is summarized by the higher initial level of mortality that intrinsically characterizes 60-year-old people, compared to 50-year-old individuals, as revealed by statistical data. Consequently, conditional on country and gender, people aged 60 are characterized by a lower residual lifetime, $e_h(0)$, as shown in Table 3, and by higher values of $q^*$.

Now, returning to the decomposition of $\beta$ provided by Strulik and Hansen (2025), $\beta = \eta\, D_h^\theta$, Eq. 31, if we replace $\eta$ with $\eta(q) = \eta\, e^{-\varepsilon q}$, we get the $q$–dependent Gompertz level parameter:

$$\beta(q) = \eta(q)\, D_h^\theta = \eta e^{-\varepsilon q} D_h^\theta = \beta\, e^{-\varepsilon q}. \tag{36}$$

Equation 36 shows that our reparameterization in Eq. 30 is fully consistent with Strulik's decomposition: prevention does not alter the deficit component $D_h^\theta$ but acts exclusively on the background term $\eta$, which in turn shifts the entire Gompertz curve downward. Thus, to quantify the effect of health prevention on mortality, it is sufficient to examine how the parameter $\beta$ varies when transitioning from the absence of prevention ($q = 0$) to the optimal level of prevention ($q = q^*$). Since Eq. 36 holds, the absolute variation in the mortality level is

$$\Delta\beta = \beta(q^*) - \beta(0) = \beta\left(e^{-\varepsilon q^*} - 1\right), \tag{37}$$

which measures the proportional decrease in the Gompertz mortality level induced by optimal prevention expenditure $q^*$. In Table 3, we report the value of $|\Delta\beta|$.

As shown by the empirical estimates, there is already a different variation within the same cohort: for example, Italian males aged 50 exhibit a reduction of approximately 0.0074, whereas Italian females at the same age show only about a 0.00007 reduction. This heterogeneity reflects differences both in the baseline deficit $D_h$ and in the intensity of prevention $q^*$. From an economic perspective, a large $\Delta\beta$ implies that prevention is highly effective in shifting the entire mortality curve downward, which yields larger gains in expected life-years. By contrast, a small reduction suggests that either the potential for reducing background mortality via prevention is limited or that initial deficits are already small.

From a financial point of view, it may be interesting to ask what the difference is between the final value of the portfolio in the presence and absence of health prevention. Recall that the portfolio is maximized by considering the logarithmic transformation of its final value, in order to capture risk aversion. However, to compare the portfolio values, we can compute the two geometric Brownian motions corresponding to the presence and absence of health prevention in $e_h(0)$ and $e_h(q^*)$, respectively. In this way, we obtain two random variables that we compare by calculating their expected value.

From an analytical point of view, the steps to be performed are the following.

$$\begin{aligned}
\mathbb{E}[X(h+e_h(q))] &= \mathbb{E}\left[X(h)\exp\left\{\left[\frac{(\mu-\rho)^2}{2\sigma^2}+\rho-q\right]e_h(q)+\frac{\mu-\rho}{\sigma}W(h+e_h(q))\right\}\right] \\
&= X(h)\exp\left\{\left[\frac{(\mu-\rho)^2}{2\sigma^2}+\rho-q\right]e_h(q)\right\}\cdot\mathbb{E}\left[\exp\left\{\frac{\mu-\rho}{\sigma}W(h+e_h(q))\right\}\right] \\
&= X(h)\exp\left\{\left[\frac{(\mu-\rho)^2}{2\sigma^2}+\rho-q\right]e_h(q)\right\}\cdot M_{W(h+e_h(q))}\left(\frac{\mu-\rho}{\sigma}\right) \\
&= X(h)\exp\left\{\left[\frac{(\mu-\rho)^2}{2\sigma^2}+\rho-q\right]e_h(q)\right\}\exp\left\{\frac{(\mu-\rho)^2}{2\sigma^2}e_h(q)\right\},
\end{aligned}$$

Here, $W(h+e_h(q))$ is a centered Normal random variable with variance $e_h(q)$ (recall that $W(t)$ is a Brownian motion defined on $[h,\widetilde{T}]$, with $W(h)=0$ a.s.). Moreover, $M_Y(t)=\mathbb{E}[e^{tY}]$ denotes the moment generating function of the random variable $Y$. Then,

$$\mathbb{E}[X(h+e_h(q))] = X(h)\exp\left\{\left[\frac{(\mu-\rho)^2}{\sigma^2}+\rho-q\right]e_h(q)\right\}. \tag{38}$$

