CESifo WORKING PAPERS

11104
2024

May 2024

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CESifo

Impressum:

CESifo Working Papers
ISSN 2364-1428 (electronic version)
Publisher and distributor: Munich Society for the Promotion of Economic Research - CESifo GmbH
The international platform of Ludwigs-Maximilians University's Center for Economic Studies and the ifo Institute
Poschingerstr. 5, 81679 Munich, Germany
Telephone +49 (0)89 2180-2740, Telefax +49 (0)89 2180-17845, email office@cesifo.de
Editor: Clemens Fuest
https://www.cesifo.org/en/wp
An electronic version of the paper may be downloaded
· from the SSRN website: www.SSRN.com
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CESifo *Working Paper No. 11104*

Beyond Covid: Pandemics and the Economics of Aging and Longevity

Abstract

In this paper, we examine the effects of the Covid-19 pandemic on individual aging and longevity with special focus on socioeconomic disparities in health outcomes. We also explore the individual-specific effects of Long Covid. We develop and calibrate a health economic model based on principles of the biology of human aging that captures the interaction between infections and chronic health deficits. Our analysis suggests that neglecting this interaction leads to a gross underestimation of the long-term health impact of the pandemic. Our model also explains large socioeconomic health differences that can be attributed to infection protection behavior.

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

Keywords: Covid-19, Long Covid, health behaviour, health deficits, health inequality, protection aversion, false beliefs, longevity.

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April 30, 2024
We would like to thank seminar participants at the University of Essen, University of Hohenheim, the VERB group of the International Health Economic Association, and the Basel Center for Health Economics for helpful comments. Financial support of the Swiss National Fund (SNF) for the project “The Socioeconomic Health Gradient and Rising Old-Age Inequality” (grant no. 100018L 15009) is gratefully acknowledged. Declarations of interest: none.

1. Introduction

In this study, we investigate a hitherto unexplored topic, namely how exposure to a pandemic and individual protection behavior affect long-term health outcomes such as the development of chronic health deficits and death from non-communicable diseases. We develop an economic model of biological aging and longevity that takes the interaction between infections and chronic diseases into account and apply it to the Covid-19 pandemic. Formally, the Covid pandemic is understood as a shock in the infectious disease environment and a temporary decline in disease prevention technology. It induces adjustment behavior and convergence towards a state in which Covid has become endemic. Our analysis shows how neglecting the interaction between infections and chronic diseases leads to a gross underestimation of the long-term health impact of the pandemic. It also explains how a pandemic affects individuals unequally, which allows us to understand the socioeconomic differences in health outcomes associated with the lifelong impact of infectious diseases.

Empirical evidence suggests that there are considerable socioeconomic inequalities in Covid-related health outcomes that are not well understood (Lassale, 2020; Niedzwiedz et al. 2020; Patel et al., 2020; for a survey see Wachtler et al., 2020). A list of possible reasons includes socioeconomic differences in protective behavior, such as seeking vaccination (e.g. Caspi et al., 2021; Okubo et al., 2021; Saban et al., 2021; Thakore et al., 2021; Kim, 2023; Pouliasi et al., 2023), and in the prevalence of pre-existing non-communicable diseases like diabetes, cardiovascular issues, and chronic respiratory conditions (Bambra et al. 2020). Moreover, for a significant share of the population (estimated at or above 10 percent), a Covid infection leads to the development of prolonged or even persistent health deficits in the aftermath of the initial infection, the so-called Long Covid syndrome. While the causes of Long Covid are not yet fully understood, it is now clear that the disease affects basically all organ systems and can be expressed as a general increase in chronic health deficits (see Altmann et al., 2023; Davis et al., 2023, for recent reviews).[1]

In our health economic model, health deficits are measured by an established methodology in the medical sciences, the frailty index (following Dalgaard and Strulik, 2014). The frailty index is the relative number of aging-related illnesses and functional limitations that an individual has, given a large number of potential health deficits (Searle et al., 2008). In line with the empirical evidence, the frailty index increases quasi-exponentially with age (e.g. Mitnitski et al., 2002a,b; Rockwood and Mitnitski, 2006, 2016; Abeliansky and Strulik, 2018; Abeliansky et al., 2020; Dalgaard et al., 2022). However, chronological age does not cause the deterioration of health. The health deficit model is built on insights from the biology of human aging (Gavrilov and

[1]We use the term 'Long Covid' throughout. Alternative terms in the literature are 'Post-acute COVID-19 syndrome' and 'Chronic COVID-19'. According to WHO (2021): "Post COVID-19 condition occurs in individuals with a history of probable or confirmed SARS-CoV-2 infection, usually 3 months from the onset of COVID-19 with symptoms that last for at least 2 months and cannot be explained by an alternative diagnosis. Common symptoms include fatigue, shortness of breath, cognitive dysfunction but also others [...] which generally have an impact on everyday functioning." On January 18, 2024, the United States Senate Committee on Health, Education, Labor and Pensions discussed Long Covid for the first time as pressing health issue; see Al-Aly (2024) for the testimony.

Gavrilova, 1991; Rutenberg et al., 2018) and conceptualizes aging as the intrinsic, cumulative, progressive, and deleterious loss of function (Arking, 2006). The observed quasi-exponential accumulation of health deficits is explained by the self-productivity of health deficits, i.e. the feature that an increase in existing health deficits leads to the faster development of new deficits (Dalgaard and Strulik, 2014; Dragone and Vanin, 2022).

In contrast to chronological aging, biological aging is malleable. It can by slowed down by health investments and accelerated by unhealthy behavior. Importantly, in the present application to the Covid pandemic, health deficits are also influenced by infections and individuals can protect themselves from infections depending on the level of medical technology. In their behavior, individuals take into account that mortality risk from both infectious diseases and chronic conditions depends on the amount of accumulated health deficits that evolve over the life cycle. They also understand, at least to a certain extent, how the development of health deficit depends on deliberate efforts to prevent and treat infectious diseases and chronic health conditions. However, we also explore the health consequences when individuals hold decidedly false beliefs about the severity of infections and the efficacy of protection.

The health deficit framework allows us to examine a number of previously neglected aspects of the Covid pandemic such as how infectious disease mortality depends on pre-existing health conditions (rather than chronological age) and how infections influence the development of chronic health deficits and post-pandemic mortality. We investigate how these mechanisms, known in medicine as immunosenescence and inflammaging, affect disease protection behavior and curative health care spending and therewith individual longevity.

The term immunosenescence refers to the aging immune system. Since aging, understood as the progressive decline of organ reserve and function, is encompassing, it includes also the gradual decline in functionality of the innate and adaptive immune system. According to current medical knowledge, increasing infectious disease mortality is therefore not due to advancing chronological age *per se* but rather to increasing frailty and the weakened immune system (e.g. Santoro et al., 2021). A recent study by Cao et al. (2022) suggests that the biological age measured in blood samples, correlates not only strongly with all-cause mortality but also with the risk of developing severe Covid disease. Several studies showed that the frailty index is an important determinant of the severity and mortality risk of a Covid infection (e.g. Petermann-Rocha et al., 2020; Howlett et al., 2021).[2]

The term inflammaging refers to a permanent low-grade and chronic state of inflammation that has been associated with many chronic diseases and, at the cellular level, with all of

[2]Aging of the innate immune system includes ineffective pathogen recognition and macrophage activation and the decreased function of epithelial barriers of the skin, lung, and gastrointestinal tract. Aging of the adaptive immune system is, *inter alia*, characterized by a decline in the number and functionality of T- and B-cells. (e.g. Miller, 1996; Weiskopf et al., 2009).

the nine hallmarks of aging.[3] Exposure to bacteria, viruses, fungi, and parasites often causes chronic pro-inflammatory conditions (Franceschi and Campisi, 2014; Franceschi et al., 2017). A significant part of the declining prevalence of chronic respiratory problems, valvular heart disease, arteriosclerosis, and joint and back problems since the early 20th century has been attributed to reduced exposure to infectious diseases (Costa, 2000). With respect to Covid infections, recent research suggests that SARS-CoV-2 is a reservoir of peptide fragments that can cause enhanced pro-inflammatory responses with potentially very harmful health effects for some patients (Zhang et al. 2024). For instance, patients with severe Covid often develop bacterial pneumonia that exacerbate proinflammatory responses (Bartleson et al., 2021).

The more or less spontaneous deterioration in health of Long Covid patients can also be adequately captured by the health deficit model. The first large-scale and long-term Long Covid study by Bowe et al. (2023) documents a massive increase in the incidence of 80 chronic health deficiencies two years after the initial infection of the Long Covid patient. We use these data, construct a frailty index, and find that health deficits in Long Covid patients increase by an average of 25 percent.

We examine two main research questions. First, we explore the short- and long-term health effects of the Covid pandemic, depending on (i) the individual state of health at the onset of the pandemic, (ii) individual aversion to protective care (e.g. disutility from wearing face masks or getting vaccinated), (iii) earnings, (iv) age at the outbreak of the pandemic, and (v) false beliefs about both the efficacy of disease protection and the prevalence of infectious diseases. By comparing the calibrated average American with other model individuals with specific characteristics, we shed light on the heterogeneity of health behavior and health outcomes in infectious disease environments. Second, we investigate how the long-term health effects of Long Covid depend on individual characteristics.

For the benchmark case, our model predicts that the Covid pandemic has shortened the remaining life expectancy of the average American at age 75 by about five months. The predicted loss of life expectancy due to Covid is more than three times lower when inflammaging is ignored. This significant underestimation of the health consequences of the Covid pandemic when inflammaging is neglected appears to be consistent with the general view that the pandemic had essentially no impact on the life expectancy of pandemic survivors.

Protection aversion naturally increases mortality from infectious diseases before, during and after the pandemic. For example, for strongly protection averse individuals (who only begin to protect themselves against infections in their late 60s), the pandemic-related increase in chronic disease mortality is predicted to be four times greater than for the benchmark American with modest protection aversion. The loss in life expectancy gets larger for protection-averse individuals with low income or pre-existing health conditions. These results contribute to a

[3]See e.g. Furman et al. (2019), Lopez-Otin et al. (2013), and Kennedy et al. (2014). The nine hallmarks of aging are: (1) genomic instability, (2) shortening telomere length, (3) epigenetic modifications, (4) loss of proteostasis, (5) deregulated nutrient sensing, (6) mitochondrial dysfunction, (7) cellular senescence, (8) stem cell exhaustion and (9) altered intracellular communication, which are all linked to sustained systemic inflammation (Lopez-Otin al., 2013). In our companion paper (Strulik and Grossmann, 2024) we provide a more detailed discussion of inflammaging in the context of the epidemiological transition.

better understanding of health inequality arising from infectious diseases, as empirically the level of protection aversion and income are negatively correlated. Interestingly, life expectancy differences between individuals are generally higher if the onset of the pandemic is experienced at an earlier age although its immediate impact on mortality risk is then considerably lower. This perhaps surprising outcome is a direct consequence of the self-productive nature of health deficits.

