

The Economics of Aging with Infectious and Chronic Diseases*

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Abstract. We develop an economic model of aging in which the susceptibility and severity of infectious diseases depend on the accumulated health deficits (immunosenescence) and the life history of infections affects the accumulation of chronic health deficits (inflammaging). Individuals invest in their health to slow down health deficit accumulation and take measures to protect themselves from infectious diseases. We calibrate the model for an average American and explore how health expenditure, life expectancy, and the value of life depend on individual characteristics, medical technology, and the disease environment. We then use counterfactual computational experiments of the U.S. epidemiological transition 1860-2010 to show that the decline of infectious diseases caused a substantial decline of chronic diseases and contributed more to increasing life expectancy than advances in the treatment of chronic diseases.

Keywords: Epidemiological transition; Health behavior; Health deficits; Immunosenescence, Infections; Inflammaging; Longevity.

JEL: D15, I10, I12, J24, J26.

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1. INTRODUCTION

In this paper, we propose a new health economic theory that captures the interaction between infections and chronic health conditions. We examine how medical technology, health policy, and income affect aging and longevity with endogenous health spending to prevent and treat both infectious and chronic diseases. To this end, we introduce to the economics profession the biological mechanisms of immunosenescence and inflammaging.

Immunosenescence is the gradual decline in functionality of the immune system. It is related to an increased susceptibility to infectious diseases, caused by declining immune responses to infections and vaccination (e.g. Goronzy and Weyand, 2013). In turn, a person’s immunological biography, i.e. the trained immunity from exposure to bacteria, viruses, fungi, and parasites (e.g. Franceschi et al., 2017, 2018) is a causative factor for inflammaging, i.e. the progressive development of chronic pro-inflammatory conditions. Recent advances in medical science emphasize that, consequently, the individual history of infection contributes to the development of chronic health deficits (Finch and Crimmins, 2004; Finch, 2010; Aiello et al., 2019, and Santoro et al., 2021). In Section 2, we explain in more detail how a person’s immunological biography is closely related to physiological aging and why frailer individuals (with more pre-existing conditions) are likely to experience more severe and potentially fatal courses of infections (e.g. Geriatric Medicine Research Collaborative et al., 2021).

The interaction between infectious diseases and chronic health deficits is affected by health behaviors such as preventive and curative health care expenditures, which jointly with biological factors are important for understanding aging and the historical evolution of human longevity. Health care spending decisions in turn are influenced by the state of medical technology and personal income. So far, the implications of the interaction between immunosenescence and inflammaging have not been considered in the economics literature. To close this gap, we integrate recent insights from medical science and gerontology into a calibrated economic model of physiological aging, understood as the intrinsic, cumulative, progressive, and deleterious loss of function (Arking, 2006). Specifically, we address the following research questions.

- (1) How does individual income, health status in young adulthood, and attitude towards prevention of infectious diseases (e.g. social distancing, masks, and vaccination) affect the mortality risk from infections, all-cause-mortality, life expectancy, and individual health spending? The answers to these questions shed new light on the drivers of socioeconomic heterogeneity in infectious mortality and the socioeconomic health gradient in general.
- (2) How much of the increase in observed life expectancy and in the value of life can be attributed to medical technological progress targeted to infectious and chronic diseases, health policy, and income growth? To answer these questions, we calibrate a stylized version of the U.S. epidemiological transition in the period 1860–2010. The calibrated model is used for counterfactual analyses that allow us, *inter alia*, to gauge the impact of medical technology for treatment of chronic diseases and disease prevention on infectious mortality risk, all-cause mortality, life expectancy, and the value of life.

In our model, individuals invest in their health to slow down health deficit accumulation and take measures to protect themselves from infectious diseases. Individuals, as they age,

accumulate chronic diseases (aging-related health deficits) at a quasi-exponential rate with age, consistent with empirical evidence (e.g. Mitnitski et al., 2002, 2006; Harttgen et al., 2013; Mitnitski and Rockwood, 2016; Abeliansky and Strulik, 2018a). That is, the presence of many health deficits favors the development of new deficits.¹ Health deficits are conveniently measured using the frailty index, which has been used in hundreds of studies in medical science and, more recently, in economics (e.g. Abeliansky and Strulik, 2018a,b; 2020; Abeliansky et al., 2020, Hosseini et al, 2022). The frailty index simply counts the relative number of health deficits and functional limitations that an individual has, given a long list of potential deficits (Searle et al., 2006). The feature that health in the health deficit model is measured by an established metric in medical science allows us to conveniently calibrate the model with morbidity and mortality data and then use it for quantitative exploration of the comparative dynamics of health behavior and health outcomes. In particular, the age-profile of both health deficits and infectious disease mortality in the calibrated model is closely connected to statistical evidence (population averages).

Our key findings can be summarized as follows. First, individuals with lower incomes or more initial health deficits have a significantly higher mortality risk from infectious diseases and all-cause mortality. Consequently, they have a significantly lower life expectancy, consistent with empirical evidence on the socioeconomic health gradient (e.g. Case and Deaton, 2005; Chetty et al., 2016; Marmot, 2015). The effectiveness of health care depends crucially on its timing. Beginning protection against infectious diseases at an older age (e.g. at age 60 rather than as a young adult) reduces life expectancy in quantitatively important ways.

Second, counterfactual analysis highlights the importance of the interaction between infections and chronic health deficits during the epidemiological transition. We estimate that the epidemiological transition (starting in the second half of the 19th century) was the main driver of the increase in life expectancy in today’s advanced countries, far more important than medical technological progress on the treatment of chronic diseases (such as the ‘cardiovascular revolution’, starting in the the 1960s, analyzed in Hansen and Strulik, 2017). Our analysis suggests that about two thirds of the gain in the value of life at age 65 can be explained by the epidemiological transition. Increases in income have had a significant impact on life expectancy, especially since the mid-20th century, as they have enabled people to take advantage of medical technological progress.

The remainder of the paper is organized as follows. The next section briefly reviews recent findings from the gerontological literature on the relationship infections and physiological aging. Section 3 discusses our contribution to the related literature. Section 4 introduces the model and discusses the comparative statics of protection and treatment of infectious diseases. Section 5 calibrates the model for an average American in the year 2010 and the probability to die from influenza or pneumonia, which were the leading causes of death from infection in the pre-Covid era. Section 6 presents the comparative dynamics of medical technology and individual

¹ Consequently, the health of unhealthy people (with many health deficits) erodes faster than that of healthy people. An example of the self-productive nature of health deficits is that even mild deficits like difficulty to move or high blood pressure are known to cause cardiovascular disease.

characteristics such as income, frailty, and anti-protection attitudes. In Section 7, the model is calibrated for a stylized epidemiological transition 1860-2010 and then used for counterfactual experiments (or as-if scenarios) in order to assess the role of progress in the prevention and treatment of infectious and chronic diseases. Section 8 concludes the paper.

2. IMMUNOSENESCENCE, INFLAMMAGING, AND PHYSIOLOGICAL AGING

As this is the first study in economics incorporating the relationship between chronic health deficits and infections, it is worth briefly explaining the physiological foundation of their interaction. Immunosenescence is caused by the deteriorating functionality of the innate immune system characterized by ineffective pathogen recognition and macrophage activation. The process is linked to a decline in number and functionality of naïve T- and B-cells. For instance, infections lead to the expansion of memory T-cells (adaptive immunosenescence), causing a decline in the T-cell repertoire diversity (e.g. Weiskopf et al., 2009; Aiello et al., 2019; Santoro et al., 2021). It has also been shown that the thymus (the organ where T-cells are built) has lost 60 percent of its original size by the age of 65 (Palmer, 2013). Immunosenescence is strongly related to the pathological condition of chronic inflammation that causes increasingly dysregulated immune responses, including reduced efficacy of vaccination in elderly persons (Goronzy and Weyand, 2013; Nakaya et al., 2015; McElhaney et al., 2020).² As they age, most elderly people develop a persistent pro-inflammatory condition (with great heterogeneity in levels) which can cause a variety of chronic diseases such as cardiovascular disease, lung and kidney disease, some cancers, rheumatoid arthritis, cognitive disability, osteoporosis, periodontitis, sarcopenia, type 2 diabetes, as well as Alzheimer and Parkinson disease (e.g. Franceschi and Campisi, 2014; Bektas et al., 2018; Ferucci et al., 2018; Franceschi et al., 2018). Chronic inflammation, referred to as inflammaging, has in fact been characterized as one of the core physiological mechanisms associated with the aging process (Kennedy et al., 2014).

A particularly severe variant of dysregulated immune function drew broader attention in the course of the Covid-19 pandemic. In a so-called cytokine storm, an infection causes a rapid and uncontrolled release of inflammatory signalling molecules that leads to inflammation of major organs such as the lungs, kidneys, and heart and may eventually cause organ failure and death (e.g. Blagosklonny, 2020; Mueller et al., 2020).

Recent studies in medical science have emphasized the role of the immunological biography for immunosenescence and inflammaging (e.g. Franceschi et al., 2017, 2018).³ An extensive literature shows, for instance, the link from infections caused by bacteria such as the *Borrelia* species and *Mycobacterium leprae* and viruses such as HIV and herpes to chronic pain and inflammatory bowel disease (ulcerative colitis and Crohn’s disease).⁴ Moreover, it is well known that

² By contrast, acute inflammatory responses play a central role in the healing process of injuries and to fight infections. They disappear when they are no longer needed.

³ Other stressors include obesity, intestinal dysbiosis, diet, social isolation, psychological stress, disturbed sleep, and exposure to air pollutants, hazardous waste products, industrial chemicals, and tobacco smoking. See Santoro et al. (2021) for a review on causes and health consequences of immunosenescence and inflammaging.

⁴ See Cohen et al. (2021) for a review. The literature has particularly highlighted the role of the Humane Cytomegalovirus (HCMV) status (e.g. Aiello et al., 2019), HIV (Desquilbet et al., 2009), and SARS-CoV-2 exposure (Huang et al., 2020) for functional T-cell changes.

streptococcal infections can cause rheumatic heart disease, which carries the risk of potentially fatal heart valve damage (Jones, 1956). In a similar vein, periodontitis is suspected to cause cardiovascular disease and diabetes mellitus via inflammatory mediators (Li et al., 2000). Another important channel from infection to chronic disease that is likely involving inflammatory processes concerns the widespread *Helicobacter pylori* infections that have been made responsible for inflammation of the stomach lining (gastritis), stomach ulcers, stomach cancer, and even coronary disease (Harvey et al., 2002).

The most recent insight to inflammaging were obtained from studying the long-run implications of Covid-19. The so-called Long-covid syndrome describes enduring symptoms like fatigue, shortness of breath, and cognitive dysfunction lasting months or even years after the onset of Covid-19. Moreover, it is now well confirmed that SARS-CoV-2 infections (typically those leading to more severe Covid-19 disease) cause respiratory diseases, diabetes, and cardiovascular diseases at higher rates than could be expected in the general population (Ayoubkhani et al., 2021).

In turn, physiological aging enhances the susceptibility to infections and infectious mortality risk. It includes, for instance, the decreased function of epithelial barriers (the protective surface tissue) of the skin, lung, and gastrointestinal tract, which facilitates the invasion of pathogens and increases the challenge for the aged innate immune system (Gomez et al., 2005). Mortality from influenza and pneumonia in the U.S., for example, increases about hundredfold from age 40 to age 85 (Vos et al., 2020). While chronological age is strongly correlated to the prevalence and severity of infectious diseases, the literature stresses that this does not imply that chronological age is causal (see, e.g., Santoro et al., 2021, among others). For example, the probability of a severe course of a coronavirus infection does not increase after celebrating another birthday, but after worsening of the inflammatory state that is closely related to chronic diseases (pre-existing conditions). A 70-year-old in good health may therefore be better protected from severe infections than a 50-year-old in poor health.

3. CONTRIBUTION TO THE LITERATURE

Our modeling of infectious diseases is partly inspired by the study of Cropper (1977) but also differs in important ways. Cropper discusses illnesses as random events in the framework of the health capital model (based on Grossman, 1972), assuming that individuals are exposed to random stressors (such as germs and viruses) and that illness is caused by sufficiently large exposure. The threshold level above which a random shock turns into illness depends on individual health capital.⁵ Utility experienced in sickness is normalized to zero, which allows for a straightforward modeling in terms of maximization of expected lifetime utility. We built on these ideas and take into account (i) that the presence of more chronic diseases increase susceptibility, severity, and mortality from infectious diseases and (ii) the feedback effect of infections on developing chronic deficits. In contrast to Cropper, we explicitly model and distinguish mortality risk from infections and chronic conditions. For example, Cropper states that her model only applies to

⁵ In Cropper (1977), as health capital depreciates, individuals become more susceptible to illness and spend a greater part of their life being sick. This creates an incentive to invest in expanding health capital.

minor illnesses because a feedback effect of illness on the general health status is not accounted for, whereas such feedback loop is at the center of our analysis. Moreover, as health capital is unobservable and has no foundation in medical science and gerontology, we set up our analysis in the framework of the health deficit model (Dalgaard and Strulik, 2014) and measure health deficits by an established metric in medical science, the frailty index (e.g. Searle et al., 2008; Mitnitski et al., 2002, 2016). This allows us to calibrate the model with data and use it for quantifiable comparative static analysis and counterfactual experiments.⁶

The original health deficit model by Dalgaard and Strulik (2014) focussed on the relationship between income and life expectancy. The framework has been extended and applied to various health economic problems like the historical evolution of years spent in retirement (Dalgaard and Strulik, 2017), the effects of health care rationing on life expectancy (Boehm et al., 2021), health and welfare effects of pension reforms in presence of a socioeconomic health gradient (Grossmann et al., 2021), addiction and self-control (Strulik, 2018, 2019a,b), life expectancy gaps across gender and marriage status (Schünemann et al., 2017a, 2020), and adaption to deteriorating health (Schünemann et al., 2017b). However, the literature has so far ignored how the life course of infections influences the development of chronic health deficits and how pre-existing chronic conditions affect the susceptibility to infections. Our main methodological contribution is to integrate into the health deficit model infectious (acute) diseases, to differentiate them from chronic diseases (aging-related health deficits), and to consider the interactions between infections and chronic diseases. In doing so, we account for the endogeneity of physiological aging and infections by modeling expenditures on treating and protecting infectious diseases and preventing and healing chronic health deficits.

