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Life Cycle Economics with Infectious and Chronic Diseases

Abstract

In this paper, we develop a life cycle model in which health and longevity are threatened by infectious and chronic diseases. The model captures that the susceptibility and severity of infectious diseases depend on the accumulated health deficits (immunosenescence) and that the life history of infections affects the accumulation of chronic health deficits (inammaging). 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. Finally, we use the model to investigate behaviour and long-term health outcomes in response to the Covid-19 pandemic. We predict that the pandemic will shorten the life expectancy of middle-aged people almost as much as that of older people because of inammaging and the self-productivity of health deficits.

JEL-Codes: D150, I100, I120, J240, J260.

Keywords: health behaviour, infections, health deficits, longevity, epidemiological transition, Covid-19, immonuosenescence, inflammaging.

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1. Introduction

In this paper we propose a new life cycle theory that captures the biological mechanisms of immunosenescence, i.e. the gradual decline in functionality of the immune system, and inflammaging, i.e. the progressive development of chronic pro-inflammatory states. Our goal is to investigate how these mechanisms, in interaction with the infectious disease environment and the state of medical technology, influence life cycle behavior, health, and longevity.

Our theory integrates into economics recent advances in medical science that emphasize the relationship between physiological aging and immunosenescence (Aiello et al., 2019, and Santoro et al., 2021). Physiological aging is conceptualized as the intrinsic, cumulative, progressive, and deleterious loss of function (Arking, 2006), which is reflected in the increasing susceptibility to infectious diseases and the accumulation of chronic health deficits. We assume that individuals invest in their health to protect themselves from infectious diseases and to slow down the process of developing chronic health deficits. Importantly, we take into account the relatively recent finding that one's immunological biography, i.e. the life history of infections, affects the emergence of chronic health deficits (e.g. Goronzy and Weyand, 2013; Franceschi et al., 2018).

Based on the two-way interaction between chronic health conditions and potentially life-threatening infections, we explore the relationship between (access to) medical technology, the disease environment, health expenditure, longevity, and the value of life in a calibrated life cycle model. 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 the disease environment and medical technological progress targeted to infectious and chronic diseases? 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 (access to) medical technology for treatment of chronic diseases and measures of disease prevention on infectious mortality risk, all-cause mortality, life expectancy, and the value of life.

(3) How does the loss in life expectancy and the value of life caused by a pandemic depend on (i) the individual age at the onset of the pandemic, (ii) the individual aversion to protective care, and (iii) the time taken to reach an endemic state of the disease? To answer these questions, we calibrate the model to the arrival of Covid-19 as a new infectious disease that is gradually becoming endemic.

We address these research questions by integrating infectious diseases into a life cycle model of health deficit accumulation (based on Dalgaard and Strulik, 2014) and by capturing the interaction between infections and chronic diseases related to immunosenescence and inflammaging. The health deficit model implies that 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. Consequently, the health of unhealthy people (with many health deficits) erodes faster than that of healthy people.[1] Health deficits are conveniently measured using the frailty index, which 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 frailty index has been used in hundreds of studies in medicine and, more recently, in economics (e.g. Abeliansky and Strulik, 2018a,b; 2020; Abeliansky et al., 2020, Hosseini et al, 2022).

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). It is consistent with the fact that more frail individuals (with many pre-existing conditions) are likely to experience a more severe and potentially fatal course of infections (e.g. Geriatric Medicine Research Collaborative et al., 2021).

Our main results are summarized as follows. First, individuals with lower income or more health deficits as young adults suffer from significantly higher mortality risk from infectious diseases and face higher all-cause mortality risk at any age. Consequently, they have 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). Moreover, initiating protection against infectious diseases at an older age (e.g., at age 60 rather than as a young adult) still 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 value of life at age 65 can be explained by the epidemiological transition.

Third, the model explains the observed steep increase of Covid-19 mortality with age, but avoids attributing causality to chronological age. We argue that the age gradient of Covid-19 mortality is nothing special, since a similar gradient is predicted for all-cause mortality from (mainly) chronic diseases, in line with Gompertz's law (Gompertz, 1825; Goldstein and Lee, 2020). Moreover, the feedback effect from infections to chronic diseases we capture in our model is consistent with the "Long-Covid" phenomenon, i.e. the presence of infection-induced damage of tissue and organs (such as the respiratory system, the cardiovascular system, the kidneys,

[1] 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.

and the gut), which results in health deficits that persist long after the infection has ended (e.g. Crook et al., 2021). More generally, this means that the future health burden of the disease will not be fully apparent until survivors of the pandemic have developed more chronic diseases and higher all-cause mortality. In fact, our analysis suggest that the Covid-19 pandemic will shorten the life expectancy of middle-aged people almost as much as that of older people, as a result of the self-productivity of health deficits in conjunction with infectious diseases.

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 life cycle 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 characteristics such as income, frailty, and anti-protection attitudes. In Section 7, the model is calibrated for a stylized epidemiological transition 1860-2000 and then used for a series of counterfactual experiments. These experiments (or as-if scenarios) make it possible to assess the role of progress in the prevention and treatment of infectious and chronic diseases during the epidemiological transition and to derive policy implications. In Section 8, the model is applied to the Covid-19 pandemic. Section 9 concludes the paper.

2. Immunosenescence 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,[2] including reduced efficacy of vaccination in elderly persons (Goronzy and Weyand, 2013; Nakaya et al., 2015; McElhaney et al., 2020). Most individuals develop a persistent pro-inflammatory state (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

[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.

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).

More generally, recent studies in medical science have emphasized the role of the immunological biography, i.e. the exposure to bacteria, viruses, fungi, and parasites over the lifetime, for immunosenescence and inflammaging (e.g. Goronzy and Weyand, 2013; Franceschi et al., 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, SARS-CoV-2, and herpes to chronic pain and inflammatory bowel disease (ulcerative colitis and Crohn's disease).[4] Another example is the post-covid syndrome (or "Long-Covid") where Covid-19 infections (typically more severe ones) lead to 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

[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) and SARS-CoV-2 exposure (Huang et al., 2020) for functional T-cell changes.

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 that (i) the presence of more chronic diseases increase susceptibility, severity, and mortality from infectious diseases and (ii) the feedback effect of infections on the developing chronic deficits. In contrast to Cropper, we explicitly model and distinguish mortality risk from infections and chronic conditions. For example, Cropper states that his model only applies to 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).[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), and the life expectancy gaps across gender and marriage status (Schünemann et al., 2017a, 2020). 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 shown in Finch and Crimmins (2004) and Finch (2010). 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

[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.