The Covid pandemic has shown that some people have persistent misconceptions about the mortality risk of a new virus (e.g. van Mulukom et al., 2022) and the efficacy of vaccines (e.g. Baeza-Rivera, 2021; Lazarus et al., 2022). We show that false beliefs do not only lead to an alleviated increase in infectious disease mortality but also to substantially faster aging and premature death from chronic diseases. For example, an individual aged 60 at the onset of the pandemic, who believes that infections are (almost) harmless and protection (almost) useless, is predicted to have 19 percent more chronic health deficits at age 70 and to die more than 6 years earlier, compared to the benchmark American with correct beliefs, all other things being equal. The life expectancy difference can be much higher, if the individual has, in addition, low income or more initial health deficits.

In addition, our analysis suggests a rather bleak outlook for Long Covid patients. The self-productivity of health deficits implies that the health gap between Long Covid patients and the calibrated benchmark American widens with advancing age. Related to that, Long Covid causes a greater loss in life expectancy if it occurs at young age. Our model predicts that the benchmark American will lose 4 years of life if getting Long Covid at age 75 and 6.6 years if it happens at age 45.

The Covid pandemic has triggered countless studies in medicine, but also a large body of research in economics. First, there is an important economic literature that focuses on how the spread of the disease is influenced by policy measures like testing or confinement. Theoretical approaches typically use epidemiological models to capture the infection dynamics of the pandemic (e.g. Acemoglu et al., 2021; Donsimoni et al., 2020), sometimes enriched with health behavior and its externalities (Brotherhood et al., 2020). Moreover, there is a growing literature on macroeconomic effects of the pandemic. For instance, Chetty et al. (2023) show that spending fell more in counties with higher rates of Covid infection, largely to avoid catching the disease rather than concerns to lose income. Goolsbee and Syverson (2021) and Chetty et al. (2023) find modest macroeconomic effects of government-imposed restrictions to contain the spread of the virus. Guerrieri et al. (2022) argue that standard fiscal stimulus policies may be quite ineffective in a pandemic. Second, there is a literature on how to optimally prioritize vaccines (e.g. Akbarpour et al., 2024; Bubar et al., 2021; Gans, 2022). For instance, Akbarpour et al. (2024) models vaccination behavior and shows that pricing of vaccines can be optimal in the presence of asymmetric information about the private willingness to pay. However, none of the previous studies has examined long-term health consequences of the pandemic.

The health-deficit framework of Dalgaard and Strulik (2014) has been applied to various contexts, but typically abstained to model infectious diseases. An exception is our accompanying paper (Strulik and Grossmann, 2024), where we examined health behavior of men and women

when infectious diseases are endemic, investigating the role of income, initial health status, and other individual characteristics. We analyzed how human aging and life expectancy during the epidemiological transition from 1860 to 2010 can be explained by the feedback effects between infections and chronic diseases. Using counterfactual historical experiments, we assessed the impact of medical technology on mortality from infectious diseases, all-cause mortality, life expectancy, and the value of life. However, we did not explore how the outbreak of a new disease (i.e. a pandemic) or an individual health shock (like Long Covid) affects health behavior and long-term health outcomes. In contrast to the present study, we have also abstracted from misconceptions about the mortality risk of infections.

The remainder of the paper is organized as follows. In section 2, we introduce the model and discuss the comparative statics of protection and treatment of infectious diseases. In section 3, we calibrate the model for an average male American before the pandemic and in Section 4 we calibrated the Covid pandemic. The results are presented in Section 5. We examine the role of initial health deficits, earnings, preferences (protection aversion), and age for short- and long-term health outcomes. We then explore the effects of false beliefs on health outcomes. Finally, we implement the Long Covid syndrome and discuss its impact on future health outcomes. Section 6 concludes the paper.

2. Model

2.1. **Individual Welfare.** Individuals live in an environment with uncertain survival and maximize their expected lifetime utility. Let $S(t)$ denote the probability of being alive at age t (survival function). As usual, we normalize the utility of being dead to zero. Individuals derive utility from consumption of a numeraire good, c. Since we are particularly interested in disease protection behavior, we additionally implement the feature that individuals may experience disutility from disease protection effort, p. Instantaneous utility is given by $v(c,p) = u(c) - \omega p$, where the utility weight of diseases protection $\omega > 0$ is called protection aversion and we focus on an iso-elastic utility function $u(c) = (c^{1-\sigma} - 1)/(1-\sigma)$, with $\sigma > 0$ (and $u(c) = \log c$ for $\sigma = 1$). Future payoffs are discounted at the time preference rate $\rho > 0$. Thus, expected lifetime utility at age τ is given by

$$V(\tau) = \int_{\tau}^{T} S(t) \cdot [u(c(t)) - \omega p(t)] \cdot \mathrm{e}^{-\rho(t-\tau)} \mathrm{d}t, \tag{1}$$

where lifespan T, i.e. the maximum length of life, is an endogenous variable, as explained below. All parameters are individual-specific. We are particularly interested in the role of protection aversion parameter ω, which governs the age at which individuals start protection ($p > 0$). By protection aversion we capture, for instance, concerns about the safety of vaccines, which have been shown to be a major reason for Covid vaccine hesitancy (Lazarus et al., 2022).[4] Empirical evidence suggests that vaccine hesitancy and vaccination rates strongly depend on socioeconomic

[4]The WHO defines vaccine hesitancy as "the delay in acceptance or refusal of vaccination despite availability of vaccination services" (MacDonald et al., 2015).

status, with protection aversion being higher for individuals with lower educational attainment and lower income (Pouliasi et al., 2023).[5]

2.2. **Survival, Mortality, and Health Deficits.** The mortality rate at age t is defined as the negative survival rate, $\dot{S}(t)/S(t) = -m(t)$, implying that the probability of being alive at age t is given by $S(t) = S(0)\mathrm{e}^{-\int_0^t m(t)\mathrm{d}\tau}$. Because of our particular focus on the interaction between death from chronic diseases and from infections we decompose the mortality rate as $m(t) = m_C(t) + m_I(t)$, in which m_C is the mortality rate from chronic diseases and m_I is the mortality rate from infections. We explicitly implement the basic insight from gerontology that death is not explained by chronological age but by the state of health (Arking, 2006). The state of health is measured by the frailty index D, which is is relative number of health deficits that are present in a person, given a long list of potential health deficits. The health deficit index has been developed by Mitnitski et al. (2001) and has used in countless studies in the medical science due to its simplicity, comparability, and excellent predictive power for health related events, such as death or entry into long-term care. See Searle et al. (2008) for details of index construction and Howlett et al. (2021) for a recent review of the literature. For the mortality rate from chronic disease, we write $m_C = \tilde{m}_C(D)$. Aside from health deficits, the mortality rate from infections additionally depends on the protective measures against infections, $m_I = \tilde{m}_I(D, p)$. Summarizing, individual survival is described by the law of motion

$$\dot{S}(t) = -\left[\tilde{m}_C(D(t)) + \tilde{m}_I(D(t), p(t))\right] \cdot S(t). \tag{2}$$

For the sake of simplicity, we henceforth omit the age index of the variables unless it is needed for clarity.

2.2.1. *Chronic Disease Mortality.* In advanced countries, the vast majority of people die from chronic (non-communicable) diseases. For example, in the U.S. in 2019, 89.6 percent of deaths were caused by chronic diseases, 4.3 percent by infections, and 6.1 percent by injuries (Vos et al., 2020). The effect of health deficits on death is well described by a power law:

$$\tilde{m}_C(D) = \xi D^{\psi}, \quad \text{for } D < \bar{D}, \tag{3}$$

where $\xi > 0$ and $\psi > 0$. The parameters of this log-linear relationship have been estimated with great precision, with the coefficient of determination, R^2, being above 0.98 in the raw correlation. The value of ψ has been estimated around 3.0, implying that a one percent increase in health deficits increases the mortality rate by about 3 percent (Mitnitski et al., 2002a,b; Dalgaard et al., 2022, Krenz and Strulik, 2023). Consistent with the literature in gerontology, there is an upper limit to health deficits beyond which survival is impossible, $\tilde{m}_C(\bar{D}) = 1$, such that $D(T) = \bar{D}$ determines the endogenous lifespan (Rockwood and Mitnitski, 2006).

2.2.2. *Infectious Diseases: Protection and Mortality.* Humans are constantly exposed to pathogenic shocks. However, these shocks lead to perceptible illness only if sufficiently severe, with

[5]Earlier evidence on A(H1N1) vaccines (against the influenza virus) confirms this pattern. According to Galarce et al. (2011, p. 5286), "those with a bachelor's or higher degree were 69 percent more likely to perceive the vaccine as safe than those with less than a high school degree."

the threshold shock level depending on the state of health. As in Strulik and Grossmann (2024), we assume that the probability of infection shocks of size s is Pareto-distributed and given by $f(s) = \nu b^{1/\nu} s^{-(\nu+1)}$, with $s \in \{1, \infty\}$ and cumulative distribution function $F(s) = 1 - b/s^{\nu}$, where $\nu > 0$ and $b > 0$. Only shocks of strength greater than $\bar{s}$ result in a potentially lethal infection. The threshold level $\bar{s}$ is inversely proportional to the frailty of the body, capturing the gradual decline in functionality of the innate and adaptive immune system (immunosenescence).

By setting $\bar{s} = 1/D$, we obtain the probability of severe infection as bD^{ν}. A higher value of b implies that more individuals at all states of health become severely sick, i.e. it characterizes a higher prevalence of infectious diseases. The term D^{ν} captures *immunosenescence*, i.e. the influence of the individual state of health (pre-conditions) on the severity of infections. The compound term bD^{ν} is equal to the mortality rate from infections in absence of preventive measures or treatment. As outlined in the Introduction, an aged and increasingly dysfunctional immune system is characterized by a reduced immune response. Moreover, it can cause some cell subsets to become hyperresponsive to infections. A particularly severe variant of dysregulated immune function drew broader attention in the course of the Covid pandemic. In a so-called cytokine storm, an infection causes a rapid and uncontrolled release of inflammatory signaling molecules that leads to inflammation of major organs such as the lungs, kidneys, and heart and may eventually cause organ failure and death (see Bartleson et al., 2021 and Merad et al., 2022, for reviews of the immunology of Covid-19). Several studies showed that the frailty index is an important determinant of the severity and mortality risk of a Covid infection (e.g. Petermann-Rocha et al., 2020; Howlett et al., 2021).