The feedback loop from infection to inflammation and chronic disease implies that generations with lower lifetime exposure to infectious diseases have experienced fewer challenges and slower immune system deterioration and therefore, *ceteris paribus*, will exhibit fewer chronic diseases in old age, as argued in Finch and Crimmins (2004) and Finch (2010). For instance, Finch and Crimmins (2004) point out that in countries with currently low mortality, *Helicobacter pylori* infections and periodontitis have been on decline because of improved public health efforts and improved dental hygiene, respectively. They argue that “changes in the epidemiological environment that occur within a given historical period would affect surviving members of cohorts for the rest of their lives” (p. 1737). With respect to the history of public health, these insights refute the traditional view of an epidemiological transition of separate stages of infectious and chronic disease decline (Omran, 1971). A modern and refined version of the epidemiological transition acknowledges that the decline of infections was accompanied by a simultaneous decline in chronic diseases (Mercer, 2018). For the U.S., it has been shown that chronic diseases and functional limitations among older men decreased by over 60 percent from the early 20th century to the 1970s, and that a significant portion of the decrease was due to

⁶ The health capital approach in Cropper (1977) inherits from Grossman (1972) the counterfactual feature that the health of healthy people (with high stock of health capital) erodes faster than the health of unhealthy people (with low stock of health capital). The literature has discussed various counterfactual predictions that are implied by this feature (e.g. Wagstaff, 1986; Case and Deaton, 2005; Almond and Currie, 2011; Strulik, 2015). By contrast, in the health deficit model, unhealthy people age faster.

declining infections (Costa 2000, 2002). We take these measured feedback links from infections to chronic health deficits into account in the calibration of our model. The calibrated model is then used for counterfactual historical experiments (in the spirit of Fogel, 1964) in order to assess the role of the epidemiological transition for advances in human life expectancy and the value of life, and to contrast it with the role of medical progress for the treatment of chronic diseases.

The epidemiological transition has recently entered mainstream economic analysis, but primarily as a tool in empirical studies to identify the impact of health on economic outcomes (e.g. Acemoglu and Johnson, 2007; Cervellati and Sunde, 2011; Hansen and Strulik, 2017; Klasing and Milionis, 2020). Goenka and Liu (2020) explore the role of infectious disease dynamics in an endogenous growth model. Since the onset of the Covid-19 pandemic, epidemiological dynamics have been integrated in various forms of macroeconomic models of which some also take into account chronological (but not biological) age as a determinant of the rates of infection, death, and recovery (e.g. Acemoglu et al., 2021; Brotherhood et al., 2020). The accumulation of chronic health deficits as the main driver of human aging and longevity, its interaction with infections, and the role of infections on health behavior are not considered in this literature.

Our contribution to the literature is thus twofold. First, we provide a novel and quantifiable framework for modeling physiological aging in economics that incorporates behavioral responses and captures fundamental insights from the gerontological and medical literature for the interaction between infections and chronic diseases. Second, we assess the implications of this feedback loop on the socioeconomic health gradient and the course of the historical epidemiological transition.

While we are not the first to address the socioeconomic health gradient with the health-deficit approach, our focus on the interaction between chronic diseases and infectious diseases allows us to explain why socially disadvantaged groups have a higher risk of mortality from infectious diseases (Wachtler et al., 2020). The focus of our study of the epidemiological transition is how the disease environment and medical technological progress affected human aging and longevity. In contrast to the literature initiated by Hall and Jones (2007), we neither want to explain the development of aggregated health expenditure (relative to income) nor the feedback effect of health expenditure on medical progress (as e.g. Frankovic and Kuhn, 2023). Rather, we aim to calibrate our model to match observed health expenditure and analyze health spending responses in comparative-dynamic analysis. For example, our study examines how infectious diseases affect longevity, physiological aging and the value of life not only through the development of the disease environment, but also through individual characteristics such as income, health behavior, and pre-existing health conditions.

4. THE MODEL

4.1. Infectious and Chronic Diseases. Consider an individual exposed to random health shocks and suppose that a health shock leads to serious illness only if it exceeds a threshold that depends on the individual's health status. Specifically, suppose the size of infection shocks, s , is Pareto-distributed with probability density function $f(s) = \nu\tilde{\beta}^\nu s^{-(\nu+1)}$, with $s \in \{1, \infty\}$ and

cumulative distribution function $F(s) = 1 - (\tilde{\beta}/s)^\nu$, $\nu > 0$, $\beta > 0$. Only shocks of sufficient strength greater than \bar{s} result in an infection that is potentially lethal. The threshold level \bar{s} is inversely proportional to the frailty of the body, reflecting the fact that old and frail persons are more susceptible to infectious diseases. Health deficits are measured by the frailty index D , which counts the relative number of health deficits a person has from a long list of potential deficits (Mitnitski and Rockwood, 2002; Searle et al., 2008; Mitnitski and Rockwood, 2016). Setting $\bar{s} = 1/D$, we obtain the probability of severe infection as βD^ν with $\beta = \tilde{\beta}^\nu$. In this parsimonious model, the parameter β affects both the prevalence of diseases and their severity. A higher value of β means that at any level of D more individuals become severely sick, all other things being equal.

Individuals can reduce the probability of a serious infection by methods of self-protection and (if available) vaccination. Moreover, there may exist an effective treatment. For simplicity we measure the effort in protection from disease (i.e. prevention and treatment) in one choice variable, p . This feature reflects that infection in a vaccinated person results in less severe disease and therefore requires less treatment to achieve a given risk of death from infection. The choice of p is bounded, $p \in [0, 1]$. The lower bound is intuitively plausible since less than no protection is not feasible. It is also plausible that an upper bound of p exists (full protection) albeit its value is arbitrary. Here we consider without loss of generality an upper bound of one. It facilitates the interpretation of p as the percentage of protection and treatment measures taken. When calibrating the model, it turns out that individuals most often choose either the lower or the upper bound as the constrained optimal solution. This seems a plausible result, since many conceivable interventions are in fact bivariate (to vaccinate or not to vaccinate). The efficacy of disease prevention and treatment is captured by parameter $\epsilon \in [0, 1)$. We assume that the probability of death from infection at any increment of time is given by $G(p, D) \equiv (1 - \epsilon p)\beta D^\nu$ which also serves as a measure of the severity of diseases caused by an infection. Protection reduces mortality risk by avoiding infections and treatment reduces it by combating germs and accelerating recovery. For given level of protection effort, mortality risk increases in health deficits D , which captures the feature that the immune system of frail people responds less well to protection and treatment (McElhaney et al., 2020). The value of ϵ represents the state of medical technology and medical knowledge in infectious disease prevention and treatment. The complementarity between ϵ and p captures the feature that advances in medical progress become only effective in reducing deaths from infection when applied by individuals. For example, if during the epidemiological transition more and better vaccines and antibiotics are developed, captured by larger ϵ , the individual benefits from these advances only according to his/her application of the new medical products.

We distinguish direct and indirect effects of infections: the direct effects consist of the development of acute health conditions which reduce the survival probability from infection, $S_I(p, D) \equiv 1 - G(p, D)$. Moreover, individuals develop chronic, aging-related diseases, that are indirectly affected by infections, in addition to other causes. This feedback effect (documented in Section 2) is specified below. The survival probability from chronic diseases, $S_C(D)$, depends on the health deficits that have been accumulated, $S'_C(D) < 0$. There exists an upper

limit of health deficits, \bar{D} , beyond which survival is impossible, $S_C(D) = 0$ for $D \geq \bar{D}$. Death is thus conceptualized as a stochastic event, which occurs with higher probability when many chronic health deficits have been accumulated, *inter alia* but not exclusively via infections. Let $S(p, D) \equiv S_I(p, D)S_C(D)$ denote the aggregate survival probability. Let t denote individual age and $\mathcal{S}(t) \equiv S(p(t), D(t))$, $\mathcal{S}_I(t) \equiv S_I(p(t), D(t))$, and $\mathcal{S}_C(t) \equiv S_C(D(t))$. The conditional (i.e. age-specific) all-cause mortality rate at age t is then obtained as $m(t) \equiv -\dot{\mathcal{S}}(t)/\mathcal{S}(t)$ and the conditional mortality rate that is directly related to infectious diseases is $m_I(t) \equiv -\dot{\mathcal{S}}_I(t)/\dot{\mathcal{S}}(t)$.⁷

Normalizing utility when dead to zero, total expected utility at age t is obtained as $\mathcal{S}(t)\mathcal{U}(t)$, where $\mathcal{U}(t)$ denotes potential utility. Potential utility depends on consumption c . In addition, it may be negatively impacted by disease prevention and treatment measures, p , reflecting, for example, time costs for doctor visits and attitudes towards wearing face masks or vaccinations. Summarizing, potential utility at age t can be written as $\mathcal{U}(t) \equiv U(c(t), p(t))$. We specify $U(c, p) = u(c) - \omega f(p)$,⁸ where $u(c)$ is consumption utility, $\omega \geq 0$ measures the degree to which disease prevention and treatment affect utility and $f(p)$ is an increasing and convex function assigning the curvature of disutility from protection measures. Consumption utility has the standard functional form $u(c) = \frac{c^{1-\sigma}-1}{1-\sigma}$ for $\sigma \neq 1$ and $u(c) = \log(c)$ otherwise, where $\sigma > 0$ is the inverse of the intertemporal elasticity of substitution (IES).

The development of chronic diseases can be accurately and straightforwardly described by the health deficit model (Dalgaard and Strulik, 2014), which, based on foundations in gerontology, assumes that individuals accumulate health deficits in a quasi-exponential way, and that exponential growth of health deficits D can be reduced by purchasing health goods and services, h . Here, we additionally take into account that infections promote the development of chronic diseases and that this process can be slowed down by disease avoidance and treatment spending p . As discussed in Section 2, infections affect chronic diseases in various ways, including direct and indirect cell transformation, chronic inflammation, and permanent tissue damage. We assume that the feedback effect from infection to chronic diseases depends on the severity of the infection, $G(p, D)$. Summarizing, chronic health deficits (measured by the frailty index D) develop according to the law of motion

$$\dot{D} = \mu [D - Ah^\gamma + B \cdot G(p, D) - a], \quad (1)$$

in which $\mu > 0$ is the (natural) force of aging. The parameters $A > 0$ and $0 < \gamma < 1$ characterize the state of medical technology in the prevention and cure of chronic diseases; the parameter $B > 0$ measures the impact of infections on health deficit accumulation; a is an environmental constant (a residual). Notice the distinct features of health care demand p and h . Health input h directly addresses the development of (chronic) aging-related health deficits: Examples are vascular stents, hearing aids, hip replacement etc. In modern societies, such health investments

⁷ Survival probabilities S_I and S_C are interdependent but this interdependence is deterministic and operates through the frailty index (i.e. both S_I and S_C are functions of D). There is no stochastic interdependence between S_I and S_C and thus the probability to survive is simply the probability of not dying from infectious diseases times the probability of not dying from chronic diseases.

⁸ In the calibrated model, potential utility $\mathcal{U}(t)$ is positive at any age, such that individuals indeed prefer to live longer.

make up the bulk of all health expenditure. Disease prevention and treatment measures p have an indirect effect on health deficit accumulation, since exposure to infectious diseases favors the emergence of new chronic health deficits. The initial age is normalized to zero and will be set to 20 years in the calibrated model. Initial health deficits are given by $D(0) = D_0 > 0$.

4.2. Individual Choice. Individuals spend their income on consumption c , expenditure for prevention and treatment of infectious diseases p , other health investments h , and saving for retirement and health expenditure in old age in form of fair annuities. This means that their budget constraint is given by

$$\dot{k} = (r + m)k + w - c - \phi_p \pi_p p - \phi_h \pi_h h, \quad (2)$$

in which k is household wealth, r is the interest rate, m is the mortality rate, and w is a flow of exogenous labor-related income, which is wage income (after taxes) before retirement and pension income after retirement. π_p denotes the price of prevention and treatment of infections and π_h is the price of other health goods. ϕ_p and ϕ_h are out-of-pocket (or coinsurance) ratios. Initial wealth is given by $k(0) = k_0 \geq 0$.

Individuals maximize expected lifetime utility

$$V = \int_0^T S(p(t), D(t)) [u(c(t)) - \omega f(p(t))] e^{-\rho t} dt$$

in which ρ is the time preference rate and T is the endogenous planning horizon. This modeling follows the medical literature of the frailty index, which supports the view that there exists an upper limit of the frailty index above which survival is impossible (Rockwood and Mitnitski, 2006). As individuals die for sure when state variable D reaches its upper limit \bar{D} , the terminal condition $D(T) = \bar{D}$ holds. Moreover, we assume a terminal condition for wealth $k(T) = \bar{k} \geq 0$. Since the terminal values of the state variables are given while the terminal time T is free, the problem constitutes a free-terminal-time problem. In sum, the problem is to maximize V subject to the law of motions (1) and (2), the initial conditions, $D(0) = D_0 \geq 0$, $k(0) = k_0 \geq 0$, and the terminal conditions for k and D . Recalling $S(p, D) = S_I(p, D)S_C(D)$, the associated current-value Hamiltonian is given by

$$\begin{aligned} \mathcal{H} = & S_I(p, D)S_C(D) [u(c) - \omega f(p)] + \lambda_k [(r + m)k + w - c - \phi_p \pi_p p - \phi_h \pi_h h] \\ & + \lambda_D \mu [D - Ah^\gamma + B \cdot G(p, D) - a], \end{aligned}$$

in which λ_k and λ_D are the shadow prices (co-state variables) of wealth and health deficits. Using $S_I(p, D) = 1 - G(p, D)$ and $G(p, D) = (1 - \epsilon p)\beta D^\nu$, the first order conditions for an interior solution of controls c , h , and p are:

$$S(p, D)u'(c) = \lambda_k \quad (3)$$

$$-\lambda_D \mu \gamma A h^{\gamma-1} = \lambda_k \phi_h \pi_h. \quad (4)$$

$$\epsilon \beta D^\nu S_C(D) [u(c) - \omega f(p)] - \lambda_D \mu \epsilon \beta B D^\nu = S(p, D)\omega + \lambda_k \phi_p \pi_p. \quad (5)$$

The left-hand sides of the first order conditions show the marginal benefits and the right-hand sides the marginal costs. Condition (3) equates the expected marginal utility from consumption

with the marginal cost from consumption, which is one unit of savings evaluated at the shadow price of wealth, λ_k . Condition (4) requires that the marginal benefit of health investments equals the marginal cost. The marginal benefit is the slowdown of health deficit accumulation caused by a unit of health investments, $\mu\gamma Ah^{\gamma-1}$, evaluated at the shadow price of health deficits, λ_D . Notice that health deficits are not an asset like k but a ‘liability’. The accumulation of health deficits contributes negatively to the objective function such that the shadow price of health deficits, λ_D , is negative. The marginal cost of health input h consists of the monetary expenditure per unit evaluated at the shadow price of wealth. The marginal benefit of disease prevention consists of the gain in expected utility, which is utility multiplied by the gain in survival probability (the first term on the LHS of (5)) and the caused slowdown in health deficit accumulation due to fewer infections, evaluated with the shadow price of health deficits (the second term on the LHS of (5)). The marginal cost consists of the expected utility cost of prevention and the monetary costs evaluated at the shadow price of wealth.