We observe that on the left-hand side of Eq. 38 we have committed a mild abuse of notation, since the stochastic process $X(t)$ also depends on the value chosen for $q$, whereas we have highlighted the dependence on $q$ only through the time value $e_h(q)$ at which the process is evaluated. However, the right-hand side is well defined and therefore, for the sake of simplicity, we do not indicate the explicit dependence of the stochastic process $X(t)$ on the parameter $q$.

Assuming an initial investment of $X(h) = \text{EUR } 100,000$, in Table 4 we have calculated the expected value of the portfolio when the life expectancy at age $h$ equals $e_h(q)$. We observe that, in every case, there is an increase in the expected value of

**Table 4** Expected portfolio value at $h + e_h(q)$ with age $h = 50, 60$ and $X(h) =$ EUR 100.000

| Country | Age ($h$) | Gender | $\mathbb{E}[X(h + e_h(0))]$ | $\mathbb{E}[X(h + e_h(q^*))]$ |
|---|---|---|---|---|
| Italy | 50 | Female | 491.890 | 491.940 |
| | | Male | 363.010 | 377.270 |
| | 60 | Female | 297.400 | 299.449 |
| | | Male | 234.630 | 245.590 |
| Poland | 50 | Female | 424.270 | 427.000 |
| | | Male | 323.130 | 341.110 |
| | 60 | Female | 264.990 | 267.950 |
| | | Male | 215.800 | 267.300 |

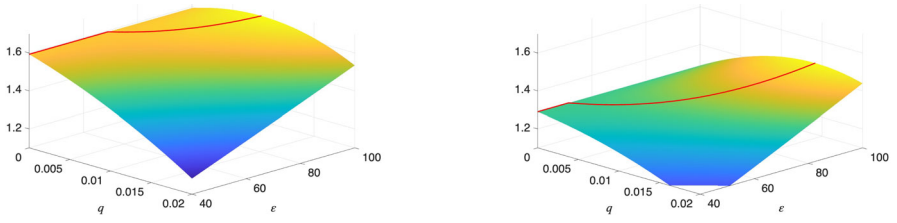
the portfolio in the presence of health prevention. This increase reflects the increase in life expectancy: in fact, we observe how in formula Eq. 38 the trade-off is made explicit between the lower return – present in square brackets, where $q$ appears with a negative sign – and the increase in $e_h(q)$, which increases in $q$.

### 6.4 Sensitivity with respect to $\varepsilon$

As explained in Sect. 6.2, by the way function $g$ is constructed (see Eq. 30), parameter $\varepsilon$ contributes to shaping the downward shift of the individual's mortality curve. The higher $\varepsilon$, the larger the extent of mortality reduction over the remaining lifetime.

In this section, we deem it useful to measure the sensitivity of the value function with respect to $\varepsilon$, since this parameter was set as a constant in the previous section. By means of an example, we focus on the case of Italian females and males aged 50 years. Figure 4 shows the graph for women (left panel), and men (right panel). On the $z$-axis, we display the objective function described in Eq. 34, against $q$ (on the $y$-axis) and $\varepsilon$ (on the $x$-axis). By shedding light on the behavior of the value function with varying $\epsilon$ and $q$, Fig. 4 allows the most general view on the outcomes of the optimization problem we consider in this paper. The red curve describes the optimal value $q^*$ for each possible choice of $\varepsilon \in [50, 100]$.