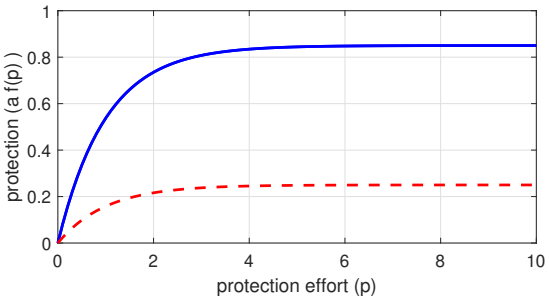
The probability of a lethal infection can be reduced by measures of disease prevention and treatment, which we label as infectious disease protection. The efficacy of protection depends on the state of medical technology, a, and protection effort, p, such that infectious disease mortality is given by

$$\tilde{m}_I(D, p) = [1 - af(p)] \cdot bD^{\nu}, \tag{4}$$

where $a > 0$. More effort reduces the probability of severe infection with decreasing returns, $f'(p) > 0$ and $f''(p) < 0$. For given level of medical technology there are steeply decreasing returns in protection effort. For example, the return of wearing two face masks instead of one or getting two vaccinations instead of one is very small. This means that protection production function $f(p)$ is bounded from above, $\lim_{p\to\infty} f(p) = 1$, such that a is the maximum reduction of the probability of severe infection that can be achieved with the available medical technology.

These features are implemented with the protection function $f(p) = 1 - \mathrm{e}^{-p}$. Figure 1 shows the degree of protection for alternative levels of protection effort and $a = 0.85$ (maximum protection 85 percent, solid blue lines) and $a = 0.25$ (dashed red lines). For example, $a = 0.25$ may represent a technology level where face masks are available and $a = 0.85$ a technology level where vaccines are available. With the specified protection function, when $a = 0.25$, the individual has almost reached the maximum possible protection for $p = 2$ (wearing two masks instead of one does not increase protection by much). For $a = 0.85$ the maximum possible protection is almost reached for $p = 4$.

FIGURE 1. Infectious Disease Protection



The figure shows the protection achieved for alternative levels of protection effort and two levels of medical technology $a = 0.85$ (blue solid lines) and $a = 0.25$ (red dashed lines). Protection is obtained as $af(p)$ with $f(p) = 1-\exp(-p)$. Protection is defined as the percentage reduction of the probability of severe infection.

2.3. **Health Deficit Accumulation.** On average, humans accumulate health deficits in quasi-exponential fashion at a rate of about 3 to 4 percent additional health deficits per year of age (e.g. Mitnitski et al., 2002a,b; Rockwood and Mitnitski, 2016; Abeliansky et al., 2020; Dalgaard et al., 2022). However, the development of new health deficits is not caused by chronological age but by the already existing health deficits. This notion of aging as the intrinsic, cumulative, progressive, and deleterious loss of function (Arking, 2006) has been introduced in health economics by Dalgaard and Strulik (2014) as a law of motion for health deficits. For our application we augment the law of motion for health deficits and write it as

$$\dot{D} = \mu \cdot \left[D - A \cdot (1 + \eta D)^{\delta} \cdot h^{\gamma} + \beta \cdot \tilde{m}_I(D, p) + \epsilon \right], \tag{5}$$

in which $\mu > 0$ is the (natural) rate of aging and ϵ is a (possibly negative) residual capturing health relevant aspects that are not explicitly modeled. The initial level $D(0) = D_0 > 0$ is given.

It is useful to first consider (5) without the A-term and the β-term and without the residual ϵ. The equation becomes $\dot{D} = \mu D$ and describes the self-productive nature of health deficit accumulation. A micro-foundation of this aging process has been provided in reliability theory (Gavrilov and Gavrilova, 1991) and in the network theory of aging (Rutenberg et al., 2018). The solution of the differential equation, $D(t) = D(0)\mathrm{e}^{\mu t}$, implies the exponential growth of health deficits with age. When $D(t)$ is inserted in (3) we obtain the exponential growth of the mortality rate with age, $m_C = \tilde{\xi}\mathrm{e}^{\mu\psi t}$ with $\tilde{\xi} \equiv \xi \cdot (D_0)^{\psi}$. The model thus explains in its reduced form Gompertz law, i.e. an exponential increase in death rates with age (Gompertz, 1825). Since μ is estimated at about 3 percent and ψ at about 3, the model explains why the mortality rate increases by about 9 to 10 percent per year of life, which is a stylized fact of human aging (Arking, 2006). The predicted increase of mortality with chronological age, however, is not causal. Mortality is caused by health deficit accumulation.

While chronological age increases relentlessly and inevitably, the accumulation of health deficits is malleable. As in Dalgaard and Strulik (2014), we assume that individuals can slow down the accumulation of health deficits by health investments h, i.e. health care for prevention

and repair of chronic diseases. The parameter A in (5) measures the general efficacy of medical technology while parameter $\gamma \in (0,1)$ measures decreasing returns in medical expenditure. The term $(1+\eta D)^{\delta}$ implements the notion that the efficacy of curative care increases with the prevalence of health deficits, parameterized by $\eta > 0$ and $\delta > 0$ (see Strulik and Werner, 2021). Since many health deficits are related (e.g. Rutenberg et al., 2018), the treatment of one deficit also slows down the development of other deficits. For example, a treatment that targets hypertension reduces also the risk of stroke, heart diseases, kidney diseases, dementia, and problems of walking fast or sleeping well.

The β-term in (5) measures the influence of severe infections on the development of new chronic diseases. It captures *inflammaging* due to exposure to pathogenes and a resulting chronic inflammatory state (e.g. Franceschi and Campisi, 2014; Franceschi et al., 2017). Consequently, the individual history of infections contributes to the development of chronic health deficits (Finch and Crimmins, 2004; Finch, 2010; Santoro et al., 2021). Sayed et al. (2021) developed an inflammatory clock of aging from biomarkers of the immune system and found it to be highly predictive of the frailty index, greater than chronological age. Recent evidence not only suggests that biological aging is associated with the risk of severe Covid infection but also that Covid may affect the epigenetic clock and telomere attrition (Cao et al., 2022). In our model, the severity of infection is naturally captured by the mortality risk from infection, m_I. Inflammaging thus means that the development of new health deficits, $\dot{D}$, is proportional to m_I with proportionality factor $\mu\beta$ and inflammaging coefficient β.

2.4. **Budget Constraint.** Individuals earn a given flow of earned income w, which consists of net wage income before retirement and pension income thereafter. Individuals face a given return on their savings and a fraction θ of their assets k is annuitized. The annuity provider cannot observe the frailty status of the individual and sets an interest rate depending on the age-specific mortality rate in the population. This means that for saving in annuities the mortality rate m is a direct function of age rather than a function of individual health measure D. Summarizing, financial income from private wealth is given by $(r+\theta m)k$.

Income is spent on consumption c, savings $\dot{k}$, and health care. Let π_h and π_p denote the price of health care aimed at the treatment and prevention of chronic and infectious diseases and ϕ_h and ϕ_p the associated out-of-pocket (or coinsurance) rates. The individual budget constraint is then given by

$$\dot{k} = (r+\theta m)k + w - c - \phi_h \pi_h h - \phi_p \pi_p p. \tag{6}$$

2.5. **Optimal Life Cycle Behavior.** Individuals maximize expected lifetime utility (1) by controlling the paths of consumption (c) and health care inputs (h, p), subject to the law of motion for survival (2), health deficit accumulation (5), and wealth accumulation (6), given the functional forms for mortality rates from chronic and infectious diseases (3) and (4), the initial endowments $k(0)$ and $D(0)$, and the terminal conditions $k(T) = \bar{k} \geq 0$ and $D(T) = \bar{D}$. The solution of this free-terminal time problem of optimal control involves the transversality conditions that the current-value Hamiltonian $\mathcal{H}(T) = 0$ and the shadow price of an increase in survival probability S (co-state variable of condition (2)) $\lambda_S(T) = 0$.

LEMMA 1. *Individual life cycle behavior is described by the solution for infectious disease protection (7) and the equations of motion for consumption (8), health investments (9), the shadow price of health deficits λ_D (10, and the shadow price of survival λ_S (11):*

$$p = \max\{0, -\log(z)\}, \qquad \textit{with } z \equiv \frac{\omega + \phi_p \pi_p c^{-\sigma}}{abD^{\nu}\left[\lambda_S + \frac{\beta\phi_h\pi_h c^{-\sigma}}{A(1+\eta D)^{\delta}\gamma h^{\gamma-1}}\right]} \tag{7}$$

$$\frac{\dot{c}}{c} = \frac{r + (\theta - 1)m - \rho}{\sigma} \tag{8}$$

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left[r + \theta m - \rho + \frac{\delta\eta\dot{D}}{1+\eta D} + \frac{\dot{\lambda}_D}{\lambda_D}\right] \tag{9}$$

$$\begin{aligned}\frac{\dot{\lambda}_D}{\lambda_D} = {} & \rho - \mu + \mu\eta\delta(1+\eta D)^{\delta-1}Ah^{\gamma} - \mu\beta(1 - af(p))b\nu D^{\nu-1} - \\ & \frac{\lambda_S\mu A(1+\eta D)^{\delta}\gamma}{c^{-\sigma}\phi_h\pi_h h^{1-\gamma}}\left[\xi\psi D^{\psi-1} + (1 - af(p))b\nu D^{\nu-1}\right]\end{aligned} \tag{10}$$

$$\dot{\lambda}_S = \left[\rho + \xi D^{\psi} + (1 - af(p))bD^{\nu}\right]\lambda_S - [u(c) - \omega p]. \tag{11}$$

Proof. See Appendix A. □

Equation (8) is the familiar Euler equation for optimal life cycle consumption. Equation (9) is the health-Euler equation for optimal investments in prevention and repair of chronic diseases.[6]

The solution for optimal disease prevention (7) takes into account a potential corner solution of no prevention when prevention aversion ω is sufficiently high. When the solution is interior, we have in the numerator of z the utility cost of disease prevention plus the monetary cost of disease prevention evaluated at the marginal utility of consumption. The term abD^{ν} measures the reduction in infectious disease mortality caused by the first unit of protection effort. The denominator of z therefore reflects the saved health care effort (evaluated at the marginal utility from consumption) and the gain from increased survival (evaluated at the shadow price λ_S) that is caused by the first unit of protection effort.

In order to understand the individual response in infectious disease protection to the outbreak of a pandemic, we next prove the following corollary.

COROLLARY 1. *Optimal behavior implies that the co-state variable λ_S is positive throughout life, $\lambda_S(t) > 0$ for all $t < T$.*

Proof. For the proof, notice that both terms in square brackets in (11) are strictly positive. Therefore, to reach $\lambda_S = 0$ at the end of life, i.e. for $t = T$, λ_S needs to be positive (and declining) throughout life. □

Noticing that λ_S is the shadow price of a marginal increase in survival ($\dot{S}$) the result is intuitively plausible: the value of living longer is positive throughout life, largest in young age, and zero at the maximum lifespan T.

[6]For the special case of no infections ($b = 0$), age-independent health technology ($\delta = 0$), no annuity savings ($\theta = 0$), and certain survival ($\lambda_S = 0$), the equation collapses to the familiar health-Euler equation of the deterministic model in Dalgaard and Strulik (2014), $\dot{h}/h = (1-\gamma)^{-1}(r - \mu)$.