Equations (3) and (4) can be used (jointly with $S(p, D) = [1 - (1 - \epsilon p)\beta D^\nu] S_C(D)$) to eliminate the shadow prices in (5). We obtain:

$$\begin{aligned} 0 &= \frac{\epsilon\beta D^\nu}{1 - (1 - \epsilon p)\beta D^\nu} [u(c) - \omega f(p)] - \omega f'(p) - u'(c) \left[\phi_p \pi_p - \frac{\phi_h \pi_h}{\gamma Ah^{\gamma-1}} \epsilon \beta B D^\nu \right] \\ &\equiv F(p; D, \epsilon, \beta, \phi_p \pi_p, \phi_h \pi_h, \omega, c, h) \end{aligned} \quad (6)$$

Let \tilde{p} be implicitly defined by $F(\tilde{p}; \cdot) = 0$. It has the interpretation as an interior solution for the level of protection against infections for given levels of consumption c and health investments h . The full partial solution (for given c and h) is given by $p = \min\{1, \max\{0, \tilde{p}\}\}$. It will turn out that in many of the applied cases, individuals select a corner solution (no protection or full protection). A discussion of the interior solution \tilde{p} is nevertheless interesting since the solution characterizes the *propensity* for infection prevention and treatment.

PROPOSITION 1. *For given levels of consumption c and health input h , the propensity for infection prevention \tilde{p} is increasing in the efficacy of protection ϵ , the severity of the disease β , the frailty index D , the impact of infections on chronic diseases B , and the cost of health investments $\phi_h \pi_h$. It is declining in the utility cost ω and the monetary cost of prevention $\phi_p \pi_p$.*

The proof is obvious from inspection of (6), noting that partial derivative $F_p(\tilde{p}; \cdot) < 0$ etc. and applying the implicit function theorem. The comparative static results confirm our intuition of how rational individuals confronted with infectious diseases should behave. The results can also be used to explain apparently irrational or ill-considered behavior. To see this, we can distinguish between beliefs and facts. The outcome of the individual calculus is based on beliefs about the size of parameters, while actual infections and the aging process are subject to actual circumstances. For example, suppose that a ‘corona denier’ perceives the threat of the disease β and its long-term consequences B to be lower than they actually are. He will then exert himself too little and perhaps not at all in order to prevent infection.

For our later analysis, we notice the model’s prediction that, *ceteris paribus*, frail persons (with pre-existing conditions) exhibit a greater propensity of disease prevention. Since individuals accumulate more health deficits with advancing age, the model explains why the propensity to

protect against diseases (by e.g. vaccination) increases with age. The result underscores that it is not chronological age that motivates older people to use protection but the increasingly frail and vulnerable state of their health that increases the benefits of protection. A protection-averse person will therefore eschew protection in young and middle age and only begin to seek it in old age, when the gains in health outweigh the utility loss from using protection.

The model also predicts that rich persons, who experience a high level of utility from consumption and for whom marginal utility from consumption $u'(c)$ is low display a greater propensity for protection. Protection provides them a greater increase in expected utility and, in terms of forgone utility, protection costs are lower. With respect to policies, we see that subsidies to prevention costs (or, in case of negative π_p monetary rewards) can incentivize prevention and that this mechanism is largest for poor individuals whose marginal utility from consumption is high due to a low level of consumption.

The rest of the model is less straightforwardly assessed. We show in the Appendix that optimal consumption and health expenditure evolve according to

$$\frac{\dot{c}}{c} = \frac{r - \rho}{\sigma} \tag{7}$$

$$\begin{aligned} \frac{\dot{h}}{h} = & \frac{1}{1 - \gamma} [r + m - \mu - \mu\nu(1 - \epsilon p)\beta BD^{\nu-1}] \\ & - \frac{1}{1 - \gamma} \frac{\mu\gamma Ah^{\gamma-1}}{\phi_h \pi_h S_I(p, D) S'_C(D) c^{-\sigma}} [\nu(1 - \epsilon p)\beta D^{\nu-1} S_C(D) - S_I(p, D) S'_C(D)] \left[\frac{c^{1-\sigma} - 1}{1 - \sigma} - \omega p \right]. \end{aligned} \tag{8}$$

Eq. (7) is the well-know Euler equation for optimal consumption. Eq. (8) is the Euler equation for health investments. The first part of the RHS, $(r + m - \mu)/(1 - \gamma)$, coincides with the outcome from the standard health deficit model (Dalgaard and Strulik, 2014), where $\beta = 0$ (no infections) and $S'_C(D) = 0$ (no survival risk from chronic diseases). It reflects the general trade-off between health investments now or later in life. When the interest rate is high, individuals prefer to save and to delegate health investment to later in life, which leads to a steeper increase of health investments with age (a larger growth rate \dot{h}/h). In contrast, when health deficits develop at a high rate μ , individuals prefer to invest in health already early in life and thus display are flatter age-profile of health investments. Here, we have two additional terms. The last term in the first line of (8) reduces the age gradient of expenditure by more in a more infectious environment (higher β) or when the impact of infections on chronic diseases are larger (higher B). The term in the second row of (8) comprises effects running through survival probabilities and is negative. Also this additional term thus flattens the age-expenditure profile and it is absolutely larger, the larger β . Moreover, the term is positively related to the marginal impact of chronic diseases on the probability of survival, $S'_C(D)$.

The full model consists of the system of ordinary differential equations (1)–(2) and (7) and (8), the algebraic equation (6), the initial values $D(0) = D_0$ and $k(0) = k_0$, the final values $D(T) = \bar{D}$ and $k(T) = \bar{k}$, and the transversality condition $\mathcal{H}(T) = 0$, which together determine the unique solution for the lifetime trajectories of c and h and the age T at which the terminal conditions are reached. The transversality condition $\mathcal{H}(T) = 0$ is the boundary condition that

needs to hold for problems of free terminal time, see Hartl and Sethi (1983) for derivation and detailed discussion of the transversality condition. The solution of the calibrated model is obtained by the relaxation method of Trimborn et al. (2008), which solves the non-linearized dynamic system up to a user-defined approximation error (which is set to 10^{-5}).

5. BENCHMARK CALIBRATION

The benchmark model is calibrated for an average U.S. American man who starts life in the year 2010 at model-age $t = 0$ when he is 20 years old. We set $\mu = 0.043$, according to the estimate of the force of aging for Canadian men in Mitnitski et al. (2002a) and initial health deficits to $D_0 = 0.027$, as obtained from the same study. Consistent with an absence of a bequest motive, we set $k_0 = \bar{k} = 0$, i.e., the benchmark individual receives no inheritance and leaves no bequest. Assuming that people save in terms of real estate ownership and equity, we set $r = 0.07$, as estimated by Jorda et al. (2017) for the long-run rate of return on equity and real estate and $r = \rho$ such that consumption is constant over the life cycle (as observed for childless households, Browning and Ejrnæs, 2009). We will show the robustness of results with respect to modified calibrations assuming lower interest rates.

In order to be comparable to previous applications of the health-deficit model, while being flexible in discussing the role of public health expenditure, we normalize the total price of health expenditure $\phi_h \pi_h$ to unity and then calibrate ϕ_h as one minus the government share of health expenditure, which is set to 0.47, according to the numbers provided in Getzen (2017, Table 8). We set $\phi_p = \phi_h$ and use the information from BEA (2022) that, in 2010, 4.0 percent of health expenditure was on infectious diseases to calibrate π_p . We implicitly assume that private insurance is actuarially fair and creates no moral hazard problems. We set w to 27,928, according to the median annual wages of single men in 2010 (in 2010 dollars), as reported in BLS (2012).⁹ The survival probability from chronic diseases is assumed to follow a logistic decay function, as in Schünemann et al. (2017b), parameterized as $S_C(D) = (1 + \psi)/(1 + \psi \exp(\xi D))$.

We assume that the utility cost function is linear, $f(p) = p$. A linear function allows us to solve explicitly for the optimal p , which leads to the convenient feature that the life cycle problem can be represented by a system of ordinary differential equations. A linear utility cost function implies that the optimal p is almost always a corner solution. As argued above, a corner solution is plausible because for many treatments there exists a prescription dose (e.g. three spoons of cough syrup every day). While it is possible to deviate from the prescription, this behavior would be inefficient and perhaps even unhealthy. So the decision is to take the prescribed treatment ($p = 1$) or not ($p = 0$). For many preventive measures the same logic holds (take one vaccination per year or not). While there may be counterexamples we think that in the majority of applications a corner solution is plausible. Moreover, the corner solution is not

⁹ Scaling w allows us to interpret model health expenditure and the value of life as real dollar values. The exogenous salary can be micro-founded easily in the present context that features an exogenous world market real interest rate. Suppose the numeraire good is produced by a price-taking representative firm according to the linear-homogenous production function $F(K, ZL)$, where K is capital input, L is labor input and Z captures the state of technology. For given time-invariant user costs of capital (interest rate plus the capital depreciation rate), the wage rate is proportional to Z . Below we feed in the historical wage series which means that we treat technological progress (changes in Z over time) as exogenous.

a unique feature of the linear cost function. In Appendix B we show that corner solutions are also a salient feature of quadratic utility cost functions and that, in any case, the linear solution is a good approximation of the non-linear solution.

For the benchmark case, we assume $\omega = 0$, implying that the average person has no attitudes or preferences against protecting or treating infectious diseases and thus will choose $p = 1$ at any age. This is an interesting case as it delivers the health outcomes when taking full advantage of the available protection and treatment technology. We address the potential variation of protection attitudes in the population with a sensitivity analysis and a robustness check. In the sensitivity analysis, we explore health behavior and health outcomes of individuals with (large) protection aversion using comparative dynamic analysis. In the robustness check, we recalibrate the model for a highly protection averse individual, re-run the comparative dynamic analysis, and compare results to those of the benchmark case.

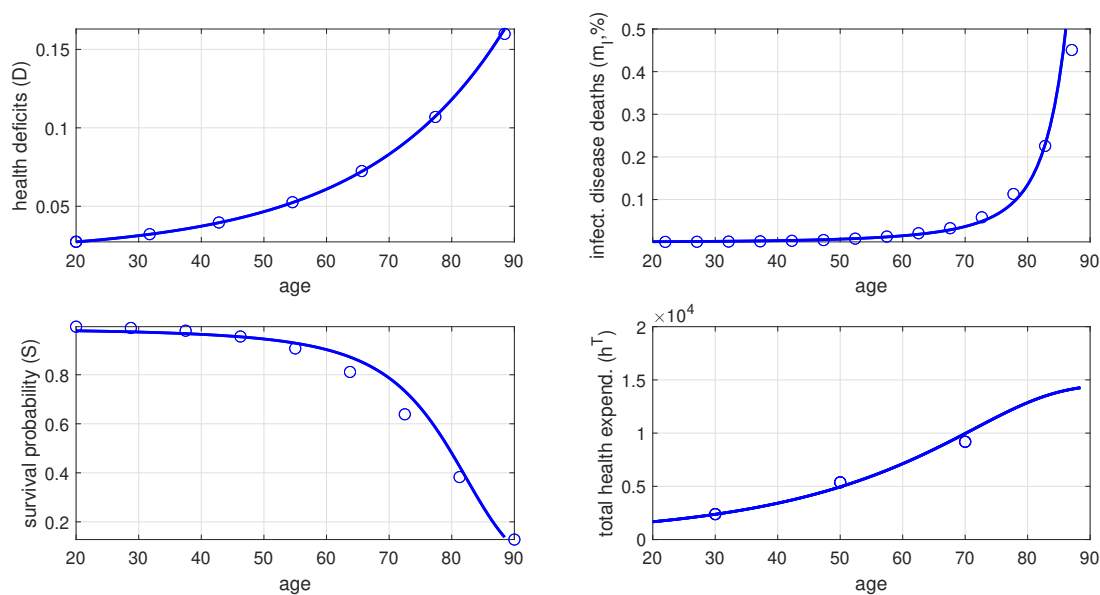
A challenging issue is the calibration of the feedback effect of infections on chronic diseases, determined by parameter B in eq. (1). As discussed above, the recent literature provides ample evidence for the existence of inflammaging, although to our knowledge no study has attempted to quantify it. We assume that B is a physiological constant and therefore time-invariant. We follow standard calibration techniques and calibrate B along with the other parameters such that the model’s predictions provide the best fit of the historical trajectory of life expectancy at age 20 when we feed into the model the historical paths of income per capita, the public share of total health expenditure, and estimates of medical progress in the prevention and treatment of infectious and chronic diseases (see Section 7 for details). We address the involved parameter uncertainty with a sensitivity analysis (Subsection 7.4). The main purpose of our quantitative experiments is not to uncover the ‘true value’ of B but to highlight the potential power of the inflammaging channel. In comparative dynamic analysis we therefore always compare the predictions derived from the calibrated value of B with the conventional view of no inflammaging ($B = 0$).

In addition, we also fit the following stylized facts: (a) The model predicts the actual accumulation of health deficits over a lifetime (as estimated by Mitnitski et al., 2002a). (b) The predicted survival curve fits the actual survival curve for American men (obtained from estimates in Strulik and Vollmer, 2013) and implies a life expectancy at 20 of 57.1 years (expected death at 77.1 years), which was the life expectancy of a 20-year-old American male in 2010 (NVSS, 2014). (c) Predicted health expenditure fits health care expenditure of American men in 2010 at the age of 30, 50, and 70 (MEPS, 2010). (d) The predicted death rate from infectious diseases fits the age profile for deaths from lower respiratory infection for U.S. males, as obtained from the Global Burden of Disease Study (GBD; Vos et al., 2020). This calibration target takes into account that even before Covid-19 lower respiratory infections (including pneumonia, bronchitis, and influenza) were by far the most common cause of death from infectious diseases in the U.S. (GBD; Armstrong, 1999). The calibration led to the estimates $B = 0.5$, $a = 0.016$, $\gamma = 0.13$, $\sigma = 1.1$, $\nu = 2$, $\psi = 0.011$ and $\xi = 38.9$ for time-invariant parameters, and $\beta = 4.2$, $\epsilon = 0.85$ and $A = 0.000155$ for the state of medical technology in recent times (benchmark calibration

for the experiments in Section 6). All calibrated parameters are summarized in Table A.1 in the Appendix.