For both genders, we observe that small values of $\varepsilon$ are associated with a zero value of $q^*$ (no health investment undertaking). Beyond a threshold value for $\varepsilon$, the




(a) Italy, female, 50 years old

(b) Italy, male, 50 years old

**Fig. 4** Sensitivity with respect to $\varepsilon$

optimal value $q^*$ becomes strictly positive and increases in $\varepsilon$. Naturally, above this threshold, it becomes meaningful to consider the effect of health investments on the portfolio performance. Overall, the outlined results, that are derived within a financial modeling framework, can be interpreted under a multifaceted perspective, that go beyond the financial domain. It may be intuitively predictable that allocating resources to health-related strategies can enhance the quality and/or length of one's own lifetime and, thus, eventually extend the time horizon over which to accumulate wealth. In our paper, we extrapolate some further characteristics around this mechanism; for instance, we show how the main financial object we deal with, the value function, behaves under different individuals' characteristics (e.g., gender), and under different levels of effectiveness. Furthermore, we show that a threshold can be identified to make the portfolio strategy meaningful (positive values of $q^*$). Such outcomes can inform policy-making initiatives that aim to consolidate health education.

## 7 Conclusion

This paper presents a novel approach to integrating health prevention expenditures into a lifetime portfolio selection model under uncertain lifetimes. By combining financial portfolio optimization techniques with actuarial mortality modeling and assumptions about the impact of prevention on mortality rates, we have developed a framework for analyzing the interplay between wealth, health investments, and longevity.

The key contribution lies not in providing directly applicable investment advice, but rather in establishing links across the financial, actuarial, and medical domains. From a financial perspective, we extend Merton's canonical portfolio selection model to account for health expenditures that can influence the random lifetime horizon. Actuarial principles were used through the Gompertz mortality law, which we reparameterized to capture the potential reduction in mortality from preventive care. Although the specific functional form adopted for this reparameterization is stylized, it paves the way for more intricate medical and demographic modeling of how health investments can shift mortality curves.

Beyond its technical formulation, the proposed framework offers a conceptual reinterpretation of preventive health expenditure as an endogenous determinant of the individual financial planning horizon. By allowing agents to partially shape their own survival distribution through prevention, the model departs from standard life-cycle portfolio problems in which longevity risk is purely exogenous. In this sense, preventive healthcare can be viewed as a form of self-insurance against mortality risk, directly affecting expected longevity and the effective duration of wealth accumulation.

The empirical analysis illustrated how optimal prevention expenditure levels can be derived for different populations by gender, age, and country. The analysis highlighted the inherent trade-offs, as greater longevity achieved through prevention comes at the cost of lower investment returns, but extends the investment horizon. The sensitivity to the parameter of prevention effectiveness $\varepsilon$ further emphasized the need for a careful calibration that integrates expert medical knowledge.

A particularly relevant empirical insight emerging from the numerical analysis is the systematic heterogeneity in optimal prevention choices across demographic groups. The results show that individuals characterized by higher baseline mortality – such as males, older cohorts, and populations with less favorable survival profiles – optimally allocate a larger share of wealth to preventive healthcare and experience larger gains in expected residual lifetime. Conversely, groups with lower baseline mortality benefit less from additional prevention in terms of longevity extension, and therefore optimally devote fewer resources to it. This pattern provides a clear economic interpretation of observed differences in health investment behavior and is consistent with well-documented empirical regularities in mortality and ageing. From a financial standpoint, these results highlight a fundamental trade-off between short-run wealth accumulation and long-run horizon extension. Preventive health expenditure reduces the instantaneous growth rate of wealth by diverting resources away from financial investment, but simultaneously lengthens the period over which returns can compound by increasing expected lifetime. The optimal prevention choice therefore balances these opposing forces and depends critically on baseline mortality conditions, which vary by age, gender, and country.

We point out that, in this paper, we devise a stylized mathematical relationship to describe the mechanism by which health investing may trigger downward shifts in the mortality curve, which simplifies the much more complex and non-obvious links between genetic and mortality risk factors and outcomes. This aspect can be interpreted as a limitation of our study. In this respect, for instance, the function responsible for shifting the mortality curve can be made more complex and more linked to the biological processes involved in individual ageing. Nevertheless, at this stage of the analysis, we deem simplicity and parsimony to be useful to uncover important, seminal patterns and features of the dynamic interaction between wealth accumulation and mortality improvements over the individual's lifetime.

A further limitation of our framework is that it abstracts from curative health expenditures, an important component of real-world healthcare systems. By focusing solely on prevention, the model isolates one analytically tractable channel through which individuals influence survival and financial choices. This simplification clarifies the key economic mechanisms but calls for caution when interpreting quantitative results in contexts where treatment and prevention interact. From a policy perspective, the analysis underscores the value of prevention in reducing vulnerability and extending life, while noting that its effectiveness depends on the availability and quality of treatment.