PROPOSITION 1. *For given levels of consumption c and health input h, optimal behavior implies that individuals increase prevention effort p when protection aversion ω or the price of protection $\phi_p\pi_p$ declines or when the product $a \cdot b$ between infectious disease technology (a) and infectious disease prevalence (b) increases.*

Proof. Follows from the derivatives of the interior solution in (7) and Lemma 1. □

The first two results are intuitively obvious. Regarding the last result, consider a pandemic characterized by an increase of infectious disease prevalence b combined with a decline in the efficacy of protection a (as vaccines may not be readily available). Individuals respond to the pandemic by calculating the trade-off between increased prevalence (incentivizing more prevention effort) and declining efficacy of protection (incentivizing less prevention effort). When the disease prevalence increases by more than protection efficacy declines, they increase their protection effort.

The optimal lifetime trajectory is characterized by the path that fulfills the initial conditions and terminal conditions, the laws of motion (2), (5), and (6), the solution (7)–(11), and the transversality conditions $\mathcal{H}(T) = 0$ and $\lambda_S(T) = 0$. We next explore the numerical solution of the calibrated model. We study the effects of the Covid pandemic and the socioeconomic health gradient in the pandemic by capturing the convergence to an endemic state via changes in disease protection technology and disease prevalence.

3. CALIBRATION

3.1. **Calibration of the Pre-Covid Era.** For evaluating the effects of a pandemic, we first need to calibrate the model for the time before the Covid pandemic. We consider an average male U.S. American who starts life in the year 2010 at model-age 0 when being 20 years old. The force of aging μ is set to 0.0337, as estimated by Abeliansky et al (2020) for Caucasian American men. We set the interest rate to $r = 0.06$. Taking the estimates by Jorda et al. (2017), this value corresponds to the long-run real return when wealth consists of 70 percent housing and 30 percent bonds. These values imply an annuity rate $r + m$ of about 7 percent at age 65 and 12 percent at age 85, which is well in line with actual annuity returns (New York Life, 2023). We set $\rho = 0.06$ such that, according to the Euler equation (8), consumption is almost constant over the life cycle (as observed for childless households; Browning and Ejrnæs, 2009) except for high mortality risk m where it is considerably decreasing towards the end of life.

We normalize the total price of health care for chronic diseases, $\phi_h\pi_h$, 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 (2019, Table 8). We set $\phi_p = \phi_h$ and calibrate π_p using the information that, in 2010, 4.0 percent of health expenditure was spent on infectious diseases (BEA, 2022).

Based on the estimates in Dalgaard et al. (2022), we set $\psi = 2.8$. The retirement age is set to 65.5 (CRR, 2018). Johnson et al. (2004) showed that about 10 percent of non-social-security-based wealth is held in form of annuities and hence we set $\theta = 0.1$. We set earnings (w before retirement) to $ 27,928, according to the earnings of single men in 2010 (BLS, 2012). In real terms, labor income of single men in 2019 were only insignificantly higher (BLS, 2020) and also

life expectancy of men remained virtually the same in 2019 (NVSS, 2022). The replacement rate (pension income divided by labor income) is set to 0.47 (OECD, 2013).

A potentially important factor in individual aversion to infectious disease prevention is vaccination. Vaccinations against influenza increase with age, but are generally less common than vaccinations against Covid. In the 2010–2020 period, coverage was around 35 percent in the 18–49 years group, around 50 percent in the 50-64 years age group, and around 70 percent in the 65+ age group (CDC, 2023a). We capture this behavior by setting a value of the ω such that the benchmark American does not protect against infections in young adulthood, chooses around 50 percent protection in middle age and reaches almost full protection after age 65.

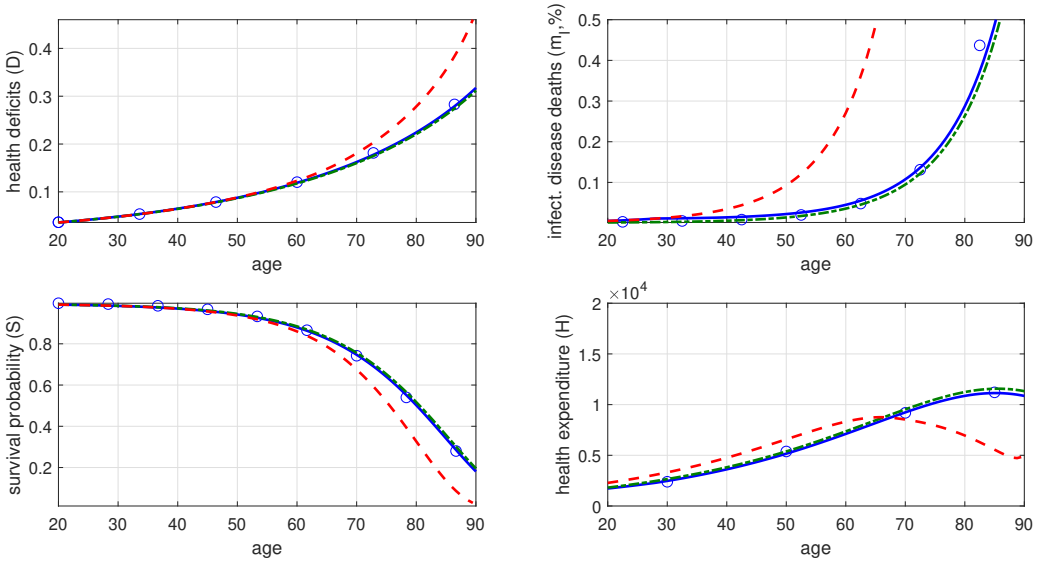
We calibrate the parameters A and β with historical data, as explained in detail in the Appendix. The third phase of the U.S. epidemiological transition reached asymptotically its end around the year 1960 and the treatment of chronic diseases took off with the Cardiovascular Revolution in the 1960s. We therefore assume a level of A close to zero in 1940 and obtain the value of $A(2010)$ that explains the increase of life expectancy at age 20 from 1950 to 2010 (of about 5 years). The parameter β is set such that the epidemiological transition, i.e. the decline of infectious disease mortality from 1850 to 1960 explains the increase of life expectancy at 20 from 1850 to 1950 (of about ten years). For these calculations, we also feed the historical series for wage growth and public health spending into the model. The model thus fully explains the historical path of adult life expectancy.

In addition, we also target the following stylized facts: (a) The accumulation health deficits over a lifetime for American men (as estimated by Abeliansky et al., 2020). (b) The actual survival curve for American men (obtained from estimates in Strulik and Vollmer, 2013), implying 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) The health care expenditure of American men in 2010 at the age of 30, 50, and 70 (data from MEPS, 2010) and age 85 (data from De Nardi et al., 2016). (d) The age profile for deaths from lower respiratory infection for U.S. males, as obtained from the Global Burden of Disease Study (Vos et al., 2020). This calibration target takes into account that even before Covid lower respiratory infections (including pneumonia, bronchitis, and influenza) were by far the most common cause of death from infectious diseases in the U.S. (Armstrong et al., 1999).

Table A.1 in the Appendix shows the calibrated parameter values. The estimated value of $\nu = 3.2$ implies that infectious diseases mortality increases with increasing frailty somewhat steeper than chronic disease mortality ($\psi = 2.8$). The estimated value of $\sigma = 1.01$ implies that the intertemporal elasticity of substitution is close to unity (the utility function is close to log-form), a usual assumption in economics that is also supported in quantitative studies (Chetty, 2006; Layard et al., 2008).

The predicted life cycle trajectories for the benchmark American are shown by blue (solid) lines in Figure 2 and circles show the targeted data. The upper left panel shows the evolution of health deficits, the upper right panel shows the infectious disease mortality in percent, the lower left panel shows the survival probability and the lower right panel shows the evolution of total health expenditure, $H \equiv \pi_h h + \pi_p p$. In line with the evidence compiled in De Nardi et al.

FIGURE 2. Calibrated Model Pre-Covid: Health Outcomes



Blue (solid) lines: model prediction for benchmark individual ($\omega = 0.03$; circles: targeted data. See text for details. Green (dash-dotted) lines: individual with no protection aversion ($\omega = 0$). Red (dashed) lines: individual with strong protection aversion ($\omega = 2.0$).

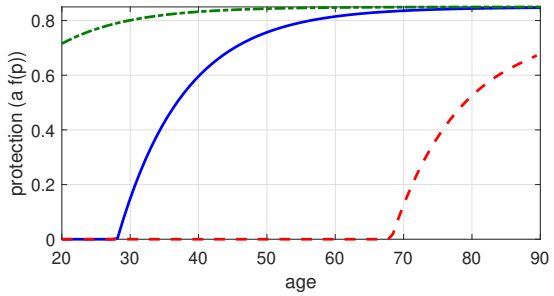
(2016), health care expenditure (net of expenditure for long-term care) reaches a plateau around age 85.

The predicted protection from infectious diseases is shown by the blue (solid) line in Figure 3. There is no protection until age 27, after which protection increases with age (that is with vulnerability to severe infections) and almost full protection is reached after age 65. The health outcomes for an individual with no protection aversion is shown by green (dash-dotted) line in Figure 2 and 3. Although the individual shows significantly more protective efforts at a young age, the health outcomes differ only slightly from the benchmark case. This is so because at young ages the probability of severe infection is very low irrespective of protection effort. This feature changes for the elderly. Red lines in Figure 2 and 3 show health outcomes and protection for an individual with strong protection aversion ($\omega = 2$). The individual starts protecting only at age 70 when there is already a significant probability of severe infection. As a result, infectious disease mortality is much higher, implying faster aging and earlier death.

4. Calibration of 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. With respect to Covid, 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.

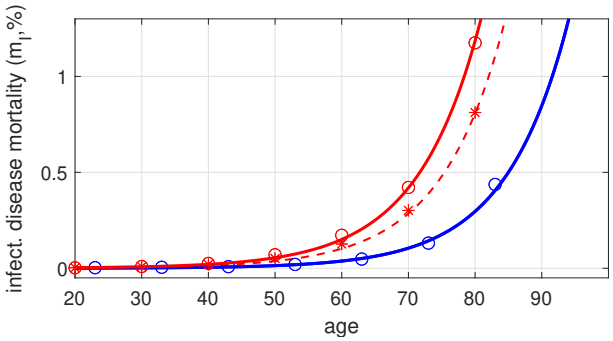
FIGURE 3. Calibrated Model Pre-Covid: Endogenous Protection Effort by Age



Blue (solid) line: model prediction for benchmark ($\omega = 0.03$). Green (dash-dotted) line: individual with no protection aversion ($\omega = 0$). Red (dashed) line: individual with strong protection aversion ($\omega = 2.0$). Protection is obtained as $af(p)$ with $f(p) = 1 - \exp(-p)$.

We begin the calibration of the Covid pandemic by considering its end. We assume that at the endemic state protection against Covid is as good as it is against influenza. Formally, denoting by $a(x)$ the state of the protection technology x years since the onset of the pandemic, we assume $a(\infty) = 0.85$, which equals the previously calibrated value. Moreover, we assume that the long-run Covid death rate equals the death rate from influenza, i.e. 0.0002 percent (American Lung Association, 2015). The targeted mortality rate from infections in our model, m_I, thus increases minimally from 0.078 percent, as implied by the benchmark calibration in Section 3, to 0.0782 percent at the new steady state. This basically means that disease severity b converges from above to reach almost its pre-pandemic value (of 2.2).