[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.

the decrease was due to declining infections (Costa 2000, 2002). We take these observations into account when calibrating the life cycle 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.

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 life cycle 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 on the interaction between infections and chronic diseases. Second, we assess the implications of this feedback loop on the socioeconomic health gradient, the course of the historical epidemiological transition, and the long-term health consequences of the Covid-19 pandemic. 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.[7] Moreover, rather than studying the impact of the epidemiological transition on economic outcomes, we examine the role of the disease environment and medical technological progress on health outcomes in interaction with endogenous health expenditure. Finally, our analysis helps predicting the loss in both life expectancy and the value of life caused by a pandemic depending on individual characteristics and the transition process to an endemic state of the disease.

4. The Model

4.1. **Infectious and Chronic Diseases.** Consider an individual exposed to random health shocks and suppose that health shocks lead to perceptible illness only if sufficiently severe, with the threshold shock level depending on individual health status. Specifically, suppose the probability of infection shocks of size s is Pareto-distributed and given by $f(s) = \nu\tilde{\beta}^{\nu}s^{-(\nu+1)}$, with $s \in \{1, \infty\}$ and cumulative distribution function $F(s) = 1 - (\tilde{\beta}/s)^{\nu}$, $\nu > 0$. Only shocks of sufficient strength greater than $\bar{s}$ result in an infection, which 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

[7] See Wachtler et al. (2020) for a review of evidence in the context of Covid-19.

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 high value of β means that individuals at all states of health $D > 0$ (i.e., except those without pre-conditions) are more likely to be severely infected. A high value of β thus characterizes an epidemic disease environment while a low value characterizes low disease severity (endemic disease, off season for seasonal diseases).

Individuals can reduce the probability of infection by methods of self-protection and (if available) vaccination. Moreover, there may exist an effective treatment. For simplicity we measure the effort in disease avoidance and treatment in one choice variable, p. This assumption reflects the feature that when vaccinated, infections lead to less severe sickness and therefore less treatment is required to achieve a given risk of mortality 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 outcome since many conceivable interventions are actually bivariate (vaccinate or not). The efficacy of disease prevention and treatment is captured by parameter $\epsilon \in [0, 1)$. The probability of death from infection at any increment of time is thus given by $(1 - \epsilon p)\beta D^{\nu}$ which also serves as our measure of the severeness of illness from infection. Protection reduces sick time by avoiding infections and treatment reduces sick time by 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.

We distinguish direct and indirect effects of infections: the direct effect consists of the development of acute health conditions which reduce the survival probability from infection, $S_I(p, D) \equiv 1 - (1 - \epsilon p)\beta D^{\nu}$. 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 index individual age and denote $\mathcal{S}(t) \equiv S(p(t), D(t))$, $\mathcal{S}_I(t) \equiv S_I(p(t), D(t))$, and $\mathcal{S}_C(t) \equiv S_C(D(t))$. The conditional (i.e. age-specific) all-cause mortality rate at time 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 $m_I(t) \equiv -\dot{\mathcal{S}}_I(t)/\dot{\mathcal{S}}(t)$.

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 $U(c,p) = u(c) - \omega p$, with 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 elasticity of intertemporal substitution and $\omega \geq 0$ measures the degree to which disease prevention and treatment affect utility.[8]

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. 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, $1 - S_I$. Summarizing, chronic health deficits (measured by the frailty index D) develop according to the law of motion

$$\dot{D} = \mu \left[D - Ah^{\gamma} + B(1 - \epsilon p)\beta D^{\nu} - a \right], \tag{1}$$

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 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

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

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 $k(0) = k_0 \geq 0$.

[8] In the calibrated model, potential utility is positive at any age, such that individuals indeed prefer to live longer.

Facing the law of motions (1) and (2), individuals maximize expected lifetime utility $V = \int_0^T S(p(t), D(t)) \left[u(c(t)) - \omega p(t)\right] \mathrm{e}^{-\rho t} \mathrm{d}t$, in which ρ is the time preference rate and T is the (endogenous) planning horizon. As individuals die for sure when state variable D reaches its upper limit, $\bar{D}$, condition $D(T) = \bar{D}$ holds. Moreover, we assume a terminal condition for wealth $k(T) = \bar{k} \geq 0$. Recalling $S(p, D) = S_I(p, D)S_C(D)$, the associated current-value Hamiltonian is given by

$$\begin{aligned}\mathcal{H} = {} & S_I(p, D)S_C(D) \left[u(c) - \omega p\right] + \lambda_k \left[(r + m)k + w - c - \phi_p \pi_p p - \phi_h \pi_h h\right] \\ & + \lambda_D \mu \left[D - Ah^\gamma + B(1 - \epsilon p)\beta D^\nu - a\right],\end{aligned}$$

in which λ_k and λ_D are the shadow prices (co-state variables) of wealth and health deficits. As time horizon T is not fixed (but endogenous to health expenditure), the transversality condition $\mathcal{H}(T) = 0$ has to hold. Using $S_I(p, D) \equiv 1 - (1 - \epsilon p)\beta D^\nu$, the first order conditions for an interior solution of controls c, h, and p are:

$$S(p, D)u'(c) = \lambda_k \tag{3}$$

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

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

The left-hand sides of the first order conditions show the marginal benefits and the right-hand sides the marginal costs. Eq. (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. Eq. (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, $\mu\gamma A h^{\gamma-1}$, 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 $S(p, D) = \left[1 - (1 - \epsilon p)\beta D^\nu\right] S_C(D)$) to eliminate the shadow prices in (5) and to obtain the interior solution for p in closed-form:

$$\tilde{p} = \frac{u(c) - \frac{1 - \beta D^\nu}{\epsilon \beta D^\nu} x}{\omega + x}, \quad x \equiv \omega + u'(c) \left[\phi_p \pi_p - \frac{\phi_h \pi_h}{\gamma A h^{\gamma - 1}} \epsilon \beta B D^\nu\right]. \tag{6}$$

Before we discuss the comparative statics, two qualifications are in order. First, (6) is a partial solution. It holds for given levels of consumption c and health investments h. Second, $\tilde{p}$ characterizes the interior solution. The full solution is given by $p = \min\left\{1, \ \max\left\{0, \ \tilde{p}\right\}\right\}$ and it will turn out that in many of the applied cases, individuals select a corner solution (no protection

or full protection). A discussion of $\tilde{p}$ 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 side-effects 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). 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 life cycle health 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. The model also predicts that, ceteris paribus, frail persons (with pre-existing conditions) exhibit a greater propensity of disease prevention. 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

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

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left[r+m-\mu-\mu\nu(1-\epsilon p)\beta B D^{\nu-1}\right] - \frac{1}{1-\gamma}\frac{\mu\gamma A h^{\gamma-1}}{\phi_h\pi_h S_I(p,D)S_C(D)c^{-\sigma}}\left[\nu(1-\epsilon p)\beta D^{\nu-1}S_C(D) - S_I(p,D)S_C'(D)\right]\left[\frac{c^{1-\sigma}-1}{1-\sigma}-\omega p\right]. \tag{8}$$

Equation (7) is the well-know Euler equation for optimal consumption. Equation (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, $S'_C(D)$.