An additional aspect to be stressed relates to the fact that non-communicable diseases lead to long-term, significant health costs for individuals, due to their chronic nature (Chowdhury et al. 2025). Women tend to experience a higher prevalence of several chronic conditions, including osteoporosis and Alzheimer's disease, than men. The literature advocates for filling knowledge gaps regarding the biological and social factors underlying the chronic conditions that predominantly affect women; these gaps significantly hinder prevention, diagnosis, and treatment efforts, and increase health costs for individuals and society (Temkin et al. 2023; National Academies of Sciences,

Engineering, and Medicine 2024). In light of this, the model proposed in our paper does not consider the complex relationships that may exist between preventive care and long-term health costs, but leaves this analysis as a further scope of research.

Ultimately, this work represents an initial step towards a unified framework bridging financial decision-making, actuarial modeling, and health economics. Future research could explore more sophisticated mortality models, integrate morbidity risk, and study behavioral aspects that influence prevention decisions. In an era of rising longevity and increasing interdependence between economic and health outcomes, adopting integrated frameworks of this kind appears essential to understand individual behavior and to inform both financial planning and public health policies over the life course.

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**Data Availability** The data that support the findings of this study are available in the HMD (Human Mortality Database), Max Planck Institute for Demographic Research (Germany), University of California, Berkeley (USA), and French Institute for Demographic Studies (France), at www.mortality.org.

## Declarations

**Conflict of Interest** The authors declare no conflict of interest.

**Competing Interests** The authors declare no competing interests.



## References

Abbate M, Gallardo-Alfaro L, del Mar BM, Tur JA (2020) Efficacy of dietary intervention or in combination with exercise on primary prevention of cardiovascular disease: A systematic review. Nutr Metab Cardiovasc Dis 30(7):1080–1093

Anderson ST, Laxminarayan R, Salant SW (2010) Diversify or focus? spending to combat infectious diseases when budgets are tight. J Health Econ 31(4):658–675

ANIA (2015) Le basi demografiche per assicurazioni di rendita a1900–2020 e a62. https://www.ania.it/studi-e-rapporti-demografici

Björk T (2005) Arbitrage theory in continuous time. Oxford Univ. Press, Oxford [u.a.]. ISBN 0199271267

Bo L, Capponi A (2016) Optimal investment in credit derivatives portfolio under contagion risk. Math Financ 26(4):785–834