FIGURE 4. Infectious Disease Mortality



The figure shows the age-specific male mortality rates for lower respiratory diseases 2019 (blue) and for Covid-19 and lower respiratory diseases combined in 2020 (red). Circles show the targeted data (from Vos et al., 2020, and CDC, 2023b) and solid lines the prediction of the calibrated model. Stars show the combined mortality rate in 2022 and the dashed line shows the model prediction under the benchmark speed of the pandemic (halftime of 2.5 years).

At the beginning of the pandemic, however, the disease environment was much more infectious and the protection technology was substantially weaker. We calibrate the values of $a(0)$ and $b(0)$ such that the model prediction fits the age-specific Covid mortality rates for men in 2020.

The CDC (2023b) reports age-specific mortality rates for the U.S. population as well as gender-specific mortality rates. To obtain our calibration targets, we multiply the age-specific mortality rates with the average ratio of the Covid mortality rate of males (which was 1.25). In Figure 4, the blue line shows the calibrated pre-Covid mortality rate and the blue circles show the pre-Covid mortality rate from lower respiratory infections (from Vos et al., 2020). The red circles show the aggregate infectious disease mortality rate in 2020 (Covid + lower respiratory). The solid red line shows the model prediction for 2020. Notice that the introduction of the new disease shifts the mortality rate upwards but leaves the gradient with respect to age unchanged. Infectious disease mortality increases at a rate of $\nu\mu = 12$ percent per year of age, meaning that it is increasing slightly faster than all-cause mortality, consistent with the observations of Goldstein and Lee (2020).

For simplicity, we assume a smooth path of the transition to the endemic state. This means that we abstract from the cyclical pattern of the disease 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

$$a(x) = a(0) + [a(\infty) - a(0)]\left[1 - \exp(-x/\zeta)^2\right] \tag{12}$$

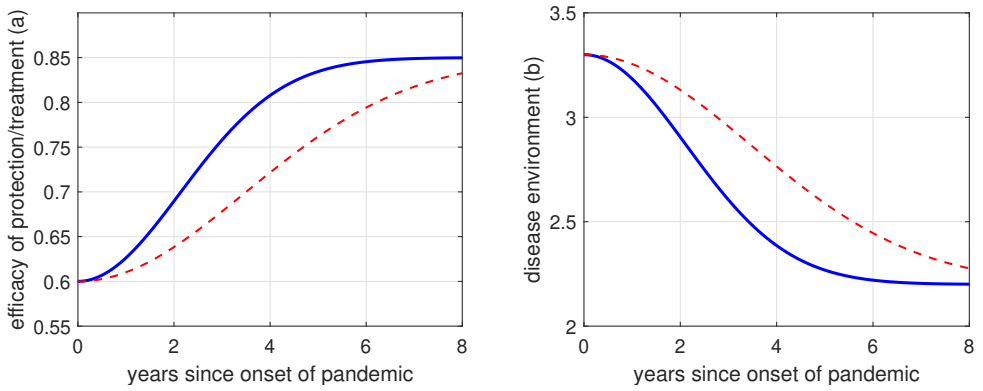
where x is the number of years since the onset of the pandemic. We assume that the disease environment evolves inversely proportional to medical technology $b(x) = b_1(1 - a(x)) + b_2$. The parameter ζ determines the half-life of the pandemic (i.e. the time at which half of the transition to the endemic state is reached). The parameters b_1 and b_2 are then determined by the targeted initial and final state of the transition, $b_1 \equiv (b(0) - b(\infty)/(a(\infty) - a(0))$ and $b_2 = b(0) - b_1(1 - a(0))$. We calibrate ζ such that the model prediction for the year 2022 provides the best fit of the actual infectious disease mortality rates in 2022. This leads to the estimate $\zeta = 3.0$, which implies a half-life of the pandemic of 2.5 years. The calibrated course of the pandemic is shown by blue solid lines in Figure 5. Red dashed lines show an alternative scenario with a half-life of the pandemic of 4 years, which we use for sensitivity analysis.

The model's prediction of age-specific mortality rates for 2022 is shown by the dashed line in Figure 4 and the underlying data for 2022 are shown by stars. The Covid mortality rate is again taken from CDC (2023b) and computed as described above. A halftime of 2.5 years seems to capture the actual age distribution of infectious disease mortality in 2022 quite well.

5. Results

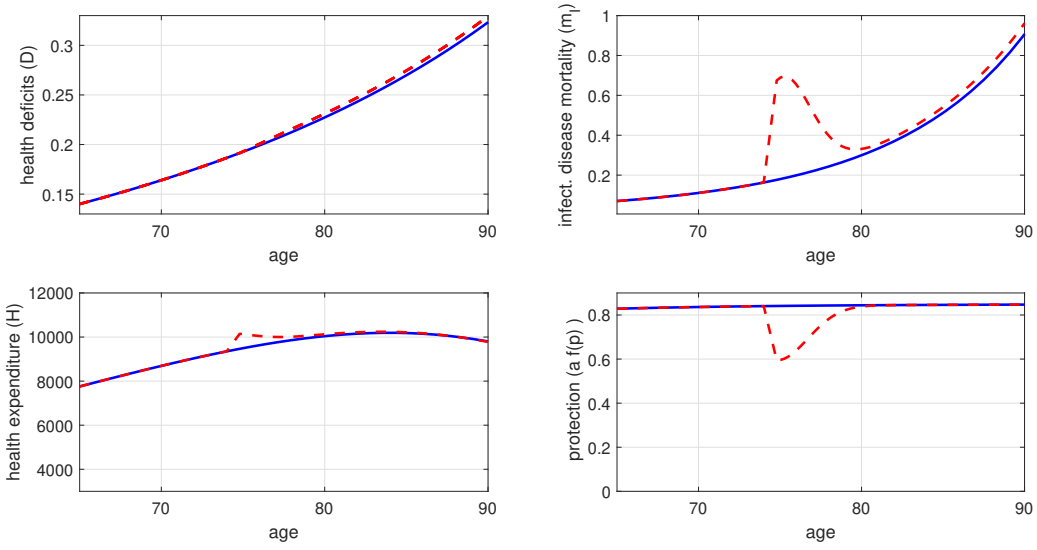
5.1. **Basic Model.** Figure 6 shows the health outcomes of the benchmark person without the pandemic (blue lines) and in the event that the pandemic occurs unexpectedly at the age of 75 (red lines). At this age, the individual has already developed many health deficits (pre-conditions) such that the probability of severe infection and the infectious mortality increases markedly. Although the individual increases protective efforts in response to the disease, actual protection decreases due to initially low protective technology. Also health expenditure rises above benchmark level due to the higher incentive to stay in good health during the pandemic. Figure 6 also shows a small increase of chronic diseases due to the feedback effect from inflammaging.

FIGURE 5. Course of the Pandemic



The figure shows the calibrated transitional dynamics of infectious disease protection technology a and the disease environment b during the Covid-19 pandemic. Solid blue lines: benchmark run (halftime of 2.5 years). Red dashed lines: alternative scenario: 4 years halftime of the pandemic.

FIGURE 6. Predictions without Covid-19 and with Covid-Shock at Age 75



Blue solid lines: model predictions without Covid-19 pandemic). Red dashed lines: predictions when Covid-19 pandemic starts at age 75; halftime 2.5 years. $H = \pi_p p + \pi_h h$.

The main results from comparative dynamic analysis are reported in Table 1 in form of simple summary statistics. For each case we show results when the considered individual that has been hit by the unanticipated pandemic is compared with (i) the own self without the pandemic and (ii) the benchmark American experiencing in the pandemic at the same age. ΔLE shows the difference in remaining life expectancy from the moment when the pandemic shock occurs. Δm_I^{s+1} is the percentage point deviation of the infectious disease mortality rate one year after onset of the pandemic and Δm_C^{s+10} is the percentage point deviation of the chronic disease

mortality rate ten years after onset of the pandemic, i.e. at the new endemic state of the disease environment. $(\Delta D/D)^{+10}$ is the relative change of chronic health deficits ten years after the onset of the pandemic; $\Delta\tilde{h}/\tilde{h}$ is the relative change in remaining lifetime health expenditure $\tilde{h}(\tau) \equiv \int_\tau^T S(t)H(t)\mathrm{d}t$ (with $H = \pi_h h + \pi_p p$), when the pandemic hits at age τ.

Case 1 of Table 1 shows health statistics for the benchmark individual considered in Figure 6. According to the first line, the pandemic is predicted to increase the mortality rate from infections one year after its outbreak at age 75 by 0.52 percentage points, i.e. m_I increases more than fourfold compared to the pre-Covid year (see Figure 6). The increased exposure to infections leads to faster aging. Ten years later, the individual has developed 2 percent more health deficits and chronic disease mortality rate is 0.55 percentage points higher than without the pandemic. The pandemic reduces life expectancy by about 5 months (0.38 years) for the benchmark individual. Given the sharp increase in infectious disease mortality, the change in life expectancy may seem small in absolute terms. It reflects the easily underestimated fact that even in a pandemic the vast majority of older people die from chronic diseases (see Goldstein and Lee, 2020, for an extensive discussion of this feature). Expected health investments $\tilde{h}$ decline somewhat after the pandemic. Since we have already seen in Figure 6 that health investments H by age increase in response to the pandemic, the result implies that decreasing survival probability (S) dominates increasing expenditure (H) in their contribution to the aggregate outcome. Naturally, the summary statistics are all zero when the benchmark individual is compared with itself (second line of case 1).

With cases 2 and 3 we explore the role of initial health deficits that develop over time until the pandemic hits (pre-existing conditions). Again, the first lines of each case show the isolated effect of the pandemic. Individuals endowed with 25 percent more initial health deficits are less healthy when the pandemic arrives and thus suffer an increase of infectious disease mortality that is about twice as high as for the benchmark individual. Compared to their own non-pandemic selves, the life expectancy loss is only slightly higher than in the benchmark case ($\Delta LE = -0.47$ vs. -0.38 years) due to the anyway shorter life expectancy. Cross-individual comparisons of lifetime outcomes, shown in the second line, however, reveal that chronic health deficits ten years after the pandemic shock are 35 percent higher and the loss of life expectancy is 4 years greater. These outcomes are a manifestation of the self-productivity of health deficits and the interaction between health deficits and the severity of infectious diseases. In case 3, we consider individuals who are endowed with 25 percent fewer initial health deficits, thus being substantially healthier when the pandemic arrives at age 75. The pandemic causes infectious disease mortality to increase by less than for the benchmark individual such that the increase in chronic disease mortality ten years later is considerably lower. Good health conditions have protected the individual not only from the direct effect of the Covid pandemic on infectious disease mortality but also from its indirect effects through inflammaging. Life expectancy is almost 5 years higher than for the benchmark American.