While most of the calibrated parameters are latent, the estimated value of σ agrees well with studies suggesting that the IES is close to unity (Chetty, 2006; Layard et al., 2008). As a non-targeted result, the model predicts a value of life of \$ 5.7 million, which is close to the estimate of \$ 6.3 million assumed in Murphy and Topel (2006).¹⁰

FIGURE 1. Life Cycle Health with Chronic and Infectious Diseases: Benchmark Model vs. Data (Males)



Lines: model prediction for an average American man; dots: targeted data (from Mitnitski et al., 2002, for health deficits; Vos et al., 2020, for infectious disease mortality; Strulik and Vollmer, 2013, for survival probability; MEPS, 2010, for health expenditure); see text for details.

Figure 1 shows the predicted life cycle trajectories for health deficits, probability of death from infectious disease, survival probability, and total health expenditure $h^T \equiv \pi_h h + \pi_p p$. Lines show the model outcome and dots indicate the targeted data. The age-specific mortality rate from infectious diseases, m_I , increases strongly with age. Interestingly, the predicted deaths from infections grow at an almost constant rate with age. The average growth rate is 11 percent. This is slightly higher than the average growth rate of all-cause mortality, which is predicted to grow on average at a rate of 10 percent, in line with estimates of Gompertz law of mortality (Strulik and Vollmer, 2013). Notice that the model does not assume that advancing chronological age *causes* the increase in infectious disease mortality. The increase has a physiological explanation in that infectious diseases are more lethal for frail individuals who have accumulated many chronic health deficits. Figure 1 suggests that the evidence on the age-profile of the infectious disease mortality can well be explained by life cycle behavior jointly with two features of our

¹⁰ The value of life at some age t_0 is computed as expected lifetime utility divided by the marginal utility from consumption at age t_0 .

model: first, that the probability to die from an infection is strictly convex (quadratic) as a function of health deficits (chronic pre-existing conditions), as implied by $\nu = 2$; and second, that the frailty index accumulates according to eq. (1), including the feedback effect of infections on the development of chronic conditions. The age-profile of the infectious disease mortality is thus related to the displayed feature that frailty index is increasing exponentially with chronological age.

6. HEALTH OUTCOMES AND HEALTH BEHAVIOR: PRESENT

6.1. Benchmark Model. In this section, we present the comparative dynamics of the benchmark model calibrated for the present for men starting protection ($p = 1$) at age 20 (i.e. from the beginning), since $\omega = 0$. In order to discuss health outcomes in a concise way, we summarize them in seven simple statistics, as shown in the first line of Table 1. Predicted life expectancy is reported as ΔLE in terms of deviation in years from the calibrated benchmark run. The other statistics are measured in percentage deviation from benchmark: $\Delta m_I(85)$ reports the relative change in infectious disease mortality rate at age 85 and $\Delta m(85)$ is the relative change in the mortality rate of all-causes at age 85. The relative change in health deficits at age 65 is reported as $\Delta D(65)$. A similar statistic could be reported for all other years of life, but age 65 seems particularly interesting as it describes the state of health of older people at the transition to retirement. The statistic $\Delta \tilde{h}$ reports the relative change in expected lifetime health expenditure $\tilde{h} \equiv \int_0^T \mathcal{S}_C \mathcal{S}_I (\pi_h h + \pi_p p) dt$. It can be interpreted as change in average health expenditure (of a society consisting of average Americans at all ages). Finally, $\Delta V(20)$ and $\Delta V(65)$ report the relative change in the value of life experienced at age 20 and age 65.

Case 1) evaluates the gains from infectious disease protection and treatment from the perspective of the average American man in the year 2010. For zero efficacy of protection/treatment, i.e. for $\epsilon = 0$, the individual experiences a more than sixfold increase of infectious disease mortality at age 85 and an expected loss of more than 8 years of life. Increased illness from infections leads to faster aging through development of chronic health deficits. Health deficits at age 65 are predicted to increase by 23 percent such that all-cause mortality rate at age 85 increases by 3 percent. Most of the increases of infectious disease mortality and the repercussions from chronic diseases are experienced at later ages such that the value of life at 65 declines strongly by almost 50 percent. Due to discounting of future payoffs, the value of life at age 20 declines more moderately, by ‘only’ 9 percent. Expected lifetime health expenditure increases mildly, by 4 percent, reflecting three counterbalancing effects. First, with zero efficacy of protection/treatment, there are no related expenses ($p = 0$). Second, any age is reached with lower probability than in the benchmark case. Because of these effects, taken for themselves, expected health expenditure should decline. The dominating effect, however, is the individual response of investing more to slow down the development of chronic diseases (increase in h) at any age. Health investments become more worthwhile with unprotected exposure to infectious diseases because avoiding chronic diseases and keeping a well functioning immune system protects against premature death from infectious diseases.

Table 1. Comparative Dynamics: Full Protection Case, Males

case	parameter change	remark	ΔLE	$\Delta m_I(85)$	$\Delta m(85)$	$\Delta D(65)$	$\Delta \bar{h}$	$\Delta V(20)$	$\Delta V(65)$
1)	$\epsilon = 0$	no protection/treatment	-8.16	611.88	3.24	23.38	3.98	-9.31	-46.77
2)	$B = 0$	no inflammaging	1.62	-0.83	0.07	-3.45	0.01	1.31	8.03
3)	$w = 1/2w_{bench}$	poorer individual	-1.32	3.87	4.99	3.14	-58.95	-54.59	-55.92
4)	$w = 1/2w_{bench}$ & $\epsilon = 0$	poorer individual and 1)	-9.22	639.53	8.23	27.37	-57.59	-58.09	-76.84
5)	$w = 2w_{bench}$	richer individual	1.56	-4.58	-5.51	-3.34	145.24	118.57	122.96
6)	$w = 2w_{bench}$ & $\epsilon = 0$	richer individual and 1)	-6.91	581.04	-2.27	16.52	152.93	94.73	32.02
7)	$D_0 = 1.1D_{0,bench}$	less healthy individual	-7.15	25.66	35.55	27.47	-7.38	-4.86	-38.00
8)	$D_0 = 1.1D_{0,bench}$ & $\epsilon = 0$	less healthy individual and 1)	-14.46	806.90	41.97	61.18	-6.07	-14.71	-78.27
9)	$\omega = 0.30$	protection starts age 40	-2.97	9.72	13.29	10.01	-7.34	-2.73	-18.11
10)	$\omega = 1.65$	protection starts age 60	-6.13	21.08	28.93	23.39	-25.23	-3.23	-49.91
11)	$\beta = 0$	eradication of infect. diseases	1.71	-100.00	-0.18	-3.43	-2.38	1.99	9.28

The table shows the predicted deviation of health behavior and health outcomes from the calibrated (male) benchmark individual with $\omega = 0$. The index *bench* identifies the calibrated benchmark value; ΔLE is the change in life expectancy measured in years. The other entries are relative deviations from benchmark measured in percent. $\Delta m_I(85)$ ($\Delta m(85)$) is the relative deviation of the mortality rate from infectious diseases (from all causes) at age 85. $\Delta D(65)$ is the relative change in health deficits at age 65. $\Delta \bar{h}$ reports the relative change in expected lifetime health expenditure. $\Delta V(20)$ is the relative deviation of lifetime utility, which equals the relative deviation of the value of life at 20. $\Delta V(65)$ is the relative deviation of the value of life at 65.

Case 2) illustrates the power of the feedback effect from infections to chronic diseases. For the hypothetical case that $B = 0$, i.e. without inflammaging caused by infectious diseases, the individual exhibits 3 percent fewer chronic health deficits at age 65 as well as fewer health deficits at any other given age. Better health at all ages increases life expectancy by 1.62 years compared to the benchmark run.

The socio-economic gradient of health is explored with help of cases 3) to 6). Case 3) considers a poor individual with half of benchmark income. As in detail discussed in Dalgaard and Strulik (2014), the interaction of strictly concave utility from instantaneous consumption and the linearity of lifetime utility in the length of life causes poorer people to invest less in their health, so that they accumulate health deficits more quickly and live shorter lives. This mechanism is also visible here. It is reinforced by the impact of chronic diseases on suffering severely from infectious diseases as well as by the feedback effect of infections on health deficit accumulation. The model predicts that poorer people experience higher mortality from infectious diseases (by 4 percent at age 85) because of their faster aging and thus less well-functioning immune systems. Life expectancy decreases by 1.3 years. Case 4) shows that poorer individuals are particularly affected by missing protection/treatment for infectious diseases. When $\epsilon = 0$, the infectious disease mortality rate at age 85 for poorer individuals increases by $(639 - 611 =) 28$ percentage points more than for the average American considered in case 1). In terms of value of life, however, the poorer individual suffers less from missing protection than the benchmark American. This result reflects the sad fact that poorer individuals are less likely to live long anyway.

Case 5) shows that an individual with twice the benchmark income invests more in health, ages more slowly and therefore experiences less mortality from infectious diseases. Comparing case 6) with case 5) shows that, in terms of health deficits at age 65 and infectious diseases mortality at age 85, richer individuals are (slightly) less harmed than the benchmark individual by the lack of protection/treatment for infectious diseases. This does not mean, however, that

they suffer less in terms of life expectancy. Their life expectancy is about one and a half year higher than for the benchmark individual when $\epsilon = 0.85$, but 7 years lower when $\epsilon = 0$, according to case 5) and 6). That is, they lose about 8.5 years from the lack of protection/treatment, which is somewhat higher than the life expectancy loss in case 1).

Cases 7) and 8) show how poor initial health affects lifetime health outcomes. Because of the self-productive nature of health deficit accumulation, an initial endowment of 10 percent more health deficits at age 20 leads to 27 percent more health deficits at age 65 in the benchmark environment, see case 7). The weaker body is less successful at fighting infections, and the mortality rate from infectious diseases increases by 26 percent, in step with all-cause mortality. Individuals in bad (initial) health are particularly affected by missing protection/treatment for infectious diseases. As shown in case 8), life expectancy declines by another 7 years compared to the protection case and by more than 14 years compared to benchmark. Missing protection leads to a pronounced increase of mortality not only from infectious diseases but from all causes due to the inflammaging effect. Health deficits at age 65 are $(61 - 27 =)$ 34 percent higher than in case of protection and 61 percent higher than in the benchmark case.

The impact of protection preferences and the timing of protection are investigated with cases 9) and 10). Recall that the benchmark individual displayed no anti-protection attitudes, captured by setting $\omega = 0$, and thus demanded full protection from the start in case of effective protection/treatment ($p = 1$ for $\epsilon = 0.85$). In case 9) we consider an individual with mild protection aversion by setting $\omega = 0.3$, which causes the individual to avoid protection and treatment before age 40 and demand full protection and treatment afterwards. Because of the delayed expenditure on mitigating infectious diseases, the individual suffers more infections and thus faster aging in middle age. At age 65, health deficits are 10 percent higher than benchmark. The self-productive nature of health deficits and the feedback loop from chronic diseases to infections imply that the mortality rate from infections and all-cause mortality at age 85 increase by 10 and 13 percent, respectively, leading to a loss of almost 3 years of life. Case 10) illustrates that these effects are exacerbated if protection and treatment are avoided until age 60. Comparing case 10) and 1) (which one might think of as protection avoidance at any time) shows that starting protection after age 60 causes 75 percent of the loss in life expectancy compared choosing $p = 0$ for the entire lifetime (loss of 6 compared to 8 years). The reason lies in the self-productive nature of health deficits and the associated behavioral response: health spending is 25 percent lower compared to the benchmark in case 10) whereas we have seen that in case 1), where $\epsilon = 0$, overall health spending even slightly increases. The result suggests that life expectancy will be significantly affected if vaccination against respiratory infectious diseases is only recommended for the elderly and not for all adults.

Finally, case 11) shows the potential gain from eradicating infectious diseases. Compared to the potential loss without protection in case 1), the gains are small, reflecting the fact that most of the gains in infectious disease control have already been made. Note, however, that this conclusion is based on considering a developed country in the 21st century (and before the outbreak of the Covid-19 disease). In the next section, we examine the model in terms of the historical epidemiological transition.

In Table A.2 and A.3 in the Appendix, we show results for a model under the assumptions $r = \rho = 0.05$ and $r = \rho = 0.03$, instead of $r = \rho = 0.07$. We recalibrate the model by slightly adjusting σ in order to still match lifetime health expenditure and life expectancy. Comparing case by case with Table 1 shows the robustness of results. The predicted relative changes deviate insignificantly from those in Table 1 and all qualitative conclusions remain valid.