Life cycle behavior and outcomes are obtained by solving equations (6)–(8), describing life cycle behavior, the law of motions (1) and (2), initial and terminal conditions for state variables k and D, and the transversality condition $\mathcal{H}(T) = 0$. 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. 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. Aside from the modeling of behavior and outcomes related to infectious diseases, the calibration follows closely the procedure in Dalgaard and Strulik (2014) and Schünemann et al. (2017a,b). Health deficits are measured by the frailty index. 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 $S_C(D) = (1 + \psi)/(1 + \psi \exp(\xi D))$. For the benchmark case, we assume $\omega = 0$, implying that the average person has no attitudes or

[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.

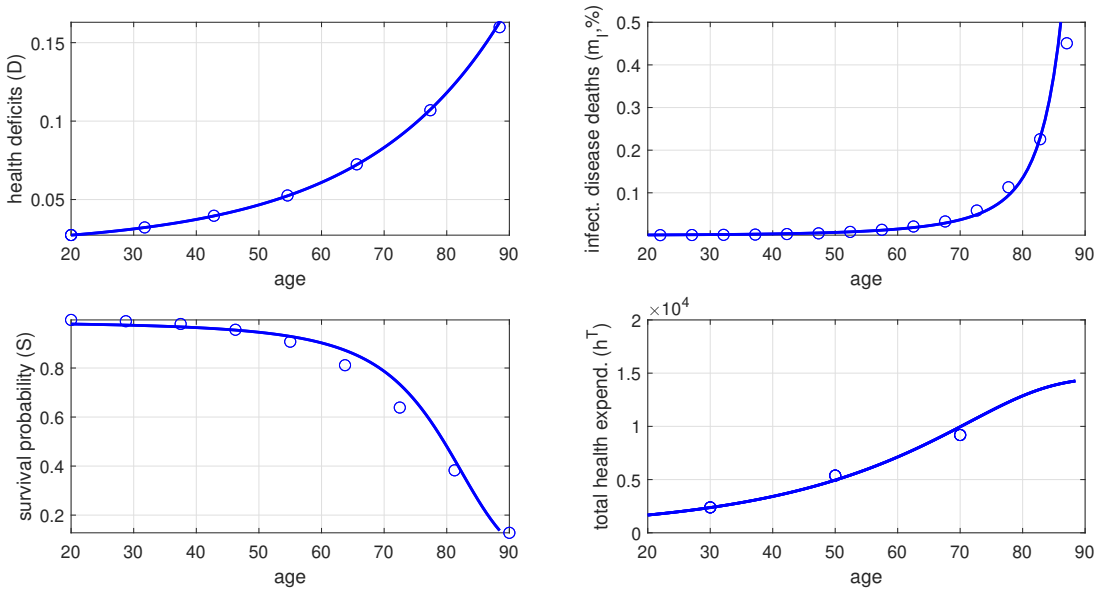
preferences against protecting or treating infectious diseases. A difficult issue is the calibration of the feedback effect of infections on chronic diseases (parameter B in eq. (1)). As discussed above, the recent literature provides ample evidence for the existence of a feedback effect, although to our knowledge no study has attempted to quantify this effect. We assume that B is time-invariant (a physiological constant) and that medical technology (as captured by parameters β, ϵ and A) systematically changes over time to explain jointly with B the historical paths of both life expectancy at age 20 and infectious disease mortality (as will be explained in detail in Section 7), feeding in the historical income path and the path of the public share of total health expenditure jointly with the remaining, time invariant parameters a, γ, σ, ν, ψ, and ξ. 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).

While most of the calibrated parameters are latent, the estimated value of σ agrees well with studies suggesting that the intertemporal elasticity of substitution 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 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, 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

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

FIGURE 1. Life Cycle Health with Chronic and Infectious Diseases



Lines: model prediction; 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.

disease mortality can well be explained by life-cycle behavior jointly with two features of our 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, the quasi-exponential health deficit accumulation process, including its feedback effect of infections on the development of chronic conditions.

6. Life Cycle Health with Endemic Diseases

This section presents the comparative dynamics of the model. In order to discuss life cycle 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 $\tilde{h} \equiv \int_0^T \mathcal{S}_C \mathcal{S}_I(\pi_h h + \pi_p p)\mathrm{d}t$. 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.

Table 1: Health and Longevity with Endemic Disease: Comparative Dynamics

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	-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 benchmark individual. 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.

Case 1) evaluates the gains from infectious disease protection and treatment from the perspective of the average American 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.

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, 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 25 percent, in step with all-cause mortality. Individuals in bad 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 percentage points 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 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 recommending vaccination against infectious diseases of the respiratory system (as caused by influenza or corona virus) for old-aged individuals only rather than for all adults can considerably affect life expectancy.

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

In Table A.1 and A.2 in the Appendix, we show results for a model recalibrated under the assumption $r = 0.05$ and $r = 0.03$. The recalibration adjusts also ρ and σ in order to match lifetime health expenditure and life expectancy. Comparing results 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.

7. Drivers of Aging, Longevity, and Health Expenditure during the Epidemiological Transition: 1860–2000

In this section we use the model to evaluate how infectious disease prevalence and efficacy of protection against infectious diseases, 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 2000.

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 1970, 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, 2022). We assume that A was basically constant before 1950 and increased by one percent per year from the late 1960s onwards in order to generate (jointly with the other time series) a good fit for the health expenditure share.