Case 4 examines the role of strong protection aversion by setting $\omega = 2$ (cf. the red lines in Figure 2). This individual experiences a considerably higher increase of infectious disease mortality by 0.9 percentage points due to the pandemic one year after its onset and consequently

Table 1: Health and Longevity with Covid-19 Pandemic: Comparative Dynamics

case	parameter change	remark	comparison	ΔLE	Δm_I^{s+1}	Δm_C^{s+10}	$(\Delta D/D)^{s+10}$	$\Delta\tilde{h}/\tilde{h}$
pandemic onset at age 75								
1)		benchmark	own without pan.	-0.38	0.52	0.55	2.00	-1.31
			bench with pan.	0.00	0.00	0.00	0.00	0.00
2)	$D_0 = 1.25D_{0,b}$	25% less healthy	own without pan.	-0.47	1.18	1.76	2.85	-2.71
			bench with pan.	-3.93	0.90	13.20	34.70	-33.16
3)	$D_0 = D_{0,b}/1.25$	25% healthier	own without pan.	-0.28	0.24	0.09	0.67	-0.67
			bench with pan.	4.84	-0.39	-5.40	-23.81	14.23
4)	$\omega = 2.0$	more protection averse	own without pan.	-0.36	0.89	2.15	3.25	-2.16
			bench with pan.	-4.08	1.47	14.99	38.31	-72.22
5)	$w = 1/2w_b$	50% poorer	own without pan.	-0.40	0.64	0.69	2.05	-1.54
			bench with pan.	-1.11	0.16	2.33	7.67	-63.52
6)	$w = 1/2w_b$, $\omega = 2.0$	poorer and 4)	own without pan.	-0.37	1.19	3.75	4.09	-2.70
			bench with pan.	-5.02	2.04	25.24	56.29	-90.84
7)	$w = 2w_b$	100% richer	own without pan.	-0.35	0.36	0.20	0.93	-1.10
			bench with pan.	1.80	-0.20	-2.22	-8.44	199.50
8)	$w = 2w_b$, $\omega = 2.0$	richer and 4)	own without pan.	-0.33	0.65	1.12	2.54	-1.67
			bench with pan.	-2.53	0.93	6.39	19.09	9.78
9)	$\beta = 0$	no inflammaging	own without pan.	-0.12	0.42	0.00	0.00	0.03
			bench with pan.	1.67	-0.09	-2.26	-8.63	45.96
10)	$\zeta = 4.9$	pandemic half-life 4 years	own without pan.	-0.67	0.55	0.95	3.39	-2.15
			bench with pan.	0.00	0.00	0.00	0.00	0.00
pandemic onset at age 60								
11)		benchmark	own without pan.	-0.20	0.11	0.03	0.44	-0.39
			bench with pan.	0.00	0.00	0.00	0.00	0.00
12)	$D_0 = 1.25D_{0,b}$	25% less healthy	own without pan.	-0.28	0.19	0.09	0.64	-0.77
			bench with pan.	-5.44	0.13	2.45	27.98	-27.56
13)	$\omega = 2.0$	more protection averse	own without pan.	-0.21	0.15	0.05	0.57	-0.66
			bench with pan.	-4.61	0.30	0.89	11.69	-49.49
14)	$D_0 = 1.25D_{0,b}$, $\omega = 2.0$	25% less healthy and 13)	own without pan.	-0.25	0.29	0.37	1.55	-1.20
			bench with pan.	-9.81	0.90	6.30	57.35	-63.93
15)	$w = 1/2w_b$, $\omega = 2.0$	poorer and 13)	own without pan.	-0.23	0.17	0.07	0.63	-0.74
			bench with pan.	-5.91	0.38	1.56	19.20	-82.12

The table shows the predicted deviations of health behavior and health outcomes from (i) the outcomes for the same individual without pandemic shock and (ii) the benchmark individual with pandemic shock. All entries are measured from shock-age onwards; w_b is wage income of the benchmark individual; $D_{0,b}$ is the initial health deficit state (at age 20) of the benchmark individual; ΔLE is the change in life expectancy at shock age; Δm_I^{s+1} is the percentage point deviation of the infectious disease mortality rate one year after onset of the pandemic; Δm_C^{s+10} is the percentage point deviation of the chronic disease mortality rate ten years after onset of the pandemic; $(\Delta D/D)^{+10}$ is the relative change of chronic health deficits ten years after the onset of the pandemic; $\Delta\tilde{h}/\tilde{h}$ is the relative change in expected lifetime health expenditure. Relative deviations are shown in percent.

a large increase of chronic disease mortality rate ten years later (more than 2 percentage points). The loss in life expectancy, however, is similar to the benchmark American. This perhaps surprising outcome is explained by the fact that the protection-averse individual has anyway a considerably shorter life expectancy. Compared to the benchmark individual who has low protection aversion (second line of case 4), however, the protection-averse individual is predicted to lose about 4 more years of life. At age 85, the individual has accumulated 38 percent more health deficits and faces a 15 percentage point higher mortality rate. In other words, the vast majority of protection averse individuals is already dead before age 85.

Cases 5 and 6 show that poor individuals, when compared to their non-pandemic selves, are similarly affected by the pandemic as the benchmark individual in terms of life expectancy and health deficits ten years after the pandemic shock. In cross-comparison with the benchmark

(second lines), however, their life expectancy is about one year shorter. The differential effect is explained by lower health investments of poorer individuals (last column in Table 1). An important conclusion is that, with and without pandemics, the socioeconomic health gradient is particularly high if earnings are negatively correlated with the extent of protection aversion.

Cases 7 and 8 again suggest that income does not play a major role in the direct health impact of the pandemic, but is generally important for mortality risk and morbidity through its impact on health care spending. A person with twice as much income as the reference person can expect to live almost two years longer (second line of case 7). Compared to a person with half the median earnings (case 5), life expectancy is almost 6 years longer. Protection aversion has less severe consequences for rich people because they can partly compensate for this through higher health care spending. A strongly protection averse individual with twice the median earnings is expected to live "only" 2.5 years shorter than the benchmark individual.

With case 9 in Table 1, we eliminate inflammaging ($\beta = 0$) in order to understand the importance of the feedback channel from infectious diseases to chronic health deficits. In this counterfactual experiment, there is by design no effect of the pandemic on Δm_C^{s+10} and $(\Delta D/D)^{s+10}$ (first line). As a result, the pandemic has a much smaller effect on life expectancy (loss of about six weeks). This means that inflammaging more than triples the impact of the pandemic on health outcomes. The second line of case 9 measures the lifetime effects of inflammaging. If there were no inflammaging, the benchmark individual would have 8 percent less health deficits at age 85 and die 1.6 years later. These results highlight the detrimental health effects of chronic inflammation that develops from infections.

Case 10 shows that all health outcomes are more severely affected when the pandemic half-life is 4 years (instead of 2.5). Since the elevated disease mortality is experienced for longer, the inflammaging effect is stronger and chronic disease mortality ten years later is higher. The pandemic shortens life expectancy for the benchmark American by 8 months (0.67 instead of 0.38 years).

The second part of Table 1 considers individuals experiencing the pandemic at age 60. While the qualitative results are the same as before, the short- and long-term effects of the pandemic are more muted due to the generally better health (compared to health at age 75); e.g. compare the first line of case 11 with case 1 ($\Delta LE = -0.2$ years instead -0.38).

A comparison of case 12 with case 3 highlights that the impact of the pandemic depends on pre-pandemic health status rather than chronological age. The initially less healthy individual in case 12 has accumulated about the same level of health deficits by age 60 as the initially healthier individual in case 3 by age 75. Consequently, both suffer the same loss of life expectancy due to the pandemic ($\Delta LE = -0.28$ years), which is explained by about the same increase of chronic health deficits ten years later. Interestingly, looking at the second line, we see that the loss in life expectancy is larger when the pandemic hits at age 60 ($\Delta LE = -5.44$ years in case 12) than when its hits at age 75 ($\Delta LE = -3.93$ years in case 3). Also protection aversion has a larger effect if the individual experiences the pandemic earlier in life (compare case 13 with case 4) despite the considerably smaller effect of protection aversion on infection mortality in

younger people. These results reflect the interaction between infections and the self-productive development of chronic health deficits.

Cases 13 and 14 show that strongly protection averse individuals display considerably higher infectious disease mortality one year after onset of the pandemic and considerably higher chronic disease mortality ten years after the onset of the pandemic. Compared to their non-pandemic selves, the pandemic does not seem to play a major role in life expectancy, an outcome which is again explained by the anyway unhealthier and shorter life of protection averse individuals. We also see that protection aversion is particularly health-damaging for individuals with pre-conditions. Bad luck and bad behavior are almost additive in their impact on life expectancy. As discussed in case 12 the individual loses 5.4 years due to poorer initial health. According to cases 13 and 14 the individual loses 4.6 years due to poorer protection and 9.8 years when both risk factors are combined.

Finally, case 15 sheds further light on the socioeconomic health gradient. Whereas the loss in life expectancy is similar to that of benchmark individual when compared to their non-pandemic selves, poor persons with protection aversion lose almost 6 years of life expectancy. Moreover, a comparison with case 6 shows that almost an additional year of life is lost if the pandemic occurs at age 60 instead of age 75. Again, the self-productive accumulation of chronic health deficits explains why relatively young individuals suffer more from the pandemic in terms of long-term health, especially when they are poor and protection averse.

5.2. **False Beliefs.** The outbreak of the Covid pandemic has confronted humanity with a new disease and it may seem implausible to assume that everyone was fully informed about the severity of the pandemic and the medical protection measures. In addition, a significant fraction of the population held beliefs that were very different from those of official health organizations. Empirical evidence suggests that such divergent beliefs are related to socioeconomic status. For instance, van Mulukom et al. (2022) provide a systematic literature review suggesting that "individuals with lower income tend to hold stronger beliefs in COVID-19 conspiracy theories" (p. 5). This has greatly affected protection behavior. Similarly, vaccine hesitancy strongly correlates with beliefs about vaccine efficacy (e.g. Baeza-Rivera, 2021; Lazarus et al., 2022).