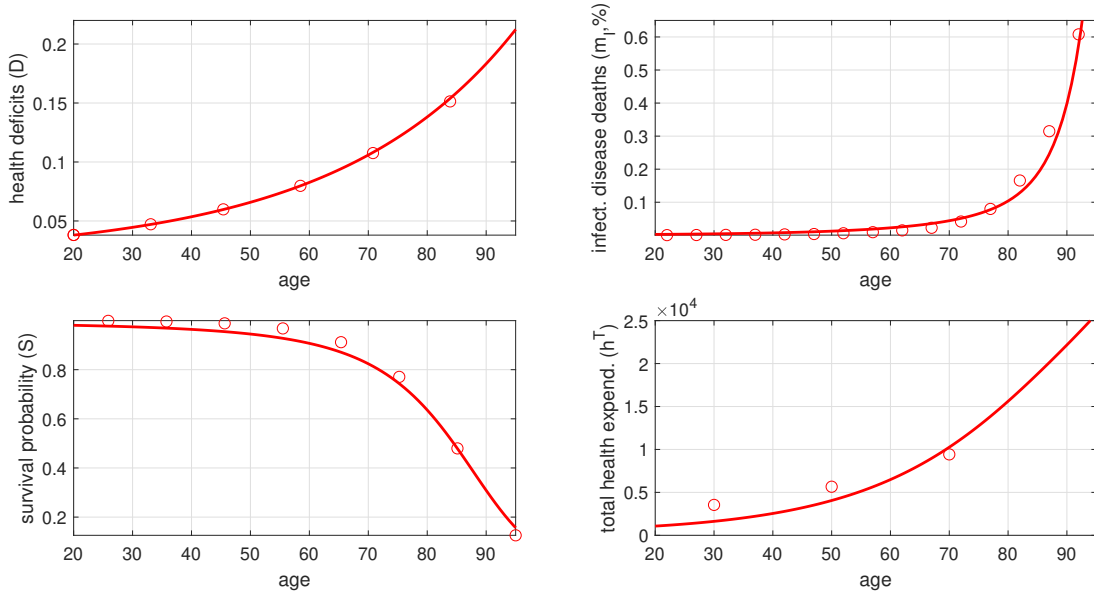
6.2. The Gender Gap In Mortality Redux. For given age, women display more chronic health deficits but lower mortality rates from all causes as well as from infections (see Schuenemann et al., 2017a, for a discussion of the morbidity-mortality paradox within the framework of the health deficit model). It is therefore an interesting question whether inflammaging and immunosenescence affect women’s health behavior and health outcomes differently. To answer this question, we recalibrate the model and set the force of aging μ to 0.031 and initial health deficits at age 20 to $D_0 = 0.038$, according to the estimates from Mitnitski et al. (2002). This means that women initially have more health deficits than men, but build up new deficits at a slower rate.

We set wage income w to 17,303, which is the average labor income for single women in the year 2010 (BLS, 2012). We retain the parameter values specifying medical technology from the benchmark calibration (assuming equal access and efficacy for women and men) and recalibrate the remaining parameters so that the model conforms to the following stylized facts: The path of health deficits matches the estimates of Mitnitski et al. (2002) for women; the implied survival curve $\mathcal{S}(t)$ matches the survival curve of females estimated from U.S. life tables (taken from Strulik and Vollmer, 2013); the path of infectious disease mortality matches the mortality rate from lower respiratory diseases for women (taken from GBD, Voss et al., 2020); health care expenditure matches average expenditure of female Americans in the year 2010 at the age of 35 and 70 (MEPS, 2010); life expectancy at 20 is 61.7 years, i.e. the expected age at death is 81.7 (NVSS, 2014);

Figure 2 shows the predicted life cycle trajectories and the targeted data points. The recalibrated parameters are $\sigma = 1.32$ (inverse of the IES), $a = 0.0165$ (residual in the health deficit accumulation process (1)), $\psi = 0.009$, and $\xi = 30.0$ (survival probability function $S_C(D)$ from chronic conditions). The remaining parameters are as specified for the benchmark model. Interestingly, the recalibration does not require a change of the infectious disease parameters β , ν , and B in order to fit female infectious disease mortality (upper right panel in Figure 2). The estimate of σ suggests that the utility function is significantly more curved for women than for men, which agrees with the observation that women have a higher degree of risk aversion (see Schuenemann et al., 2017a, for a detailed discussion of this feature).

Table 2 shows the results from the comparative dynamic analysis for women. The overall impression from a comparison with the results for men (Table 1) is a general agreement of the size of the effects. The most striking deviation is that women, who would like to protect themselves early on if the technology were available, would lose 2.5 more years of life than men if protection against disease was not available ($\epsilon = 0$), i.e. would lose more than 10 years. The effect on infectious disease mortality $m_I(85)$ is, however, about the same as for men. The results can be understood by the inflammaging mechanism. Because women have more health deficits

FIGURE 2. Life Cycle Health with Chronic and Infectious Diseases, Females



Lines: model prediction; dots: targeted data for females (from Mitnitski et al., 2002, for health deficits; Vos et al., 2020, for infectious disease mortality; Strulik and Vollmer, 2013, for survival probability; MEPS, 2010, for health expenditure); see text for details.

Table 2. Comparative Dynamics: Full Protection Case, Females

case	parameter change	remark	ΔLE	$\Delta m_I(85)$	$\Delta m(85)$	$\Delta D(65)$	$\Delta \tilde{h}$	$\Delta V(20)$	$\Delta V(65)$
1)	$\epsilon = 0$	no protection/treatment	-10.64	619.79	2.28	20.31	-0.47	-40.11	-48.01
2)	$B = 0$	no inflammaging	2.09	-0.59	0.68	-2.60	1.44	5.16	6.07
3)	$w = 1/2w_{bench}$	poorer individual	-0.77	2.01	2.49	2.37	-63.18	-135.18	-60.81
4)	$w = 1/2w_{bench}$ & $\epsilon = 0$	poorer individual and 1)	-11.23	633.87	4.76	23.28	-62.31	-147.84	-79.03
5)	$w = 2w_{bench}$	richer individual	0.89	-2.25	-2.78	-0.50	170.86	319.42	131.57
6)	$w = 2w_{bench}$ & $\epsilon = 0$	richer individual and 1)	-9.98	604.26	-0.44	19.78	156.96	201.18	15.25
7)	$D_0 = 1.1D_{0,bench}$	less healthy individual	-5.96	18.58	26.87	18.78	-6.65	-13.06	-28.12
8)	$D_0 = 1.1D_{0,bench}$ & $\epsilon = 0$	less healthy individual and 1)	-15.92	770.09	32.72	45.54	-10.24	-55.33	-70.76
9)	$\omega = 0.13$	protection starts age 40	-2.94	8.87	12.60	9.14	-7.37	-8.43	-18.10
10)	$\omega = 0.70$	protection starts age 60	-6.75	293.68	41.47	19.33	-25.76	-13.24	-43.32
11)	$\beta = 0$	eradication of infect. diseases	2.34	-100.00	0.16	-2.59	-0.84	8.37	8.33

The table shows the predicted deviation of health behavior and health outcomes from the calibrated (female) benchmark individual with $\omega = 0$. The index *bench* identifies the calibrated benchmark value; ΔLE is the change in life expectancy measured in years. The other entries are relative deviations from benchmark measured in percent. $\Delta m_I(85)$ ($\Delta m(85)$) is the relative deviation of the mortality rate from infectious diseases (from all causes) at age 85. $\Delta D(65)$ is the relative change in health deficits at age 65. $\Delta \tilde{h}$ reports the relative change in expected lifetime health expenditure. $\Delta V(20)$ is the relative deviation of lifetime utility, which equals the relative deviation of the value of life at 20. $\Delta V(65)$ is the relative deviation of the value of life at 65.

(as D_0 is higher) but age more slowly than men (as the force of aging parameter μ is lower), the increase in infections due to the lack of protection and treatment technology has a greater impact on the development of chronic diseases. For the same reason, women would also benefit more than men from the absence of the inflammaging mechanism ($B = 0$) and the eradication of infectious diseases ($\beta = 0$).

Table 3. Comparative Dynamics: High Protection Aversion, Males

case	parameter change	remark	ΔLE	$\Delta m_I(85)$	$\Delta m(85)$	$\Delta D(65)$	$\Delta \tilde{h}$	$\Delta V(20)$	$\Delta V(65)$
1)	$\epsilon = 0$	no protection/treatment	-0.58	-12.37	-15.93	-2.19	27.34	-3.69	7.34
2)	$B = 0$	no inflammaging	5.01	-15.16	-16.79	-11.97	28.62	1.79	32.93
3)	$w = 1/2w_{bench}$	poorer individual	-1.45	4.08	5.07	3.35	-60.45	-54.94	-56.32
4)	$w = 1/2w_{bench}$ & $\epsilon = 0$	poorer individual and 1)	-2.03	-8.31	-11.06	3.41	-49.16	-56.33	-56.20
5)	$w = 2w_{bench}$	richer individual	1.71	-4.89	-6.05	-5.66	151.78	120.30	145.83
6)	$w = 2w_{bench}$ & $\epsilon = 0$	richer individual and 1)	1.15	-16.95	-21.35	-5.63	217.37	111.27	138.73
7)	$D_0 = 1.1D_{0,bench}$	less healthy individual	-7.59	30.45	41.80	29.96	-8.56	-5.48	-46.39
8)	$D_0 = 1.1D_{0,bench}$ & $\epsilon = 0$	less healthy individual and 1)	-8.30	13.67	18.53	28.94	15.08	-9.84	-39.48
9)	$\omega = 0$	protection starts age 20	4.34	-87.29	-17.18	-10.38	25.81	1.39	30.64
11)	$\beta = 0$	eradication of infect. diseases	5.30	-100.00	-17.33	-11.90	23.98	2.73	36.28

The table shows the predicted deviation of health behavior and health outcomes from the calibrated (male) benchmark individual with $\omega = 1.25$ (protection starts at age 70). The index *bench* identifies the calibrated benchmark value; ΔLE is the change in life expectancy measured in years. The other entries are relative deviations from benchmark measured in percent. $\Delta m_I(85)$ ($\Delta m(85)$) is the relative deviation of the mortality rate from infectious diseases (from all causes) at age 85. $\Delta D(65)$ is the relative change in health deficits at age 65. $\Delta \tilde{h}$ reports the relative change in expected lifetime health expenditure. $\Delta V(20)$ is the relative deviation of lifetime utility, which equals the relative deviation of the value of life at 20. $\Delta V(65)$ is the relative deviation of the value of life at 65.

6.3. Protection Aversion. We next check the robustness of results to the assumption that the average American dislikes protecting and treating infectious diseases. To generate a scenario that differs substantially from the benchmark case, we set the protection aversion parameter $\omega = 1.25$ such that the reference American man begins with protection and treatment at age 70. In terms of vaccination behavior, a late onset of protection appears to be at odds with Covid vaccination rates, but is more relevant to influenza vaccination rates. According to the CDC, 68.7% of the 25- to 49-year-old age group and 95% of the 65-plus age group have completed the primary Covid vaccination series (CDC, 2023a) while only 37.1% of the 18- to 49-year-old age group and 73.9% of the 65-plus age group received an influenza vaccine (CDC, 2023b).

Targeting the same trajectories as for the benchmark calibration in Section 5, we recalibrate $\beta = 2.1$ (down from 4.2). A drastic adjustment of β is needed in order to fit the life cycle trajectory of infectious disease mortality. It means that a less virulent disease environment offsets the greater reluctance to take preventive and treatment measures. A couple of other parameters need mild adjustments: σ increases from 1.095 to 1.130 (to fit health expenditure) and a increases from 0.016 to 0.017 (to fit life expectancy). The increase of the residual parameter a means that the non-disease environment is a bit healthier than in the benchmark run. The other parameters are kept at benchmark level. Remarkably, the inflammaging parameter can also be preserved at benchmark level ($B = 0.5$). This claim is verified in Appendix C where we recalibrated the epidemiological transition for the case of protection aversion.

We next investigate how protection aversion affects the results of the comparative dynamic analysis. Table 3 shows the results for men. Naturally, the unavailability of protection and treatment technologies ($\epsilon = 0$) has a relatively small impact on the individual that anyways starts protecting only at age 70 (case 1). In contrast, this individual would greatly benefit from the (hypothetical) absence of the inflammaging channel ($B = 0$) and gain 5 more years of life (case 2). A similar conclusion holds for the eradication of infectious diseases ($\beta = 0$), which would provide more than 5 extra years of life (case 11). Case 10 shows that the individual

Table 4: Comparative Dynamics: High Protection Aversion, Females

case	parameter change	remark	ΔLE	$\Delta m_I(85)$	$\Delta m(85)$	$\Delta D(65)$	$\Delta \tilde{h}$	$\Delta V(20)$	$\Delta V(65)$
1)	$\epsilon = 0$	no protection/treatment	-2.03	-15.29	-20.42	0.09	19.15	-5.72	-3.92
2)	$B = 0$	no inflammaging	4.93	-17.56	-19.67	-9.44	26.58	3.39	23.51
3)	$w = 1/2w_{bench}$	poorer individual	-0.81	2.14	2.71	2.58	-63.96	-58.87	-61.15
4)	$w = 1/2w_{bench}$ & $\epsilon = 0$	poorer individual and 1)	-2.83	-13.38	-18.19	0.59	-56.61	-60.72	-58.77
5)	$w = 2w_{bench}$	richer individual	0.93	-2.44	-3.01	-2.56	173.04	138.97	151.56
6)	$w = 2w_{bench}$ & $\epsilon = 0$	richer individual and 1)	-1.12	-17.42	-22.89	-2.39	219.75	121.78	135.86
7)	$D_0 = 1.1D_{0,bench}$	less healthy individual	-6.24	22.45	32.17	18.07	-7.66	-6.49	-27.22
8)	$D_0 = 1.1D_{0,bench}$ & $\epsilon = 0$	less healthy individual and 1)	-8.34	2.79	3.99	19.55	8.04	-13.15	-32.24
9)	$\omega = 0$	protection starts age 20	4.45	-87.76	-20.94	-8.18	20.56	3.31	24.08
11)	$\beta = 0$	eradication of infect. diseases	5.75	-100.00	-20.91	-9.38	20.10	5.70	29.32

The table shows the predicted deviation of health behavior and health outcomes from the calibrated (female) benchmark individual with $\omega = 0.43$ (protection starts at age 70). The index *bench* identifies the calibrated benchmark value; ΔLE is the change in life expectancy measured in years. The other entries are relative deviations from benchmark measured in percent. $\Delta m_I(85)$ ($\Delta m(85)$) is the relative deviation of the mortality rate from infectious diseases (from all causes) at age 85. $\Delta D(65)$ is the relative change in health deficits at age 65. $\Delta \tilde{h}$ reports the relative change in expected lifetime health expenditure. $\Delta V(20)$ is the relative deviation of lifetime utility, which equals the relative deviation of the value of life at 20. $\Delta V(65)$ is the relative deviation of the value of life at 65.

unprotected into old age could gain 4.3 extra years if he took advantage of prevention and treatment throughout his life. This result is not the exact mirror image of case 10 from Table 1 due to the much milder disease environment. Results for variations in income (cases 3 and 6) and initial health deficits (case 7) are similar to the case of $\omega = 0$ (Table 1) while results are, again, different when $\epsilon = 0$ holds as well (cases 4, 7, and 8).