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 el 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). The model-population consists of the benchmark individual 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

$$d_I \equiv \frac{\int_{t_0}^{T} \mathcal{S}(t) m_I(t) \mathrm{d}t}{\int_{t_0}^{T} \mathcal{S}(t) \mathrm{d}t} \tag{9}$$

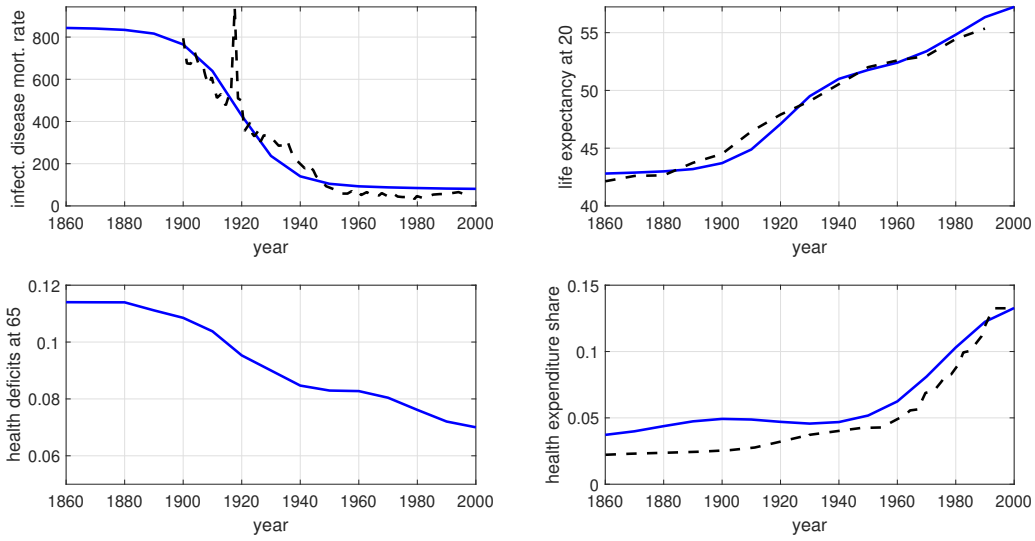
from the model output for the representative individual that faces the parameter values for that year (i.e. time-variant medical technology parameters β, ϵ and A). Finally, we calibrate the value of the interaction effect B such that the predicted time series for life expectancy at 20 provides the best 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.

The calibrated time series of β, ϵ, A, w and $\phi_h = \phi_p$ are shown in Figure A.1 in the Appendix. 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 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).

7.2. **Predictions.** Figure 2 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 2 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).

FIGURE 2. Life Cycle Health during the Epidemiological Transition 1860–2000



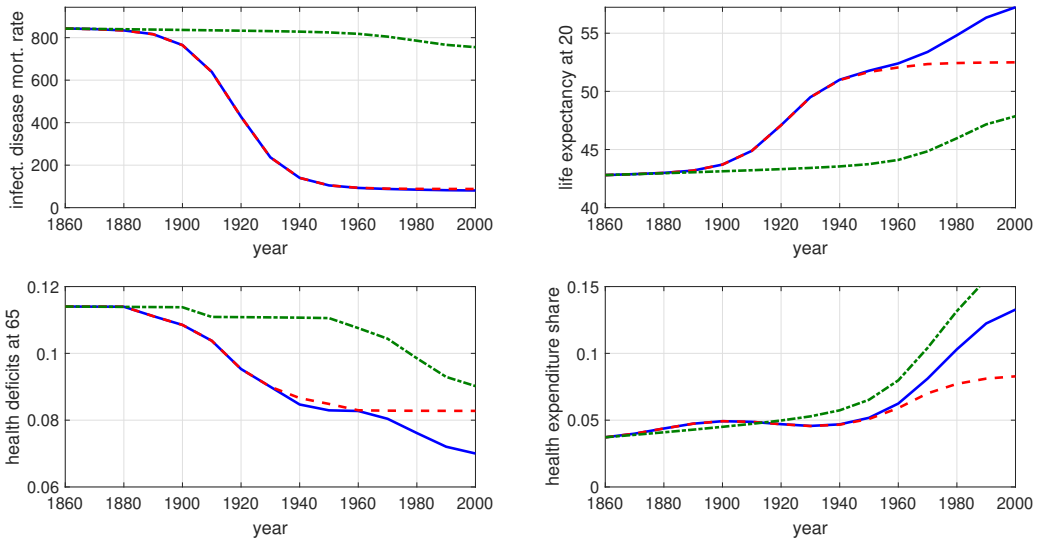
Blue solid lines: model prediction. Black dashed lines: data (see text for details). Infectious disease mortality is measured by the crude mortality rate (deaths per 100,000). Health deficits at age 65 are measured by the frailty index D. The health expenditure share is measured by total health expenditure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

As a measure of chronic health deficits, the lower left panel in Figure 2 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 decline to occupational shifts. Assigning the unexplained part to infections would imply a 47 percent of the decline of chronic conditions explained by infections.[11]

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 3, blue lines repeat the benchmark model and red dashed lines 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, health deficits at age 65 would be about 15 percent larger and remaining life expectancy at age 20 would be about 4 years shorter in the year 2000, as shown in the lower left and upper right panel of Figure 3, respectively.

FIGURE 3. The Epidemiological Transition: Constant Disease Environment and Health Technology



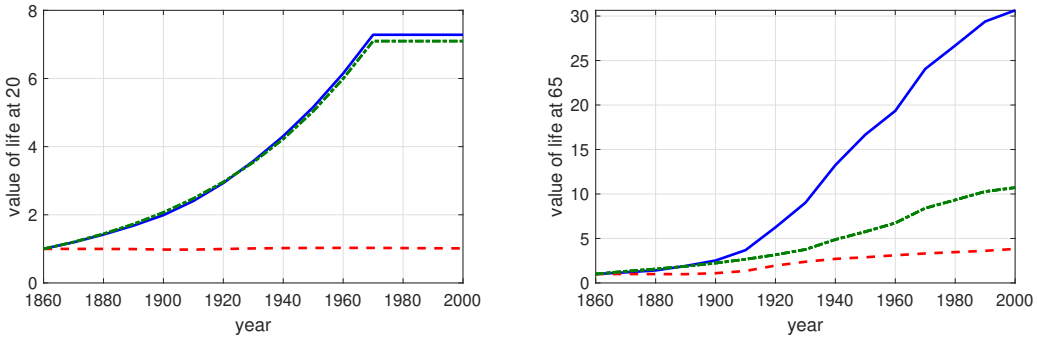
Blue solid lines: benchmark run; red dashed lines: constant medical technology for treatment of chronic diseases (constant A). Green dash-dotted lines: constant medical technology for treatment of infectious diseases and constant disease environment (constant ϵ and constant β).

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

[11]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.