Bo L, Wang Y, Yang X (2010) An optimal portfolio problem in a defaultable market. Adv Appl Probab 42(3):689–705
Bolin K, Caputo MR (2020) Consumption and investment demand when health evolves stochastically. J Econ Dyn Control 114:103893
Brennan MJ, Xia Y (2002) Dynamic asset allocation under inflation. J Financ 57(3):1201–1238
Carannante M, D'Amato V, Haberman S, Menzietti M (2024) Frailty-based mortality models and reserving for longevity risk. The Geneva Papers on Risk and Insurance - Issues and Practice 49:320–339
Choi M, Sempungu JK, Lee EH, Lee YH (2024) Living longer but in poor health: healthcare system responses to ageing populations in industrialised countries based on the Findings from the Global Burden of Disease Study 2019. BMC Public Health 24(576)
Chowdhury NMRA, Urme SA, Hridoy A, Nusrat F, Miah MS (2025) Exploring the effects catastrophic health expenditure due to non-communicable diseases and coping strategies: A case study among households in bangladesh. Public Health in Practice 10(100642). https://doi.org/10.1016/j.puhip.2025.100642
Cocchi C, Zazzara MB, Levati E, Calvani R, Onder G (2025) How to promote healthy ageing across the life cycle. Eur J Intern Med 135:5–13
Cocco JF, Gomes FJ (2012) Longevity risk, retirement savings, and financial innovation. J Financ Econ 103:507–529
Currie ID (2016) On fitting generalized linear and non-linear models of mortality. Scand Actuar J 2016(4):356–383
Dalgaard C-J, Strulik H (2014) Optimal aging and death: Understanding the preston curve. J Eur Econ Assoc 12(3):672–701
Ehrlich I (2000) Uncertain lifetime, life protection, and the value of life saving. J Health Econ 19(3):341–367
Ehrlich I, Chuma H (1990) A Model of the Demand for Longevity and the Value of Life Extension. J Polit Econ 98(4):761–782
Ehrlich I, Yin Y (2005) Explaining Diversities in Age-Specific Life Expectancies and Values of Life Saving: A Numerical Analysis. J Risk Uncertain 31:129–162
Ferrari G, Zhu A (2023) On a Merton Problem with Irreversible Healthcare Investment. arXiv:2212.05317
Fisher I (1930) The Theory of Interest: As Determined by Impatience to Spend Income and Opportunity to Invest It. MacMillan, New York
Fleming WH, Pang T (2004) An application of stochastic control theory to financial economics. SIAM J Control Optim 43(2):502–531
Foster L, Walker A (2021) Active Ageing across the Life Course: Towards a Comprehensive Approach to Prevention. Biomed Res Int 2021:6650414
Gersovitz M, Hammer JS (2004) The economical control of infectious diseases. Econ J 114(492):1–27
Gianfredi V, Nucci D, Pennisi F, Maggi S, Veronese N, Soysal P (2025) Aging, longevity, and healthy aging: the public health approach. Aging Clin Exp Res 37:125
Goenka A, Liu L, Nguyen MH (2014) Infectious diseases and economic growth. J Math Econ 50:34–53
Goldman SM, Lightwood J (2002) Cost optimization in the sis model of infectious disease with treatment. Topics in Econ Anal Pol 2(1)
Gompertz B (1825) On the nature of the function expressive of the law of human mortality on a new mode of determining the value of life contingencies. Philos Trans R Soc Lond 115:513–583
Grossman M (1972) On the Concept of Health Capital and the Demand for Health. J Polit Econ 80(2):223–255
Guasoni P, Huang Y-J (2019) Consumption, investment and healthcare with ageing. Finance Stochast 23:313–358
Gulis G, Zidkova R, Meier Z (2025) Changes in disease burden and epidemiological transitions. Scientific Reports 15(8961)
Gulliford M (2003) Commentary: Epidemiological transition and socioeconomic inequalities in blood pressure in jamaica. Int J Epidemiol 32(3):408–409
Guo J, Huang X, Dou L, Yan M, Shen T, Tang W, Li J (2022) Aging and aging-related diseases: from molecular mechanisms to interventions and treatments. Signal Transduction and Targeted Therapy 7(391)
Hall RE (2010) Forward-Looking Decision Making: Dynamic Programming Models Applied to Health, Risk, Employment, and Financial Stability. Princeton University Press. ISBN 9780691142425