Table 4 shows the corresponding results for women. From the case for men we take $\beta = 2.1$ and calibrate ω such that women start using protection and treatment at age 70. This leads to the estimate of $\omega = 0.43$. The utility cost is lower than for men (where $\omega = 1.25$) because the level of income and thus the level of utility from consumption is lower for women, which means that protection behavior matters relatively more for women’s utility. Overall, the results for women are very similar to those of men. The most striking deviation is that women would still lose 2 years of life without protection technology (case 1), although they would start protection late if the technology were available. The higher life expectancy loss compared to men can be understood by the generally higher life expectancy of women.

As for men, the expected gain from the (hypothetical) elimination of the inflammaging channel (case 10) and disease eradication (case 11) is considerably higher than for $\omega = 0$ when the calibrated individual refuses to use the available protection and treatment technologies in young and middle age. In other words, the relatively small inflammaging effect on life expectancy and other key outcomes in the benchmark case (case 2 in Table 1 and 2 for men and women, respectively) results from the interaction of advanced technology of infectious disease prevention and the full protection behavior of the respective benchmark individual.

7. DRIVERS OF AGING, LONGEVITY, AND HEALTH EXPENDITURE DURING THE EPIDEMIOLOGICAL TRANSITION: 1860–2010

In this section we use the model to evaluate how infectious disease prevalence and efficacy of protection against infectious diseases as well as income growth, public health care, and medical

innovations in chronic disease treatment contributed to the evolution of health, longevity, and health expenditure during the epidemiological transition. The concept of the epidemiological transition has been developed by Omran (1971) who also specifically addressed the American transition in Omran (1977). Omran divides the historical epidemiological development in three stages: (i) the age of pestilence and famine, (ii) the age of receding pandemics, (iii) the age of degenerate and man-made disease. We are entering with the analysis at the dawn of the second phase, which, according to Omran, began in the United States in the mid-19th century, i.e. we consider the epidemiological transition from 1860 to 2010.

7.1. Calibrating the Epidemiological Transition. A stylized epidemiological transition is calibrated by feeding into the model the time series of β , ϵ , w , A , and ϕ_j , $j = h, p$. For the time series of wage income w , we use the stylized fact of a virtually trendless rate of real GDP per capita growth since 1860 (Bolt and Van Zanden, 2020). Wage income for average American men grew in lockstep with GDP per capita until about the 1970s and basically stagnated afterwards (Acemoglu and Autor, 2011). We thus impose a stylized path for wage income along which w grew at the average rate of GDP per capita from 1860 to 1980, namely at 1.54 percent p.a. (computed from Bolt and Van Zanden, 2020) and then stagnated at the level calibrated for the benchmark model.

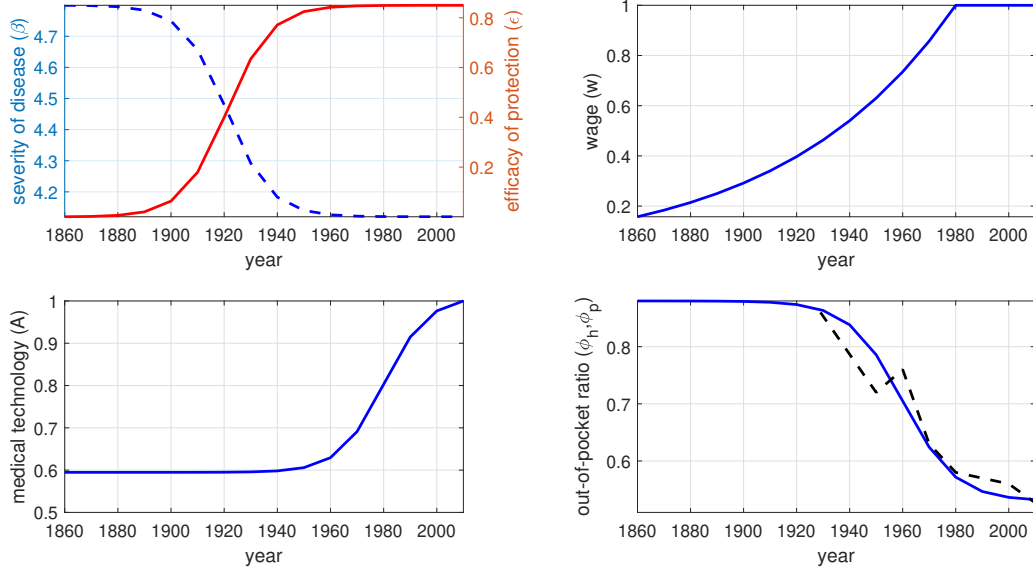
The remaining time trends are approximated by increasing or decreasing logistic functions. Getzen (2017) provides a scattered time series for the government share of health expenditure, rising from 14 percent in 1929 to 49 percent in 2015. We assume that before 1929 the government share was about constant at a level of 10 percent, approximate the time series by a logistic function, and set co-payment rates $\phi_h = \phi_p$ equal to one minus the government share.

The parameter A reflects the level of medical technology in prevention and treatment of chronic diseases. A consensus view in the literature is that there has been little technological progress before the 1960s and substantial progress afterwards, originating from the ‘cardiovascular revolution’ (Hansen and Strulik, 2017). Improved medical technology is one potential reason for increased demand for health insurance and treatments, and thus for a rising health expenditure share over time (Weisbrod, 1991; Frankovic and Kuhn, 2023). We assume that A grew at a minimal rate until 1950, picked up momentum in the 1950s and 60s, and increased by one percent per year from 1970 onwards in order to generate (jointly with the other time series) a good fit for the health expenditure share.¹¹

We calibrate the time series of β and ϵ by fitting logistic functions to the observed time series for the crude mortality rate from infectious diseases 1900–2000, obtained from Armstrong et al. (1999). In contrast to the age-specific mortality rate m , which measures for a given time interval the number of (possibly cause-specific) deaths in a given age-group relative to the population at risk, the crude mortality rate is a population statistic that measures the (possibly cause-specific) number of deaths in a given time interval relative to the total population (all ages).

¹¹ Specifically, we assume that technology advanced according to the logistic function $A(t) = 6.58 \times 10^{-4} / [1 + \exp(0.12(1990 - t))]$. Alternatively, we experimented with a piecewise defined function assuming an annual growth rate of 0.15% before 1960 and 1.0% thereafter, which led to insignificant changes in the results. The reason for the invariance of results to mild modifications of the medical progress function is that people spent relatively little on health in the early period.

FIGURE 3. The Epidemiological Transition: Calibration



The figure shows the calibrated time paths used in the discussion of the epidemiological transition. See text for details. The black dashed line in the lower-right panel shows the evolution of one minus the public share of total health expenditure according to Getzen (2017). The time series of A shows medical technology relative to medical technology in the year 2000.

The model-population consists of the benchmark individual from Subsection 6.1 observed at different ages at the same time. The longitudinal predictions of the model are then interpreted as cross-sectional results whereby the survival probabilities are interpreted as population shares. This allows the computation of crude deaths rates from infectious disease in a given year for the population at age t_0 and older as

$$d_I(t_0) \equiv \frac{\int_{t_0}^T \mathcal{S}(t) m_I(t) dt}{\int_{t_0}^T \mathcal{S}(t) dt} \quad (9)$$

from the model output for the representative individual that faces the parameter values for that year (i.e. time-variant medical technology parameters β , ϵ and A).

Finally, as already indicated in Section 5, we calibrate the value of inflammaging parameter B such that the predicted time series for life expectancy at 20 provides the best fit of the data. The data for life expectancy is taken from Lee (2001) whereby we use the mean of the reported upper and lower bound as the calibration target. This provides the estimate of $B = 0.5$. The calibration strategy implies that the historical evolution of life expectancy is fully explained by the model. Because of the involved parameter uncertainty, we conduct sensitivity analysis to alternative values of B at the end of this section. In any case, it is evident from the advances in the literature on immunosenescence and inflammaging surveyed in Section 2, that $B > 0$ is highly plausible and that the epidemiological transition played a major role in longevity gains. We also gained confidence in the robustness of the estimated value of B because the recalibrations

of the model for females and protection-averse individuals did not require readjustment of the inflammation parameter.

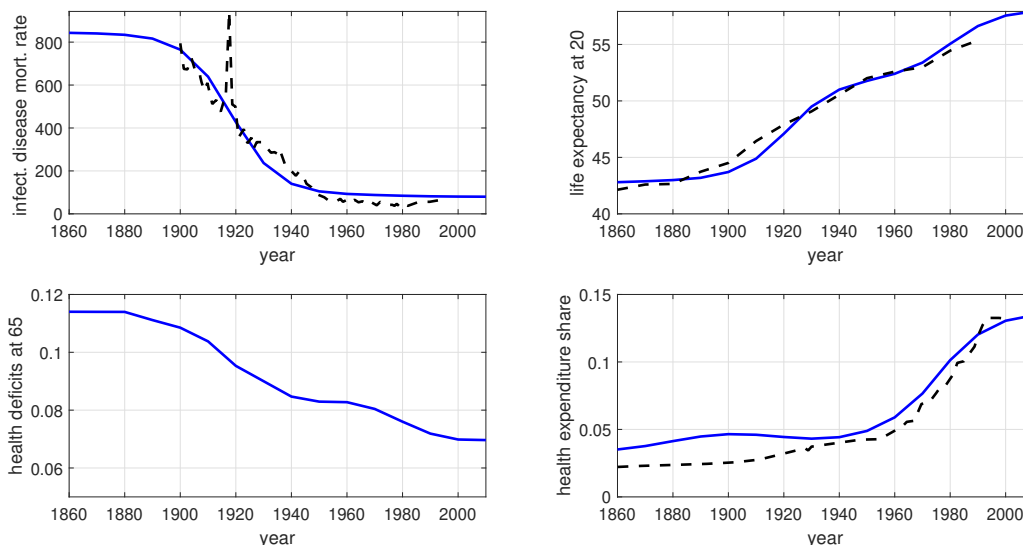
The calibrated time series of β , ϵ , A , w and $\phi_h = \phi_p$ are shown in Figure 3. The efficacy of prevention/treatment of infectious diseases ϵ increases from virtually nil to 0.85 while the infectious disease severity/prevalence parameter value β declines from 4.8 to 4.1, capturing the feature that some infectious diseases became extinct during the epidemiological transition. The ‘age of receding pandemics’ is thus characterized by declining prevalence of pathogens and improved methods of protection and treatment. The rapid decrease of infectious disease mortality during the epidemiological transition (see Figure 4) is explained by a rapid decline of disease prevalence β and a rapid improvement of treatment technology ϵ . The model is agnostic about the drivers of these processes. The literature lists public health projects (such as filtering and chlorinating water, sewerage, drainage, and street cleaning), the diffusion of medical knowledge (germ theory), better nutrition, vaccination, and new treatment technologies, such as the antibiotics developed in the 1930 and 1940s, as the main pathways (e.g. Omran, 1977; Cutler et al., 2006).¹²

7.2. The Epidemiological Transition. Figure 4 shows in the upper part the predicted time series for deaths from infectious diseases per 100,000 people and life expectancy at 20 (blue solid lines) along with the targeted data (black dashed lines). The model fails to predict the influenza pandemic from 1918-1919 but otherwise approximates the historical time series reasonably well. The lower right panel of Figure 4 shows the predicted health expenditure share of GDP. For that purpose, we assumed a capital share $\alpha = 0.4$ and approximated GDP per capita as $Y/L = w/(1 - \alpha)$. The dashed line shows the historical health share of GDP, as estimated in Getzen (2017).

As a measure of chronic health deficits, the lower left panel in Figure 4 shows the predicted frailty index at age 65, i.e. around the typical retirement age (similar trends could be drawn at other ages). The model predicts a large decline of health deficits and frailty long before the onset of medical advances in treatment of chronic diseases. On average, health deficits at 65 decline at a rate of 0.43 percent per later year of birth. Between 1900 and 1970, health deficits declined by 26 percent, a value which can be compared with estimates from Costa (2000). Costa found that the prevalence of chronic respiratory problems, valvular heart disease, arteriosclerosis, and joint and back problems in men aged 50–74 declined by 66 percent from the early 1900s to the 1970s. Costa estimates that 18 percent of the decline can be attributed to reduced exposure to infectious diseases (i.e. a decline of 12 percent). This important and unique study has the limitations that the 1900s-sample consists of U.S. Army veterans while the 1970s-sample consists of a representative U.S. population (which may cause an overestimation of the decline in health deficits) and that infections of veterans were only measured during their spell in the Army (which may cause an underestimation of the infections pathway). Costa attributes 29 percent of the

¹² The data series for infectious disease mortality and (public) health spending are only available for population averages. We therefore implicitly assume that average males are in this respect sufficiently similar to the population average. This seems a plausible assumption given the results from Subsection 6.2, which showed the similarity of the results for men and women.