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 3. If the disease environment remained constant, there would have been little increase in life expectancy until the 1960s and only a slight decline of chronic health conditions due to rising income and health investments. 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. Between 1900 and 1970 health deficits would have declined by 8 percent instead of 26 percent as in the benchmark scenario. The moderate health improvements are associated with the presence of a small (indirect) effect on the prevalence of severe infectious diseases only and the contribution of infectious diseases to health deficit accumulation via the captured inflammaging channel ($B > 0$). 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.

FIGURE 4. The Epidemiological Transition and the Value of Life at Age 20 and 65



The figure shows the value of life relative to the value of life in 1860. Left: at age 20; right: at age 65. Blue solid lines: benchmark run; red dashed lines: holding income constant; green dash-dotted lines: holding the disease environment constant (no epidemiological transition).

Finally, we consider the impact of the epidemiological transition on the value of life experienced at age 20 and at age 65. The calibrated benchmark case is shown by blue solid lines in Figure 4. The value of life is expressed in units of the value of life in 1860. The model predicts that, from 1860 to 2000, the value of life increases by about factor 7 for 20 years olds and by factor 30 for 65 years olds. The large difference arises because longevity gains are added at the end of life and are thus more heavily discounted by 20-years-olds than by 65 years-olds. This feature is also responsible for the result that 20 years-olds experience hardly any gain in value of life when income is held constant, as shown by the red dashed lines. The value of life at 65, in contrast, increases almost fivefold when income is held constant. Green dash-dotted lines show the counterfactual value of life when there is no epidemiological transition (holding β and ϵ constant). While there is hardly any effect on the value of life at 20 (again reflecting discounting), the value of life at 65 deteriorates heavily. The total increase of the value of life

declines from factor 30 to factor 10, leading to the conclusion that two thirds of the gain in value of life at age 65 are explained by the epidemiological transition.

8. Life Cycle Health with Pandemic Disease

8.1. **Calibrating the Covid-19 Pandemic.** In contrast to endemic diseases, pandemics are characterized by a high and unstable number of infected individuals and are therefore best conceptualized as shocks in the life cycle model. Eventually, when infections converge to a low trendless value, the disease becomes endemic (or disappears).

We begin the calibration of the pandemic by considering its end. Although the future of the pandemic is not known there seems to be widespread agreement among scientists that the disease will not disappear (Phillips, 2021). Instead, it converges to an endemic state in which it continues to contribute to deaths from infections. We assume that, at this state, protection against Covid-19 is as good as against the influenza. Formally, denoting by $\epsilon(x)$ the state of the protection technology x years since the onset of the pandemic, we assume $\epsilon(\infty) = 0.85$), which equals the previously calibrated value. Moreover, we assume that the long run Covid-19 death rate equals the death rate from influenza, i.e. 0.0018 percent (Vos et al., 2020).[12] The death rate from infections thus increases from 0.029 percent, as implied by the benchmark calibration in Section 3, to 0.031 percent at the new steady state. This leads to the estimate $\beta = 4.5$ (from 4.2 at benchmark).

At the beginning of the pandemic, the protective technology was substantially weaker. For the calibration of $\epsilon(0)$, we target a Covid-19 crude death rate of 0.115 percent, which is the average death rate in the years 2020–2021, as reported in CDC (2022). Taking into account the pre-existing infectious diseases, this means that when Covid-19 arrives, the death rate from infections rises to 0.144 percent. It implies the estimate $\epsilon(0) = 0.37$.

The resulting health outcomes for $\epsilon(0) = 0.37$ and $\beta = 4.5$ compared to the benchmark are shown in Figure A.2 in the Appendix. That is, before considering transitional dynamics, we first expose the benchmark individual at all ages to the disease at pandemic level and assume that the pandemic is permanent (an approach that follows Goldstein and Lee, 2020). A remarkable feature is that the log of the mortality rate m_I is linearly increasing in age, implying a constant growth rate of the force of mortality. Since ν has not been re-calibrated, the Covid-19 death rates are basically scaled versions of deaths from pneumonia and influenza. The model predicts that the Covid-19 death rate increases with age at a rate of about 10.8 percent until about age 70, after which death rates accelerate. An almost constant growth of the death rate at 11 percent is in line with the estimates by Ferguson et al. (2020), which were used in Hall et al. (2020). Goldstein and Lee (2020) computed a similar constant growth rate for European countries but a slightly lower rate for the U.S. In line with the findings from Goldstein and Lee, the model predicts that the growth rate of Covid-19 deaths slightly exceeds the predicted growth rate from all-causes mortality, which is obtained as 10 percent, in line with empirical estimates of Gompertz' law of mortality (see e.g. Strulik and Vollmer, 2013).

[12] Crude deaths rates from infectious diseases, d_I are, again, computed by (9), using the contemporaneous parameter values.

Figure A.2 also shows a significant increase of chronic diseases due to the feedback effect from inflammaging. For example, a frailty index of 0.14, which was previously reached at age 84, is reached already at age 79 after the onset of the pandemic. This means that deaths caused by Covid-19 are substantially larger than the measured deaths that are directly attributed to Covid-19 infections. The all-cause crude death rate (per year) is predicted to increase by 0.105 percent. This value is in line with the two-year Covid-19 excess death rate in the years 2020 and 2021, estimated by Wang et al. (2022) as 0.18 percent.[13]

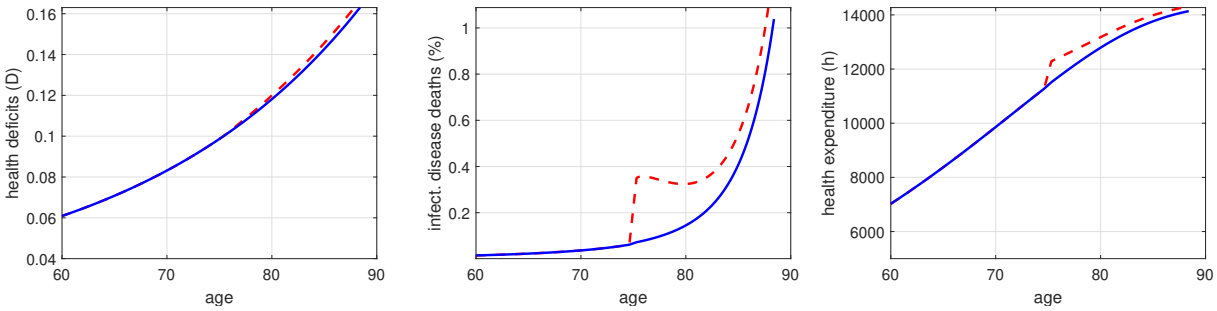
8.2. **Life Cycle Dynamics with Transition to an Endemic State.** We next use the life cycle model to assess the influence of age, frailty (pre-existing conditions), and inflammaging on changes in morbidity and mortality caused by the pandemic. For simplicity, we assume a smooth path of the protection parameter $\epsilon(x)$ in the transition to an endemic state, ignoring the cyclical pattern caused by the season and the arrival of new mutants of the virus (these features could be added without providing further insights). We calibrate the path of protection and treatment technology using the logistic function