HMD (2025) Human Mortality Database. Max Planck Institute for Demographic Research (Germany), University of California, Berkeley (USA), and French Institute for Demographic Studies (France). Available at www.mortality.org (data downloaded on January 21, 2025)
Huang H, Milevsky M, Salisbury TS (2012) Optimal retirement consumption with a stochastic force of mortality. Insurance: Math Econ 15:282–291
Hugonnier J, Pelgrin F, St-Amour P (2013) Health and (other) asset holdings. Rev Econ Stud 80:663–710
Jin D, Khan NU, Gu W, Lei H, Goel A, Chen T (2025) Informatics strategies for early detection and risk mitigation in pancreatic cancer patients. Neoplasia 60:101129
Kim I, An H, Yun S, Park HY (2024) Effectiveness of community-based interventions of older adults living alone: a systematic review and meta-analysis. Epidemiology and health 46:e2024013
Kim TS, Omberg E (1996) Dynamic nonmyopic portfolio behavior. Rev Financ Stud 9(1):141–161
Kuh D, Karunananthan S, Cooper R (2014) A life-course approach to healthy ageing: maintaining physical capability. Proc Nutr Soc 73(2):237–248
La Torre D, Maggistro R (2024) Multi-agent dynamic financial portfolio management: a differential game approach. Annals of Operations Research
La Torre D, Malik T, Marsiglio S (2020) Optimal control of prevention and treatment in a basic macroeconomic-epidemiological model. Math Soc Sci 100:100–108
La Torre D, Liuzzi D, Marsiglio S (2021) Epidemics and macroeconomic outcomes: Social distancing intensity and duration. J Math Econ 93:102473
Liu J (2007) Portfolio selection in stochastic environments. Rev Financ Stud 20(1):1–39
Luy M, Minagawa Y (2014) Gender gaps-life expectancy and proportion of life in poor health. Health Rep 25(12):12–19
Ma G, Zhu SP (2022) Revisiting the merton problem: from hara to cara utility. Comput Econ 59:651–686
Ma G, Zhu SP, Kang B (2020) A numerical solution of optimal portfolio selection problem with general utility functions. Comput Econ 55(3):957–981
Maggistro R, Marino M, Martire A (2025) A dynamic game approach for optimal consumption, investment and life insurance problem. Ann Oper Res 346:1377–1398
Mansyur S, Irwan AM, Arafat R, Hardianto Y (2022) Effective health education methods to improve self-care in older people with chronic heart failure: A systematic review. Health Sciences Review 5:100060
Merton RC (1969) Lifetime Portfolio Selection under Uncertainty: The Continuous-Time Case. Rev Econ Stat 51:247–257
Merton RC (1971) Optimal consumption and portfolio rules in a continuous time model. J Econ Theory 3(4):373–413
Milevsky MA (2020) Calibrating Gompertz in reverse: What is your longevity-risk-adjusted global age? Insurance: Math Econ 92:147–161
Modigliani F (1986) Lifecycle, individual thrift and the wealth of nations. Am Econ Rev 76(3):297–313
Modigliani F, Brumberg R (1954) Utility analysis and the consumption function: an interpretation of cross-section data. In: Kurihara K (ed) Post Keynesian Economics. Rutgers University Press, New Brunswick NJ, pp 388–436
Morand OF (2004) Economic growth, longevity and the epidemiological transition. Eur J Health Econ 5:166–174
National Academies of Sciences, Engineering, and Medicine (2024) Advancing Research on Chronic Conditions in Women. The National Academies Press, Washington, DC. https://doi.org/10.17226/27757
OECD/WHO (2015) Promoting Health, Preventing Disease: The Economic Case. Open University Press-McGraw-Hill, Buckingam
Olivieri A, Pitacco E (2011) Introduction to Insurance Mathematics. Technical and Financial Features of Risk Transfers. Springer, Berlin, Heidelberg
Patwardhan V, Gil GF, Arrieta A, Cagney J, DeGraw E, Herbert ME, Khalil M, Mullany EC, O'Connell EM, Spencer CN, Stein C, Valikhanova A, Gakidou E, Flor LS (2024) Differences across the lifespan between females and males in the top 20 causes of disease burden globally: a systematic analysis of the Global Burden of Disease Study 2021. The Lancet Public Health 9(5):E282–E294
Richard S (1975) Optimal consumption, portfolio and life insurance rules for an uncertain lived individual in a continuous time model. J Financ Econ 2:187–203
Strulik H, Grossmann V (2024) The economics of aging with infectious and chronic diseases. Econ Hum Biol 52:101319
Strulik H, Hansen C (2025) A microfoundation of the gompertz law: Health deficits, aging, and mortality. J Econ Behav Organ 218:150–168

Temkin SM, Barr E, H M et al (2023) Chronic conditions in women: the development of a national institutes of health framework. BMC Women's Health 23(162). https://doi.org/10.1186/s12905-023-02319-x
Wang F, Wang J-D (2021) Investing preventive care and economic development in ageing societies: empirical evidences from oecd countries. Health Economics Review 11(18)
Whitaker K (2020) Earlier diagnosis: the importance of cancer symptoms. Lancet Oncol 21(1):6–8
Wouterse B, Santos JV, Hiligsmann M (2025) Future directions for the economics of prevention. Expert Review of Pharmacoeconomics & Outcomes Research 1–4
Yaari M (1964) On the consumer's lifetime allocation process. Int Econ Rev 5(3):173–185
Yaari M (1965) Uncertain lifetime, life insurance and the theory of the consumer. Rev Econ Stud 32(2):137–150
Zariphopoulou T (1999) Optimal investment and consumption models with non-linear stock dynamics. Math Methods Oper Res 50(2):271–296
Zhao K (2015) The impact of the correlation between health expenditure and survival probability on the demand for insurance. Eur Econ Rev 75:98–111