FIGURE 4. Health Outcomes and Health Expenditure during the Epidemiological Transition 1860–2010



Blue solid lines: model prediction. Black dashed lines: data (see text for details). Infectious disease mortality is measured by the crude mortality rate (deaths per 100,000). Health deficits at age 65 are measured by the frailty index D . The health expenditure share is measured by total health expenditure per person divided by GDP per capita, which is inferred from wages, assuming a capital share of 0.4.

decline to occupational shifts. Assigning the unexplained part to infections would imply a 47 percent of the decline of chronic conditions explained by infections.¹³

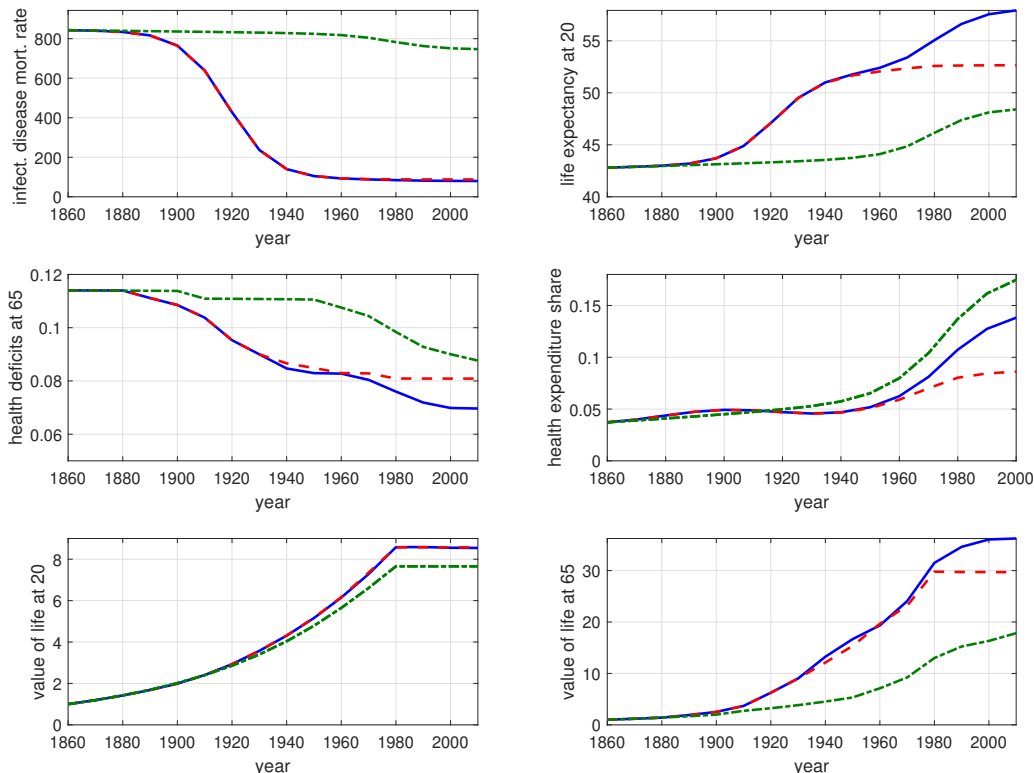
7.3. The Role of the Health Technology. The model is now ready to use for counterfactual historical experiments (in the spirit of Fogel, 1964). Specifically, we explore how health technology, broadly defined, contributed to health and longevity trends. In Figure 5, blue lines in the upper four panels repeat the benchmark solution from Figure 4. Additionally, we consider in the bottom panels of Figure 5 the impact of the epidemiological transition on the value of life experienced at age 20 and at age 65. The value of life is expressed in units of the value of life in 1860. The model predicts that, from 1860 to 2010, the value of life increases by about factor 7 for 20-year-olds and by factor 30 for 65-year-olds. The large difference arises because longevity gains are added at the end of life and are thus more heavily discounted by 20-year-olds than by 65 years-olds. The trend in value of life of 20-year-olds is predominantly driven by the trend of income and is thus basically flat when income stops growing after 1970. For the 65-year-olds, in contrast, the value of life is largely driven by trends in health and longevity and its almost linear growth is not much affected by the income trend.

Red dashed lines in Figure 5 show results when medical technology in the treatment of chronic diseases (captured by the parameter A) is kept constant at pre-1960s levels. In this case,

¹³ The empirical frailty index includes chronic conditions and functional limitations (Searle et al., 2008). In an accompanying article, Costa (2002) shows that the historical decline of chronic diseases was accompanied by a decline in functional limitations, on average at a rate of 0.6 percent per year.

predicted health outcomes deviate visibly from the benchmark case only after 1960. Without progress in treatment of chronic diseases, health deficits at age 65 would be about 15 percent larger in the year 2010, remaining life expectancy at age 20 would be about 4 years shorter, and the value of life at age 65 would be about 20 percent lower.

FIGURE 5. The Epidemiological Transition: Constant Disease Environment and Health Technology



Blue solid lines: benchmark run; red dashed lines: constant medical technology for treatment of chronic diseases (constant A). Green dash-dotted lines: constant medical technology for treatment of infectious diseases and constant disease environment (constant ϵ and constant β).

The main counterfactual experiment is the consideration of a constant disease environment, which is captured by keeping the efficacy of protection and treatment for infectious diseases ϵ and disease prevalence β at their 1860 levels. It seems plausible to assume that these two parameters work together, i.e. that there is a reduction in disease prevalence only if the effectiveness of disease control improves. The predicted outcome is shown by green dash-dotted lines in Figure 5. If the disease environment remained constant, there would have been little increase in life expectancy until the 1960s and only a moderate decline of chronic health conditions due to rising income and health investments. Between 1900 and 1970 health deficits at age 65 would have declined by 8 percent instead of 26 percent as in the benchmark scenario. After 1960, life expectancy would have increased by not more than about 4 years because of medical advances in the treatment of chronic diseases (increase in A) and the associated decrease in chronic diseases at the age of

65. Without the decline in infectious disease the value of life at age 65 in the year 2010 would be about half of its predicted value in the benchmark scenario.

The improvements of chronic health are associated with a slowdown of immunosenescence and cause a moderate decline in the prevalence of infectious diseases. Health expenditure exceeds benchmark expenditure after 1920 (when income has grown sufficiently), reflecting the fact that, without improvements in protection technology, it becomes more important to invest in slowing down health deficit accumulation to mitigate immunosenescence.

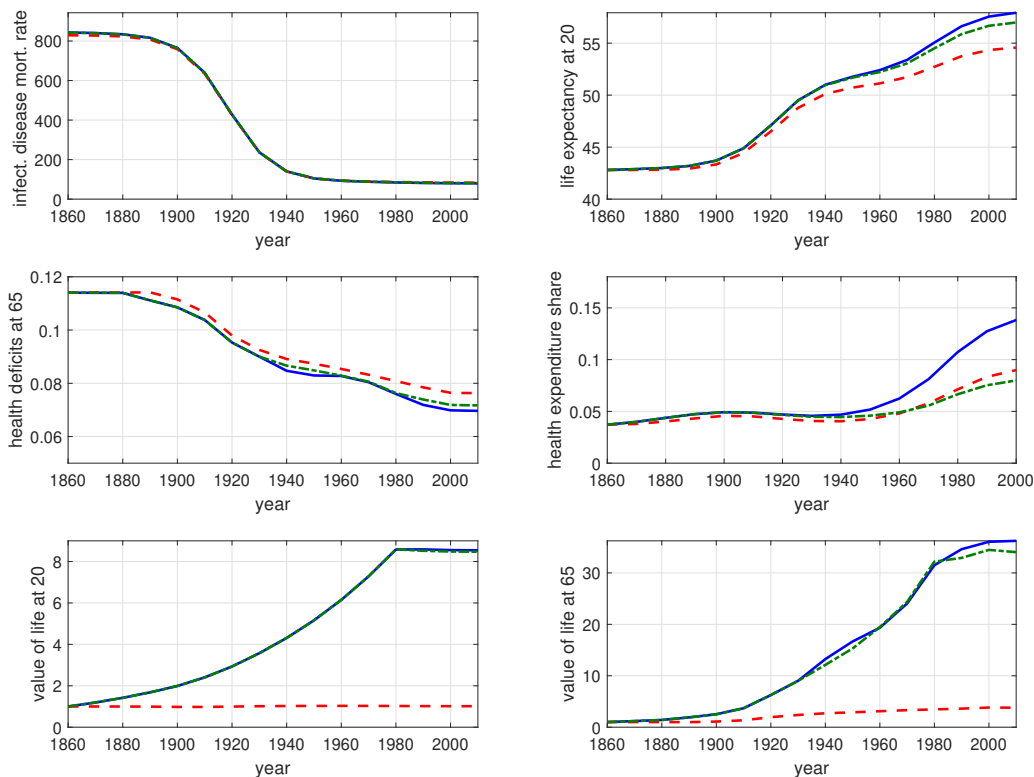
7.4. The Role of Income and Public Health Expenditure. Our next counterfactual experiments, shown in Figure 6, examine the development when wage income is kept at its 1860 level (red dashed lines) and when the public health expenditure share ($1 - \phi_h, 1 - \phi_p$) is kept at its 1860 level (green dash-dotted lines). In both cases, there is almost no change in the predicted path for infectious disease mortality compared to the benchmark run (upper left panel in Figure 6). For a correct assessment of these results, it should be remembered that public health expenditure is captured in the model exclusively in the form of subsidies for private health expenditure. Investment in public health infrastructure is implicitly included in the time series of medical technology (ϵ) and disease environment (β).

Without the increasing trends of income and public health expenditure, health deficits are larger and life expectancy is shorter, and the deviation from the benchmark run increases over time. Life expectancy in 2010 would be two years shorter without income growth and just under a year shorter without increases in public health spending. As shown in the middle right panel of Figure 6, these outcomes are explained by the reduced health expenditure in both scenarios. The greatest effect of absent income growth is on the value of life. As shown in the bottom panels of Figure 6, missing income growth has the greatest affect on the value of life, which stays almost constant when income remains at its 1860 level. Combined with the results from Figure 5, this means that the large gains in value of life at age 65 from improved medical technology were possible because income was also growing.

For the assessment of the predicted relatively modest impact of income on life expectancy it is helpful to recall that the calibrated average American is a relatively poor person whose income did not grow since the 1980s. In another counterfactual scenario (not shown) we assumed that the average American's income would continue to grow at 1.5 percent per year through 2010. This led to the prediction of a two-year gain in life expectancy compared to the benchmark run, implying that the relatively richer person gained in total 4 years from income growth. This result is another expression of the general finding that rich people benefit more from advances in medical technology because of their higher propensity to invest in health (see Dalgaard and Strulik, 2014 and Strulik, 2022, for a detailed discussion).

7.5. The Role of Inflammaging. In order to assess the importance of inflammaging, we next consider a scenario where the epidemiological transition takes place as calibrated but the inflammaging parameter B is set to zero. For that purpose, we recalibrate the 'residual parameter' a in health deficit accumulation process (1) to a slightly lower value ($a = 0.0155$) such that the model provides the same fit of the data as the benchmark model in the year 2000. The predicted

FIGURE 6. The Epidemiological Transition: Constant Income and Public Health Share

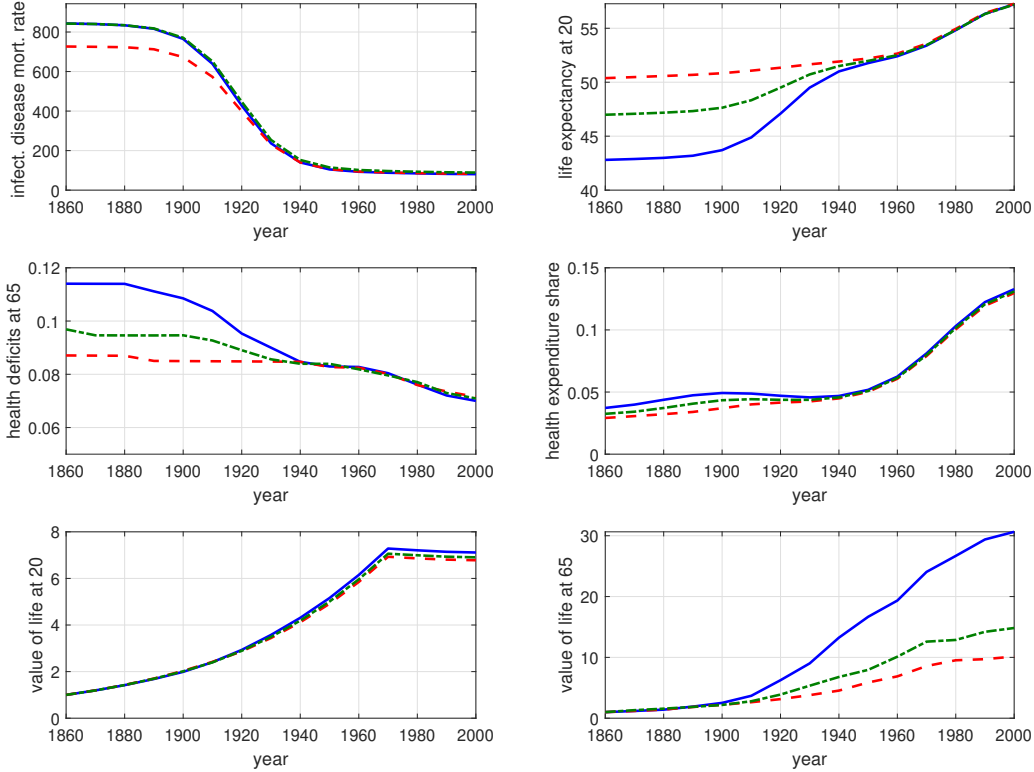


Blue solid lines: benchmark run; red dashed lines: constant wage income (constant w). Green dash-dotted lines: public health expenditure share (constant ϕ_h and constant ϕ_p).

health outcomes are shown by red dashed lines in Figure 7 and blue lines show again the benchmark outcomes. We see that, without inflammaging, health and longevity at the beginning of the epidemiological transition are grossly overestimated. Life expectancy at 20 would be about 51 years in 1860 (instead of 41 years) and would improve by only 3 years between 1860 and 1960 (instead of more than 10 years). Green lines in Figure 7 show an intermediate case for $B = 0.2$, leading to a moderate declining of chronic health deficits before 1960 and the prediction of a life expectancy of 47 in 1860.

If health and longevity trends are not fully explained by the model, they need to be explained by ‘something else’. To estimate this exogenous force, we finally adapt the technique developed in Oster (2019) to the case of computational experiments by asking how large an omitted factor needs to be in order to explain away the inflammaging effect. Specifically, we fit a time series of the residual term a in eq. (1) to get the long-run trend of life expectancy right. We find that residual term needs to increase by 48 percent from 1860 to 2010. While there are certainly omitted trends contributing to increasing life expectancy, not all of them would be consistent with the observation of a pronounced decline in chronic conditions. Declining deaths from violence, for example, would not be a candidate.

FIGURE 7. The Epidemiological Transition and Inflammaging



Blue solid lines: benchmark run ($B = 0.5$); red dashed lines: no inflammaging ($B = 0$); green dash-dotted lines: intermediate case ($B = 0.2$). Parameter a is recalibrated to match life expectancy at age 20 in the year 2000.

8. CONCLUDING REMARKS

This paper has presented a new economic theory of physiological aging and longevity that takes into account the interaction between infectious and chronic diseases jointly with health spending decisions. The theory introduced the gerontological mechanisms of immunosenescence and inflammation to the economics profession. These mechanisms are essential to understand why disease severity and mortality increase with age without ascribing causality to chronological age and why a life history of many infections leads to a more rapid development of new chronic health deficits.

Our model takes into account that increasing investments in health slow down the accumulation of chronic health deficits and make individuals better prepared for disease shocks. We also allow for investments to prevent and treat infectious diseases which not only reduce the probability of death from infection but also mitigate the feedback effect to chronic diseases due to inflammaging. We investigated the sources of the socioeconomic health gradient and employed the model in counterfactual analysis to assess the drivers of American epidemiological transition from 1860 to 2010.