$$\epsilon(x) = \epsilon(0) + [\epsilon(\infty) - \epsilon(0)] \left[1 - \exp(-x/\theta)^{\zeta}\right] \tag{10}$$

where x is the number of years since the onset of the pandemic. We calibrate a fast and slow scenario. In the fast scenario, ϵ improves quickly (adjustment halftime of 3 years) and in the fast scenario ϵ improves only gradually initially and then with increasing speed (adjustment halftime of 5 years). The two scenarios are shown in Figure A.3 in the Appendix.

Example life cycle trajectories are shown in Figure 5. Blue lines repeat the outcome for the calibrated benchmark individual (from Section 5) with no pandemic experience. Red dashed lines show the same individual when experiencing at age 75 an onset of the calibrated pandemic with an adjustment halftime of the protection technology of 3 years. For better visibility of the decisive part of the life cycle, ages below 60 are not shown.

FIGURE 5. Life Cycle Health with Covid-19 Pandemic



Blue solid lines: benchmark (no Covid-19) model prediction. Red dashed lines: calibration to Covid-19 with onset at age 75 and adjustment halftime of ϵ of 3 years.

[13]Since the Covid-19 pandemic started at the end of January 2020, the estimate in Wang et al. (2022) indicates an excess death rate of around 0.1 percent in the first year of the pandemic.

As shown in the middle panel of Figure 5, mortality from infections explodes on impact and gradually decreases without ever returning to its original path. The deviation after the transition is caused by two effects: (i) the disease environment with endemic Covid-19 is less healthy than before the pandemic (increase in $\beta > 0$); (ii) increased exposure to infections has led to faster accumulation of health deficits (driven by $B > 0$), visible in the panel on the left-hand side of Figure 5, and the associated faster aging of the immune system has increased the susceptibility to infections (driven by $\nu > 0$). The panel on the right-hand side of Figure 5 shows that the individual responds to the pandemic by investing more in health to slow down aging and thereby reduces the susceptibility to infectious diseases.

Table 2 reports the results from comparative dynamics in form of simple life cycle statistics. These statistics are similarly constructed as in Table 1 with some important modifications. First, the deviation is no longer shown in relation to the calibrated benchmark but displays, for any case, the (relative) deviation in outcomes for the same individual with and without pandemic experience.[14] Moreover, the summary statistics are computed from the age at pandemic shock onwards. For example, ΔLE in case 1) is the change in life expectancy at age 75 caused by the pandemic and ΔV is the relative change in the value of life experienced at age 75 caused by the pandemic. Likewise, Δd_I and Δd are the changes in the average annual death rate from infections and all-causes for the population aged 75+.[15] The new statistics ΔD^{+10} is the change in health deficits measured 10 years after the onset of the pandemic. It allows to assess the impact of the pandemic on the speed of aging and the development of chronic diseases. Taking into account that most values are small, relative deviations are reported in percent.

Case 1) of Table 2 shows morbidity and mortality statistics for the individual considered in Figure 5. The pandemic is predicted to increase the death rate from infections by almost 100 percent and increased exposure to infections leads to faster aging. Ten years into the pandemic, the individual has developed 2 percent more health deficits, increasing the all-cause mortality by 7.3 percent. Life expectancy is predicted to decline by about half a year. Given the sharp increase in death rates, the change in life expectancy may seem small in absolute terms. However, since the remaining life expectancy at age 75 is about 8 years, the pandemic is expected to shorten the life of 75-year-olds by more than 6 percent. Expected health investments decline after the pandemic. Since we have already seen in Figure 5 that health investments by age increase in response to the pandemic shock, the result implies that decreasing survival rates dominate increasing expenditure in their contribution to the aggregate outcome.

Case 2) and 3) consider (otherwise identical) individuals affected by the same pandemic shock at age 60 and 45, again for an adjustment haltime of ϵ of 3 years. Due to their better state of health, younger individuals face a much lower (though still substantially increased) risk of death from infectious disease. Since the disease is experienced as less severe, the feedback effect on the development of chronic health deficits and all-cause mortality is also smaller. However, the

[14] The calibrated benchmark and the outcome without pandemic differ, for instance, if we consider an individual whose labor income w, initial health deficits D_0, or disutility from protection ω differs from the calibrated values in Section 5.

[15] In analogy to (9), we define the crude mortality rate from all causes after shock age t_0 by $d \equiv \int_{t_0}^{T} \mathcal{S}(t) m(t) \mathrm{d}t / \int_{t_0}^{T} \mathcal{S}(t) \mathrm{d}t$.

Table 2: Health and Longevity with Covid-19 Pandemic: Comparative Dynamics

case	parameter	remark	shock age	ΔLE	Δd_I	Δd	ΔD^{+10}	$\Delta\tilde{h}$	ΔV
1)	$\theta = 4.1$, $\zeta = 1.3$	adjustment halftime 3 years	75	-0.53	98.52	7.33	1.97	-3.86	-4.57
2)	$\theta = 4.1$, $\zeta = 1.3$	adjustment halftime 3 years	60	-0.46	27.15	2.45	0.78	-0.86	-1.15
3)	$\theta = 4.1$, $\zeta = 1.3$	adjustment halftime 3 years	45	-0.42	13.83	1.30	0.64	-0.52	-0.63
4)	$\theta = 6.0$, $\zeta = 2.3$	adjustment halftime 5 years	75	-0.73	145.79	10.33	3.20	-4.98	-6.34
5)	$\theta = 6.0$, $\zeta = 2.3$	adjustment halftime 5 years	60	-0.62	36.09	3.37	1.14	-1.51	-1.87
6)	$\theta = 6.0$, $\zeta = 2.3$	adjustment halftime 5 years	45	-0.56	16.65	1.73	0.93	-0.86	-0.96
7)	$D_0 = 1.1D_{0,bench}$, $\theta = 4.1$, $\zeta = 1.3$	10 percent less healthy and 2)	60	-0.50	45.93	3.94	1.54	-2.73	-3.09
8)	$D_0 = 1.1D_{0,bench}$, $\theta = 6.0$, $\zeta = 2.3$	10 percent less healthy and 5)	60	-0.69	63.97	5.49	1.91	-3.72	-4.26
9)	$\omega = 2, \theta = 4.1$, $\zeta = 1.3$	lower protection (mean $p = 0.7$) and 2)	60	-1.62	102.09	9.16	5.08	-28.30	-7.38
10)	$\omega = 4, \theta = 4.1$, $\zeta = 1.3$	much lower protection (mean $p = 0.25$) and 2)	60	-2.87	450.58	22.72	5.88	-49.95	5.21
11)	$B = 0$, $\theta = 4.1$, $\zeta = 1.3$	no feedback effect and 2)	60	-0.03	22.63	0.22	-0.00	-0.09	-0.36
12)	$B = 0$, $\theta = 6.0$, $\zeta = 2.3$	no feedback effect and 5)	60	-0.05	29.04	0.29	-0.00	-0.14	-0.47
13)	$B = 0$, $\theta = 4.1$, $\zeta = 1.3$	no feedback effect and 2)	75	-0.07	76.24	1.07	-0.00	-0.87	-1.30
14)	$B = 0$, $\theta = 6.0$, $\zeta = 2.3$	no feedback effect and 5)	75	-0.11	106.36	1.49	-0.00	-1.28	-1.74