Formally, individuals believe that the disease environment is b_B and the medical technology is a_B. Replacing, a and b in the first-order and co-state equations (Lemma 1) leads to the solution:

$$p = \max\left\{0, -\log(z)\right\}, \qquad \text{with } z \equiv \frac{\omega + \phi_p \pi_p c^{-\sigma}}{a_B b_B D^{\nu}\left[\lambda_S + \frac{\beta \phi_h \pi_h c^{-\sigma}}{A(1+\eta D)^{\delta}\gamma h^{\gamma-1}}\right]} \tag{13}$$

$$\frac{\dot{h}}{h} = \frac{1}{1-\gamma}\left[r + \theta m - \rho + \frac{\delta \eta \dot{D}}{1+\eta D} + \frac{\dot{\lambda}_D}{\lambda_D}\right] \qquad \text{with} \tag{14}$$

$$\begin{aligned}\frac{\dot{\lambda}_D}{\lambda_D} = {} & \mu - \rho - \lambda_S \frac{\mu A (1+\eta D)^{\delta}\gamma c^{\sigma}}{\phi_h \pi_h h^{1-\gamma}}\left[\xi\psi D^{\psi-1} + (1 - a_B f(p)) b_B \nu D^{\nu-1}\right] \\ & + \mu\eta\delta A (1+\eta D)^{\delta-1} h^{\gamma} - \mu\beta(1 - a_B f(p)) b_B \nu D^{\nu-1}\end{aligned} \tag{15}$$

$$\dot{\lambda}_S = \lambda_S\left[\rho + \xi D^{\psi} + (1 - a_B f(p)) b_B D^{\nu}\right] - \left[u(c) - \omega p\right]. \tag{16}$$

The rest of the model remains as specified above. Importantly, the actual law of motion for survival probability (2) and health deficits (5) are still based on the actual values of a and b.

In the experiments reported in Table 2 we focus on permanently false beliefs (e.g. capturing the convinced Covid denier).[7] Table 2 has a similar structure to Table 1, whereby we focus on a pandemic shock at the age of 60. In all cases, the individual believes that infections are almost harmless ($b_B = 0.05$) and protection almost useless ($a_B = 0.05$).[8]

Case 1 of Table 2 considers the benchmark individual with false beliefs. We see that such individuals experience a substantially higher infectious disease mortality at the onset of the pandemic ($\Delta m_I = 0.95$ compared to $\Delta m_I = 0.11$ in case 11 of Table 1). However, compared to their non-pandemic selves, they do not seem to suffer much from the pandemic in terms of long-term health outcomes. This observation, which also holds for the other cases (first lines in Table 2) is again explained by the fact that the pandemic is only a short episode in the lifetime of individuals and individuals with false beliefs are not protecting themselves from infections over the whole life cycle. The second lines in Table 2 reveal that false beliefs have a quite dramatic impact on 60-year olds when we compare the life outcomes with those of the benchmark person with correct beliefs. For case 1, we find that false beliefs lead to a 6.3 years shorter life expectancy and 19 percent more health deficits by age 70.

Case 2 shows that more than an additional year is lost if individuals with false beliefs have only half of benchmark income (ΔLE=-7.55). The socioeconomic health gradient is therefore particularly large if low earnings are correlated with false beliefs.

According to cases 3 and 4, protection aversion increases health disparities, albeit its differential effect is small compared to Table 1. The reason for this is that false beliefs alone already reduce protection efforts to a large extent.

Cases 5 and 6 demonstrate that the health impacts of false beliefs are particularly strong for unhealthy individuals. The interaction of anyway bad initial health and inflammaging is amplified by insufficient protection. Consequently, in case 5, the frailty index at age 70 is 83 percent above benchmark and life is more than twelve years shorter. If, in addition, the unhealthier individual has also half of benchmark income, the life expectancy difference to the benchmark individual rises to 13.2 years (case 6).

5.3. **Long Covid.** So far, our analysis assumed that the Covid pandemic influenced individual health solely through an increase in the disease environment and a temporary reduction in protection efficacy. In other words, for most people the Covid pandemic operated mainly as 'just

[7]Effects would be less pronounced if initially ill-informed rational individuals updated beliefs. They would be more pronounced if individuals assumed that also parameter β (measuring the inflammaging effect of infectious diseases) is lower than the actual value, in addition to false beliefs about of a and b.

[8]We omit the cases of belief in absolutely harmless infections ($b_B = 0$) and absolutely useless protection ($a_B = 0$) for computational reasons. We also abstain analyzing the effects of false beliefs of a and b separately, as beliefs in inferior protection technology and harmless infections are correlated. We checked though, that the magnitude of the effects on e.g. life expectancy in Table 2 are mainly driven by the false belief $b_B = 0 < b$. Noteworthy, a false belief $a_B = 0 < a$ in isolation gives an incentive to invest in health (i.e. health input h is decreasing in a_B while protection effort p is decreasing). This is because the perceived effect of higher frailty on infectious disease mortality risk is higher, the lower the perceived efficacy of the protection technology, according to eq. (4).

Table 2: False Beliefs: Comparative Dynamics

case	parameter change	remark	comparison	ΔLE	Δm_I^{s+1}	Δm_C^{s+10}	$(\Delta D/D)^{s+10}$	$\Delta\tilde{h}/\tilde{h}$
pandemic onset at age 60								
1)			own without pan.	-0.20	0.95	0.06	0.54	-0.85
			bench with pan.	-6.29	0.85	1.54	18.89	-82.45
2)	$w = 1/2w_b$	poor individual	own without pan.	-0.22	1.06	0.08	0.60	-1.09
			bench with pan.	-7.55	0.96	2.28	26.34	-94.16
3)	$\omega = 2.0$	protection averse	own without pan.	-0.20	0.95	0.06	0.54	-0.85
			bench with pan.	-6.57	0.85	1.51	18.52	-83.85
4)	$\omega = 2.0$, $w = 1/2w_b$	prot. averse and poor	own without pan.	-0.22	1.06	0.08	0.60	-1.09
			bench with pan.	-7.83	0.97	2.26	25.95	-94.62
5)	$D_0 = 1.25D_{0,b}$	less healthy	own without pan.	-0.30	2.49	0.68	1.88	-1.78
			bench with pan.	-12.30	2.40	10.95	82.71	-90.23
6)	$D_0 = 1.25D_{0,b}$, $w = 1/2w_b$	less healthy and poor	own without pan.	-0.31	2.88	1.34	3.00	-2.34
			bench with pan.	-13.19	2.80	14.43	98.36	-96.85

The table shows results for individuals believing that infections are almost harmless ($b_B = 0.05$) and protection is almost useless ($a_B = 0.05$). Predicted deviations of health behavior and health outcomes are shown in comparison to (i) the outcomes for the same individual without pandemic shock and (ii) the benchmark individual with correct believes under the pandemic shock. All entries are measured from shock-age onwards; w_b is the wage income of the benchmark individual; $D_{0,b}$ is the initial health deficit state (at age 20) of the benchmark individual; ΔLE is the change in life expectancy at shock age; Δm_I^{s+1} is the percentage point deviation of the infectious disease mortality rate one year after onset of the pandemic; Δm_C^{s+10} is the percentage point deviation of the chronic disease mortality rate ten years after onset of the pandemic; $(\Delta D/D)^{s+10}$ is the relative change of chronic health deficits ten years after the onset of the pandemic; $\Delta\tilde{h}/\tilde{h}$ is the relative change in expected lifetime health expenditure. Relative deviations are shown in percent.

as another influenza'. A sizable minority of the population, however, responded to infections with a previously unseen severe accumulation of chronic health deficits. This response is best known as Long Covid syndrome rather than under its medical term post-acute sequelae of Covid-19 (PASC).

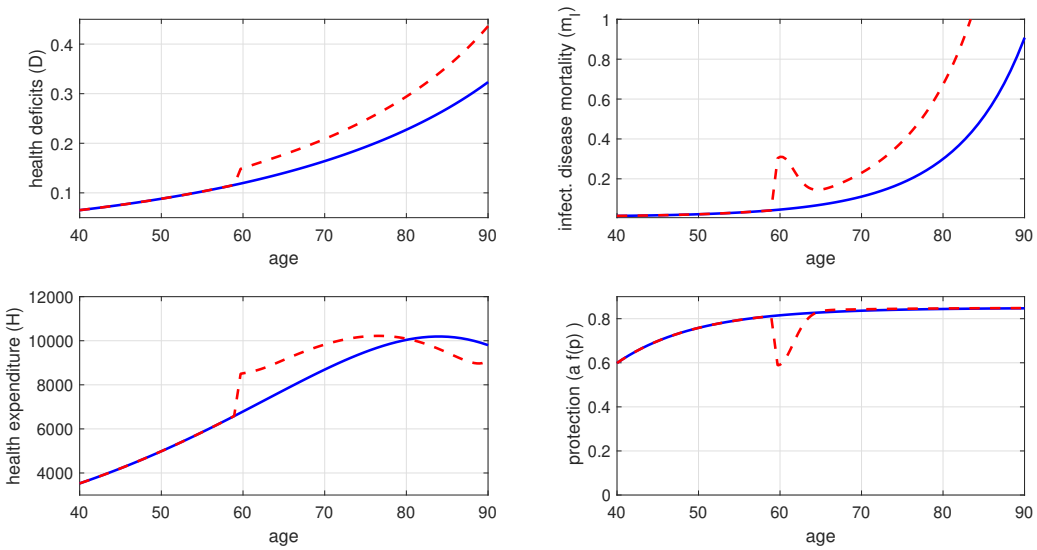
Long Covid is characterized by a large number of symptoms such as extreme fatigue, memory problems, headaches, sleep problems, depression, digestive symptoms, and a long list of pulmonary, cardiovascular, and musculoskeletal illnesses. A recent cohort study considering 80 potential health deficits found that Long Covid affected basically all human organ systems (Bowe et al., 2023). While hospitalized patients faced a substantially higher risk of Long Covid, it was shown that the disease affected also about 7 percent of the non-hospitalized population. The study by Bowe et al. is the first large-scale cohort study to follow individuals for two years post-infection. It demonstrated a high persistence of the Long Covid attributed health deficits. Although a two-year observation period is too short to draw firm conclusions, we take the available evidence of persistence as motivation to model Long Covid as a permanent, lifelong deterioration in chronic health caused by Covid infection.[9]

The health deficit model is ideally suited to implement Long Covid in health economic theory. Specifically, we assume that for Long Covid patients, a Covid infection triggered a spontaneous

[9] A limitation of the study is that it focuses on a population of elderly men (U.S. veterans of average age 61, standard deviation 16 years). We are not aware of other long-run cohort studies that report the universe of health deficits from Covid infections for other population groups.

and permanent increase of the health deficits index in the year after the infection. This black-box treatment of Long Covid seems appropriate as long as the actual causes of the disease are not fully understood. We used the data provided in Bowe et al. (2023, Figure 1 and Supplement Table 12), constructed a frailty index of 80 health deficits, and computed the relative increase frailty index of the infected group vs. the non-infected control group. We found that for [0.5, 1, 1.5, 2] years after infection the frailty is by factor [1.62, 1.29, 1.23, 1.25] higher in the infected group. After an initial decline, the frailty index is thus permanently around 25 percent above the non-pandemic level.

FIGURE 7. Long-Covid-Shock at Age 60



Blue solid lines: benchmark model predictions (no Covid pandemic). Red dashed lines: Long Covid; pandemic starts at age 60; halftime 2.5 years.