That said, we shall stress that our goal was not to provide exact numbers on socioeconomic differences in health outcomes or gains in longevity and in the value of life from the epidemiological transition. The parameter uncertainty in our stylized model of the complex interaction between immunosenescence and inflammation is too great for this. Rather, our analysis generated several insights into mechanisms and the importance of this interaction for health outcomes from an economic perspective with potential policy relevance.

First, we find that susceptibility to infectious diseases is higher for individuals with lower income at any age and that the lack of access to protection and treatment reduces the life expectancy of the poor in particular. Second, our analysis suggests that a later onset of protection against infectious diseases through the inflammatory channel may have serious implications for health and life expectancy. This casts doubt on health policies that focus vaccination programs on the elderly. Third, our model supports a refined view of the epidemiological transition that replaces the traditional view of separate stages of infectious disease and chronic disease decline. Specifically, we showed that declining prevalence of infectious diseases and better access to protection against infections contributed significantly to the decline in chronic disease and to longer life spans before the onset of medical advances in chronic disease treatment in the 1960s.

This study is the first attempt to integrate the interaction between infections and chronic diseases into the economics of aging, and as such many interesting aspects have not yet been explored. The most obvious potential extension of the model is the inclusion of a childhood period. Dalgaard et al. (2021) have developed a theory of aging from conception to death that conceptualizes childhood as a period of accumulation of organ reserves and decreasing frailty. In this framework, infections would be particularly severe in old age and in early childhood as it is observed for many infectious diseases (Glynn and Moss, 2020). Another potentially fruitful way to proceed is the integration of unhealthy behavior such as smoking or overeating in a model of aging that considers both chronic health deficits (as Strulik, 2018, 2019a,b) and infections. Obesity, for example, is known to be one of the strongest correlates of inflammatory aging (Bektas et al., 2018) and it would be interesting to examine this aging force jointly with infections. Finally, the model may be tailored and applied to investigate life expectancy losses from pandemics like Covid-19.

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APPENDIX

Appendix A. Derivation of (7) and (8). The costate equations associated with the utility maximization problem are:

$$\lambda_k(r + m) = \lambda_k \rho - \dot{\lambda}_k \quad (\text{A.1})$$

$$[-\nu(1 - \epsilon p)\beta D^{\nu-1} S_C + S_I S'_C] [u(c) - \omega p] + \lambda_D \mu + \lambda_D \mu B \nu (1 - \epsilon p) D^{\nu-1} = \lambda_D \rho - \dot{\lambda}_D \quad (\text{A.2})$$

From differentiation of (3):

$$\frac{\dot{S}}{S} - \sigma \frac{\dot{c}}{c} = \frac{\dot{\lambda}_k}{\lambda_k} \quad (\text{A.3})$$

Inserting (A.3) into (A.1) and using the definition of the mortality rate $m = -\dot{S}/S$ provides (7) in the text. Inserting (3) and (4) into (A.2) provides

$$\frac{\dot{\lambda}_D}{\lambda_D} = \rho - \mu - \mu \nu B (1 - \epsilon p) \beta D^{\nu-1} - \frac{\mu \gamma A h^{\gamma-1}}{\phi_h \pi_h c^{-\sigma}} [-\nu(1 - \epsilon p) \beta D^{\nu-1} S_C + S_I S'_C] [u(c) - \omega p] \quad (\text{A.4})$$

From differentiation of (4):

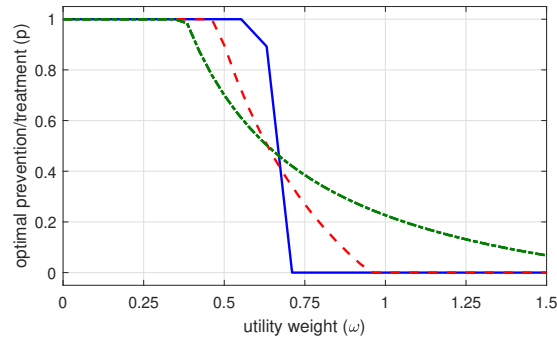
$$\frac{\dot{h}}{h} = \frac{1}{1 - \gamma} \left(\frac{\dot{\lambda}_D}{\lambda_D} - \frac{\dot{\lambda}_k}{\lambda_k} \right) \quad (\text{A.5})$$

Inserting (A.1) and (A.4) into (A.5) provides (8) in the text.

Appendix B. Quadratic Utility Cost Function. We now explore how optimal protection p deviates from the benchmark solution when the utility function is quadratic (rather than linear). For that purpose, we specified $f(p) = \zeta (p + \xi p^2)$ with $\zeta = 1/(1 + \xi)$. The normalization of ζ ensures that $f(p) \in (0, 1)$ for $p \in (0, 1)$ such that the disutility function preserves the scale of the benchmark model. In both models the scale is determined by ω . We then solve the implicit function (6) for the benchmark parameters of our model, the life cycle means of c , h , D of the benchmark solution, and alternative values of ξ and obtain the optimal solution for alternative values for ω . Results are shown in Figure A.1. The blue line shows the results for $\xi = 0$, which are the results for the benchmark model with linear utility cost, implying that the optimal p is almost always at 0 or 1. Red dashed lines show the optimal p when $\xi = 1/2$, i.e. when the quadratic term gets half the weight of the linear term. Over a wide range of ω -values the optimal solution is also at a corner and coincides with solution of the linear specification. Finally, we consider $\xi = 2$, i.e. the quadratic term gets twice the weight of the linear term. Even then, the optimal solution remains at full protection for sufficiently low ω s.

Appendix C. The Epidemiological Transition with Protection Aversion. For the calibration of the simulated epidemiological transition 1860–2010, we kept all time series from the main text except the time series of β which falls from 5.4 to 2.1, i.e. the germ environment undergoes a stronger transformation than in the benchmark run. Remarkably, the value of the inflammaging parameter is robust to the recalibration ($B = 0.5$ as in the benchmark run). Figure A.2 shows the predicted paths for the epidemiological transition. The predicted time paths for disease mortality, life expectancy, and health deficits at 65 are visually almost indistinguishable from the benchmark case (Figure 3 in the paper). The non-targeted prediction of the health expenditure share agrees somewhat less well with the historical data series than in

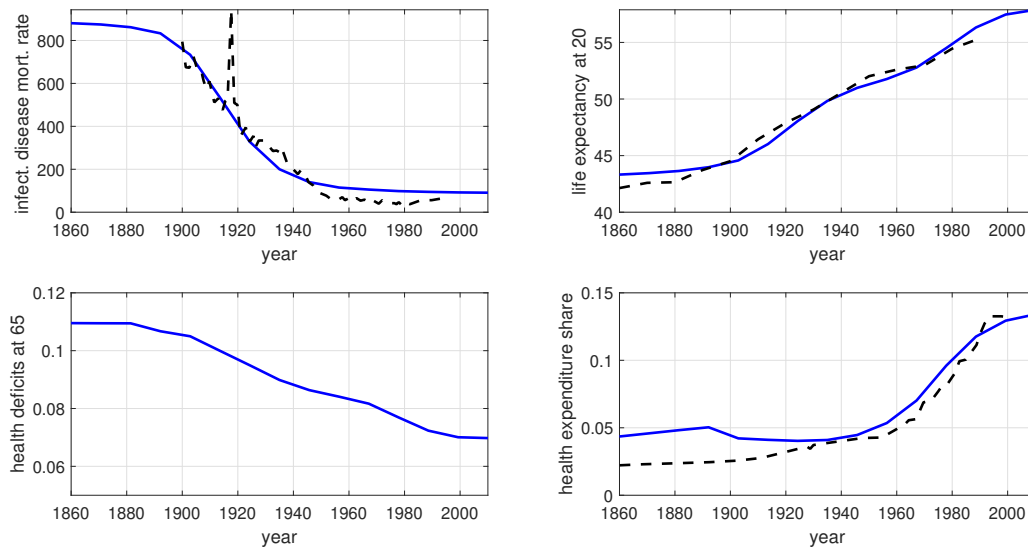
Figure A.1 Optimal Prevention/Treatment (p) for Alternative Utility Cost Functions



The figure shows the optimal solution for p given the benchmark parameterization of the model. Blue solid line: linear utility cost ωp . Red dashed line and green dash-dotted line quadratic utility cost $\omega \zeta (p + \xi p^2)$ and $\zeta = 1/(1 + \xi)$. Red dashed line: $\xi = 1/2$; green dash-dotted line: $\xi = 2$.

the benchmark run. In conclusion, the parameterization of the model is quite robust against variation in ω and the entailed protection behavior.

Figure A.2 Epidemiological Transition 1860–2010: High ω Case



Blue solid lines: model prediction. Black dashed lines: data (see text for details). Infectious disease mortality is measured by the crude mortality rate (deaths per 100,000). Health deficits at age 65 are measured by the frailty index D . The health expenditure share is measured by total health expenditure per person divided by GDP per capita, which is inferred from wages, assuming a capital share of 0.4.

Appendix D. Additional Results

Table A.1: Parameter Values

Parameter	Meaning	Men		Women	
		Pr. Age 20	Pr. Age 70	Pr. Age 20	Pr. Age 70
μ	natural rate of aging	0.043	0.043	0.031	0.031
A	medical technology (scale)	0.00155	0.00155	0.00155	0.00155
γ	medical technology (curvature)	0.13	0.13	0.13	0.13
B	inflammaging coefficient	0.5	0.5	0.5	0.5
ν	immunosenescence coefficient	2.0	2.0	2.0	2.0
β	infectious disease prevalence	4.2	2.1	4.2	2.1
ϵ	infectious disease technology	0.85	0.85	0.85	0.85
a	aging residual	0.0160	0.0170	0.0165	0.0185
ω	disutility from protection	0.0	1.25	0.0	0.43
σ	inverse of the IES	1.09	1.13	1.32	1.34
ρ	time preference rate	0.07	0.07	0.07	0.07
r	interest rate	0.07	0.07	0.07	0.07
w	wage income	27,928	27,928	17,303	17,303
ϕ_h, ϕ_p	coinsurance ratio	0.53	0.53	0.53	0.53
π_h	unit price health care (chronic)	1.89	1.89	1.89	1.89
π_p	unit price health care (infection)	189	189	189	189
ψ	survival function (chronic)	0.011	0.011	0.009	0.009
ξ	survival function (chronic)	39	39	30	30
D_0	initial health deficits	0.027	0.027	0.038	0.038

Pr. Age means protection age, i.e. the age at which the calibrated individual starts using disease prevention and treatment.

Table A.2: Robustness of Comparative Dynamics: $r = 0.05$

case	par. change	remark	ΔLE	$\Delta m_I(85)$	$\Delta m(85)$	$\Delta D(65)$	$\Delta \bar{h}$	$\Delta V(20)$	$\Delta V(65)$
1)	$\epsilon = 0$	no protection/treatment	-8.25	616.23	3.92	23.74	2.24	-10.95	-49.48
2)	$B = 0$	no inflammaging	1.64	-0.82	-0.05	-3.43	0.28	1.68	9.11
3)	$\Delta w = w/2$	a poorer individual	-1.33	4.10	5.16	5.55	-57.28	-52.74	-57.57
4)	$\Delta w = w/2$ & $\epsilon = 0$	a poorer individual and 1)	-9.30	644.12	8.98	30.89	-56.60	-57.22	-79.41
5)	$\Delta w = 2w$	richer individual	1.58	-4.60	-5.71	-3.36	135.81	110.24	116.91
6)	$\Delta w = 2w$ & $\epsilon = 0$	richer individual and 1)	-7.01	585.08	-1.68	19.63	139.68	84.25	11.11
7)	$\Delta D_0 = 0.1D_0$	less healthy individual	-7.23	26.27	36.24	27.78	-9.76	-5.06	-39.39
8)	$\Delta D_0 = 0.1D_0$ & $\epsilon = 0$	less healthy individual and 1)	-14.57	814.04	43.27	65.66	-9.30	-15.51	-82.33
9)	$\omega = 0.80$	protection starts age 40	-2.96	9.85	13.29	10.10	-11.34	-3.68	-21.72
10)	$\omega = 2.90$	protection starts age 60	-6.18	21.52	29.35	23.62	-29.92	-2.41	-53.50
11)	$\beta = 0$	eradication of infect. diseases	1.74	-100.00	-0.31	-3.42	-2.14	2.46	10.46

The table shows the predicted deviation of health behavior and health outcomes as in Table 1 for the recalibrated model with $r = \rho = 0.05$, $\sigma = 1.02$. Other parameters as for Table 1.

Table A.3: Robustness of Comparative Dynamics: $r = 0.03$

case	par. change	remark	ΔLE	$\Delta m_I(85)$	$\Delta m(85)$	$\Delta D(65)$	$\Delta \bar{h}$	$\Delta V(20)$	$\Delta V(65)$
1)	$\epsilon = 0$	no protection/treatment	-8.36	620.00	4.66	27.09	0.32	-10.91	-54.89
2)	$B = 0$	no inflammaging	1.67	-0.95	-0.24	-3.50	0.73	1.74	9.84
3)	$\Delta w = w/2$	a poorer individual	-1.34	4.18	5.29	5.63	-55.77	-50.89	-55.83
4)	$\Delta w = w/2$ & $\epsilon = 0$	a poorer individual and 1)	-9.40	648.04	9.79	31.26	-56.13	-55.82	-79.33
5)	$\Delta w = 2w$	richer individual	1.60	-4.70	-5.89	-3.45	128.20	102.61	111.20
6)	$\Delta w = 2w$ & $\epsilon = 0$	richer individual and 1)	-7.14	588.79	-0.99	19.92	127.80	78.05	5.18
7)	$\Delta D_0 = 0.1D_0$	less healthy individual	-7.33	26.79	37.01	28.05	-12.77	-4.49	-40.04
8)	$\Delta D_0 = 0.1D_0$ & $\epsilon = 0$	less healthy individual and 1)	-14.70	820.56	44.68	66.21	-13.52	-14.62	-82.83
9)	$\omega = 3.00$	protection starts age 40	-3.20	10.75	14.48	12.82	-19.29	-5.62	-32.13
10)	$\omega = 6.90$	protection starts age 60	-6.43	22.61	30.88	24.02	-37.54	-1.08	-58.19
11)	$\beta = 0$	eradication of infect. diseases	1.77	-100.00	-0.50	-3.49	-1.67	2.66	11.31

The table shows the predicted deviation of health behavior and health outcomes as in Table 1 for the recalibrated model with $r = \rho = 0.03$, $\sigma = 0.90$. Other parameters as for Table 1.