The table shows the predicted deviation of health behavior and health outcomes from the outcomes for the same individual without pandemic shock. All entries are measured from shock-age onwards. The index *bench* identifies the calibrated benchmark value; ΔLE is the change in life expectancy at shock age. The other entries are percentage deviations from the no-shock case. Δd_I (Δd) is the relative deviation of the crude mortality rate from infectious diseases (from all causes) at shock age; ΔD^{+10} is the relative change of chronic health deficits, measured at shock age + 10; $\Delta\tilde{h}$ is the relative change in expected health expenditure; ΔV is the relative deviation of the value of life at shock age.

predicted impact of the pandemic on life expectancy is almost the same regardless of age at the onset of the pandemic. This perhaps surprising finding is explained by the permanent deterioration of the infectious disease environment (higher β) that younger individuals are exposed to for a longer time. In other words, the arrival of the new virus has sizable long-lasting effects even for younger individuals who experience a comparatively small increase in health deficits immediately after the pandemic shock. As will become apparent, salient for the result is the feedback effect from infectious diseases on the development of chronic health deficits ($B > 0$), associated with chronic inflammation. However, the decrease in the value of life is significantly lower for younger individuals because the expected end of life is further away. Cases 4) to 6) confirm these results for a slower transition to an endemic state (adjustment haltime of ϵ of 5 years). The effects of the pandemic are getting bigger, life expectancy, for example, is now falling by 0.6-0.7 years.

Comparative dynamics with respect to initial health deficits (pre-existing conditions) are shown in cases 7) and 8), which consider an individual who is initially 10 percent less healthy (initial health deficits $D_0 = 0.297$ instead of $D_0 = 0.27$). The self-productivity of health deficits implies that at age 45 (60, 75) he/she has 20 (26, 30) percent more health deficits than the benchmark individual (not shown). As a result, when hit by the pandemic shock at age 60, the increase in infection mortality is about 46 percent (short duration of the pandemic) or 64 percent (long duration of the pandemic) higher than for the benchmark individual considered in the respective cases 2) and 5). That frail people (with more pre-existing conditions) suffer particularly badly from a long-lasting pandemic is also seen in the increase in health deficits 10 years after the onset of the pandemic and the increase in all-cause mortality.

The role of protection effort is evaluated by cases 9) and 10). Recall that by setting $\omega = 0$ the benchmark individual is calibrated to chose maximum protection. By setting $\omega = 2$, case

9) considers an individual with moderate anti-protection preferences (concerning perhaps mask wearing or social distancing). The average protection level chosen by the individual is 70 percent. The decrease in life expectancy and the increase in infectious and all-cause mortality caused by the pandemic is more than three times higher than for the benchmark individual. Case 10) considers an even stronger anti-protection attitude (perhaps concerning vaccination) for which protection effort declines to about a quarter of maximum level. As a result, the increase in the infectious disease mortality rate is more than four times higher than in the moderate case and life expectancy declines by almost three years.

Finally, cases 11)–14) highlight the importance of inflammaging for these results. When B is set to zero, aging and chronic health conditions remain unaffected by the pandemic. Although mortality from infectious diseases is increasing significantly during the pandemic, the impact on life expectancy is rather small. This result reflects the fact that even during a severe pandemic, most people survive or die from chronic diseases. The greatest health burden of the Covid-19 pandemic is therefore its impact on aging and the development of chronic diseases, and it may be felt by middle-aged and elderly survivors of infections only long after the new disease has become endemic.[16]

9. Concluding Remarks

This paper has presented a new economic life cycle theory of physiological aging and longevity that takes into account the interaction between infectious and chronic diseases. 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. Since chronological age is immutable while physiological age is malleable, these insights underline the importance of the health care system for avoiding high hospitalization rates and high death tolls in pandemics. The life cycle model features 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, employed the model in counterfactual analysis to assess the drivers of American epidemiological transition from 1860 to 2000, and studied the sudden introduction of Covid-19 as a pandemic that is gradually becoming endemic.

Our analysis generated several insights into mechanisms and quantitative importance for health outcomes and health care expenditure with potential policy relevance. 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. Moreover, our model supports a refined view of the epidemiological transition that

[16]See Goldstein and Lee (2020) for an extensive discussion of the small effect of epidemic mortality on life expectancy when there is no feedback effect on the health of survivors.

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 longer life spans before the onset of medical advances in chronic disease treatment in the 1960s.

Relatedly, the model predicts that poor and unhealthy people are hit particularly hard by the Covid-19 pandemic and that poverty and frailty are mutually reinforcing in their adverse effects on disease susceptibility and mortality. We have also shown how frailty causes an age-gradient of Covid-19 deaths which is very similar to that of all-cause mortality and how individual characteristics such as frailty and anti-protection preferences affect morbidity and mortality. Taking chronic inflammation into account explains why people continue to suffer from chronic conditions caused by the pandemic long after infections have ended (Long-Covid syndrome). As a result of inflammaging, the greatest future health burden of the Covid pandemic will thus only become apparent when survivors of the pandemic suffer from an infection-induced increase in frailty and excess mortality. Predictions of the calibrated model suggest that the pandemic will reduce life expectancy for 45-year-olds by about as much as for 75-year-olds, although the latter are much more affected in terms of mortality from infections. The reason is that middle-aged individuals are exposed to increased infection-induced aging for a longer period of time, in which increased health deficits can lead to the emergence of new health deficits.

This study is the first attempt to integrate the interaction between infections and chronic diseases into life-cycle economics, 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.

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Appendix

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

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

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

From differentiation of (3):

$$\frac{\dot{\mathcal{S}}}{\mathcal{S}} - \sigma\frac{\dot{c}}{c} = \frac{\dot{\lambda}_k}{\lambda_k} \tag{A.3}$$

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

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

From differentiation of (4):

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left(\frac{\dot{\lambda}_D}{\lambda_D} - \frac{\dot{\lambda}_k}{\lambda_k}\right) \tag{A.4}$$

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

Table A.1: Robustness of Comparative Dynamics: $r = 0.05$

case	par. 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	-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

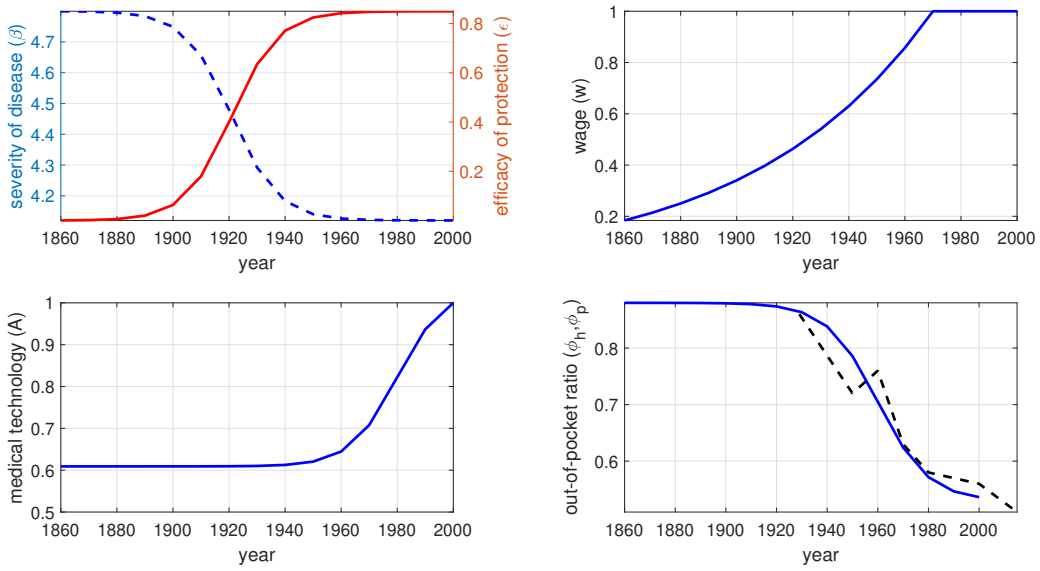
The table shows the predicted deviation of health behavior and health outcomes as in Table 1 for the recalibrated model with $r = \rho = 0.05$, $\sigma = 1.02$. Other parameters as for Table 1.

Table A.2: Robustness of Comparative Dynamics: $r = 0.03$

case	par. 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	-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

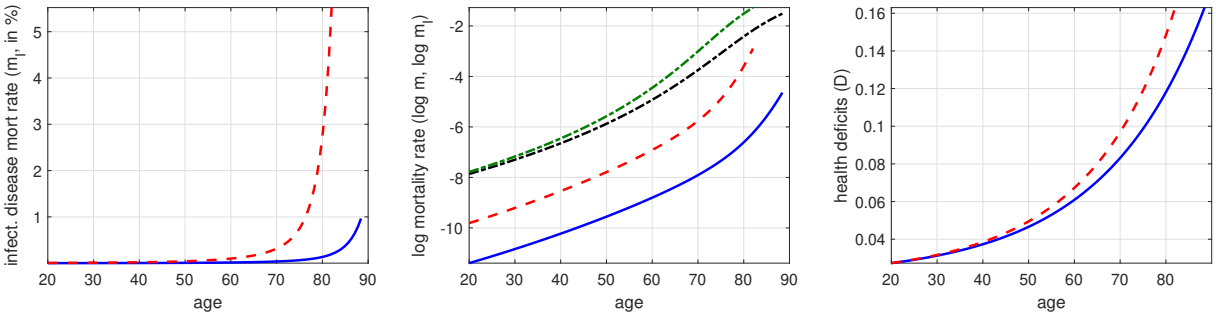
The table shows the predicted deviation of health behavior and health outcomes as in Table 1 for the recalibrated model with $r = \rho = 0.03$, $\sigma = 0.90$. Other parameters as for Table 1.

Figure A.1: The Epidemiological Transition: Calibration



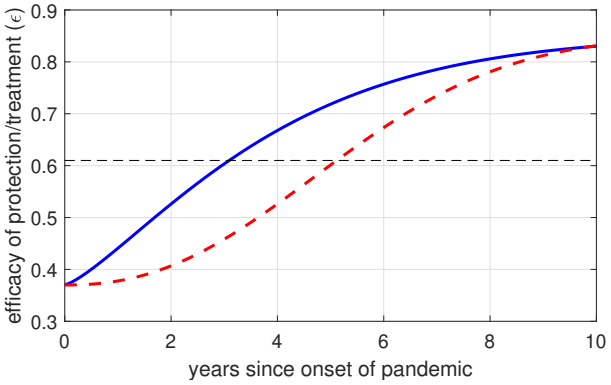
The figure shows the calibrated time paths used in the discussion of the epidemiological transition. See text for details. The black dashed line in the lower-right panel shows the evolution of one minus the public share of total health expenditure according to Getzen (2017). The time series of A shows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

Figure A.2: Covid as a New Endemic Disease



Blue solid lines: benchmark (no Covid) model prediction. Red dashed lines: calibration to Covid-19. The Figure counterfactually assumes that Covid is permanent at the current age-specific death rate. In the center panel, the blue solid line and the red dashed line show log of the infectious diseases mortality rate before and after Covid and dash-dotted lines show the log of the all-cause mortality rate before Covid (black) and after Covid (green).

Figure A.3: Course of the Pandemic



The figure shows the imposed transitional dynamics of protection and treatment technology during the Covid pandemic, as given by eq. (**??**). Solid blue lines: $\theta = 4.1$, $\zeta = 1.3$ (adjustment halftime of 3 years). Red dashed lines: $\theta = 6.0$, $\zeta = 2.3$ (adjustment halftime of 5 years).