The red (dashed) lines in Figure 7 show the life cycle trajectories when getting Long Covid (i.e. health deficits increase by 25 percent) at age 60 at the onset of the pandemic with a half-life of 2.5 years. Blue lines show the unshocked benchmark case. The most remarkable result is the prediction that the deviation of health deficits from benchmark widens after the end of the pandemic. This sad result is an expression of the self-productivity of health deficits implied by the deficit-accumulation process (5) discussed in sections 1 and 2. Applied to Long Covid, health deficits that have emerged as symptoms of Long Covid (e.g. fatigue, headaches, stomach and muscle pain or shortness of breath) lead to the earlier onset of other age-related health deficits (cardiovascular or cognitive diseases) via direct biological pathways or through the inability to exercise or cognitive activity.

The results of our comparative dynamics analysis are shown in Table 3, which is again constructed as Table 1 and 2 with the difference that the second line of each case now compares with a benchmark individual who is also suffering from Long Covid (LC). When getting Long Covid at age 75, the benchmark individual can expect a 4 years shorter life (case 1 of Table 3, first line). The life expectancy loss caused by the pandemic is 10 times higher than without

Table 3: Health and Longevity with Long Covid: Comparative Dynamics

case	parameter	remark	comparison	ΔLE	Δm_I^{s+1}	Δm_C^{s+10}	$(\Delta D/D)^{s+10}$	$\Delta\tilde{h}/\tilde{h}$
			Long Covid at Age 75					
1)		benchmark	own without LC	-4.00	1.19	11.51	32.31	-22.41
			bench with LC	-0.00	0.00	-0.00	-0.00	-0.00
2)	$\omega = 2.0$	protection aversion	own without LC	-2.82	2.65	10.52	14.41	-28.24
			bench with LC	-3.00	2.55	12.30	17.77	-69.73
			Long Covid at Age 60					
3)		benchmark	own without LC	-5.53	0.27	2.35	27.32	-14.79
			bench with LC.	-0.00	0.00	-0.00	-0.00	-0.00
4)	$\omega = 2.0$	protection aversion	own without LC	-4.57	0.64	3.57	29.93	-18.69
			bench with LC	-3.53	0.63	2.09	13.79	-47.07
			Long Covid at Age 45					
5)		benchmark	own without LC	-6.58	0.05	0.58	25.28	-9.10
			bench with LC.	-0.00	0.00	-0.00	-0.00	-0.00
6)	$\omega = 2.0$	protection aversion	own without LC	-6.26	0.12	0.70	28.78	-13.42
			bench with LC	-4.62	0.12	0.14	3.98	-33.99
7)	$w_{\|age>45} = w_R$	LC-induced retirement	own without LC	-6.92	0.05	0.58	25.39	-52.09
			bench with LC	-0.34	0.00	0.00	0.09	-47.29

The table shows the predicted deviations of health behavior and health outcomes from (i) the outcomes for the same individual without pandemic shock and (ii) the benchmark individual with pandemic shock. All entries are measured from shock-age onwards. The index *bench* identifies the calibrated benchmark value of initial health deficits; ΔLE is the change in life expectancy at shock age; Δm_I^{s+1} is the percentage point deviation of the infectious disease mortality rate one year after onset of the pandemic; Δm_C^{s+10} is the percentage point deviation of the chronic disease mortality rate ten years after onset of the pandemic; $(\Delta D/D)^{+10}$ is the relative change of chronic health deficits ten years after the onset of the pandemic; $\Delta\tilde{h}/\tilde{h}$ is the relative change in expected lifetime health expenditure. Relative deviations are shown in percent.

getting Long Covid and the increase in infectious disease mortality at the onset of the pandemic is more than twice as high (cf. case 1 of Table 1). Ten years later, the initially 25 percent increase of health deficits has widened to 32 percent, leading to an increase in mortality from chronic diseases that is 11 percentage points higher.

Protection-averse individuals lose "only" 2.8 years of life due to Long Covid compared to their non-pandemic selves (case 2, first line). Again, the result is explained by the fact that life expectancy at 75 is anyway short with protection aversion. Cross-comparison with the benchmark individual shows that protection-averse individuals lose 3 years (case 2, second line). Recalling from case 4 of Table 1 that the protection-averse individual without Long Covid can expect to live 4 years less in a pandemic, the combined loss of life expectancy from protection aversion and Long Covid is therefore 7 years.

The most remarkable result in Table 3 is that, in contrast to the 'normal' Covid infection from Table 1, the impact of the pandemic is *greater* when it occurs at younger age. The reason is again the cumulative and progressive nature of health deficit accumulation. If Long Covid occurs at a young age, the self-productive nature of health deficit accumulation has more time to unfold. The life expectancy loss for the only mildly protection averse benchmark individual caused by the pandemic is 5.5 years when getting Long Covid at age 60 (case 3, first line), compared to 4 years when the pandemic starts at age 75, and 6.6 years when it occurs at age 45 (case 5, first line). Protection aversion of Long Covid patients leads to a 6 months higher loss in life expectancy when the pandemic starts at age 60 rather than age 75 (3.5 vs. 3.0 years; see

case 4, second line) and the loss is 1.6 years higher when the individual gets Long Covid already at age 45 (case 6, second line).

Finally, in case 7, we assume that getting Long Covid causes early retirement (w declines to w_R at age 45 when the pandemic shock occurs, i.e. 47 percent of earnings during work life). The income loss reduces health expenditures. Consequently, the pandemic causes for individuals getting Long Covid at age 45 a loss in life expectancy of 6.9 years rather than 6.6 years without early retirement (first line of case 7). The income loss also causes an about 50 percent decline of health investments for Long Covid patients, which leads to a further longevity loss of about 4 months (second line).

6. Conclusion

In this paper, we have examined short-run and long-term consequences of the Covid pandemic for biological aging and infectious and all-cause mortality. Additionally, we highlighted socioeconomic disparities in health outcomes associated with health behaviors and initial health conditions. Our calibrated model is empirically founded in modern medical research and reflects (i) the self-productive nature of health deficit accumulation and (ii) the interaction between infections and chronic health deficits.

We have produced two sets of results. First, we compared individuals of different types with their own non-pandemic selves. These counterfactual computational experiments identified the individual health impact of the pandemic. Second, we computed the lifelong health effects relative to the benchmark individual, conditional on income, initial health deficits, protection aversion, and beliefs.

We have shown that the interaction of infections and chronic diseases implies that the predicted long-term health consequences of the pandemic for the average American are about three times larger than the direct health effects (that ignore the inflammaging channel). While the within-individual effects of the pandemic are relatively similar for all considered individuals we found large differences in lifetime health outcome across individuals. We have shown that socioeconomic differences in morbidity and life expectancy are strongly influenced by attitudes and misconceptions that lead to differential disease protection behavior. Protection aversion and false beliefs about the health risks of infection or the usefulness of protective measures are particularly harmful to low-income individuals because they have fewer resources to counteract the consequences of their behavior through increased investments in chronic disease prevention and repair. Because lower-income individuals have been shown to be, on average, more protection-averse and prone to false beliefs, our results suggest that the pandemic has caused and will continue to cause major socioeconomic disparities in chronic health conditions.

Furthermore, and somewhat surprisingly at first glance, we found that the health inequalities explained by the model between people with different levels of protection aversion, initial health deficits or income are more pronounced in middle-aged people at the time of the pandemic shock than in older people. Likewise, we have shown that the loss of health and life expectancy due to Long Covid, which is significant in all age groups, is even greater if the disease has occurred at a relatively young age. The reason for these results is the self-productivity of health deficits,

which means that a health shock entails a greater accumulation of further health deficits when it is experienced earlier in life.

Finally, our analysis suggests that the pandemic-induced recession of 2020-2021 was likely not the greatest economic cost of the Covid pandemic. The greatest cost may be experienced in the long-run through the permanent loss of productivity induced by the premature withdrawal of the Long Covid patients from the labor market and the future challenges to health insurance systems due to the faster development of chronic health deficits in the general population.

However, it should be noted that the future outlook could be less bleak for several reasons. The sample population of the currently available long-term studies of Long Covid was exposed to infection until the end of the year 2020, i.e. before vaccination became widely available. Other recent studies found that vaccination significantly reduces the risk of developing Long Covid (Byambasuren et al., 2023) as well as the average frailty increase in Long Covid patients (Al-Aly et al., 2022). Furthermore, the future may bring effective treatment for Long Covid patients, or some manifestations of Long Covid symptoms may prove to be less persistent than thought today.

Appendix A: Proof of Lemma 1

To prove Lemma 1, i.e. the solution of the life cycle problem, we first use (1)–(6) to write the current-value Hamiltonian as:

$$\mathcal{H} = S\left[u(c) - \omega p\right] + \lambda_k\left[w + (r+\theta m)k - c - \phi_h\pi_h h - \phi_p\pi_p p\right] + \lambda_D\mu\left[D - A(1+\eta D)^{\delta}h^{\gamma} + \beta(1-af(p))bD^{\nu} - \epsilon\right] - \lambda_S S\left[\xi D^{\psi} + (1-af(p))bD^{\nu}\right], \quad \text{(A.1)}$$

where λ_S, λ_D, and λ_k are co-state variables associated with differential equations (2), (5), and (6), respectively.

Using $u(c) = \frac{c^{1-\sigma}-1}{1-\sigma}$ and $f(p) = 1 - \mathrm{e}^{-p}$, the first-order conditions for controls c, h, p and co-state equations for state variables k, D, S are:

$$\left[\frac{\partial\mathcal{H}}{\partial c} = 0 \Leftrightarrow\right] Sc^{-\sigma} = \lambda_k \quad \text{(A.2)}$$

$$\left[\frac{\partial\mathcal{H}}{\partial h} = 0 \Leftrightarrow\right] -\lambda_k\phi_h\pi_h = \lambda_D\mu A(1+\eta D)^{\delta}\gamma h^{\gamma-1} \Leftrightarrow -\lambda_D = \frac{\phi_h\pi_h}{\mu\gamma}\frac{\lambda_k h^{1-\gamma}}{A(1+\eta D)^{\delta}} > 0 \quad \text{(A.3)}$$

$$\left[\frac{\partial\mathcal{H}}{\partial p} =\right] -S\omega - \lambda_k\pi_p\phi_p - \lambda_D\mu\beta abD^{\nu}\mathrm{e}^{-p} + \lambda_S SabD^{\nu}\mathrm{e}^{-p} \leq 0 \quad \text{with} = \text{for } p > 0 \quad \text{(A.4)}$$

$$\left[\frac{\partial\mathcal{H}}{\partial k} =\right] \lambda_k(r+\theta m) = \lambda_k\rho - \dot{\lambda}_k \Leftrightarrow -\frac{\dot{\lambda}_k}{\lambda_k} = r + \theta m - \rho \quad \text{(A.5)}$$

$$\left[\frac{\partial\mathcal{H}}{\partial D} =\right] \lambda_D\mu - \lambda_D\mu\eta\delta(1+\eta D)^{\delta-1}Ah^{\gamma} + \lambda_D\mu\beta(1-af(p))b\nu D^{\nu-1} - \lambda_S S\left[\xi\psi D^{\psi-1} + (1-af(p))b\nu D^{\nu-1}\right] = \lambda_D\rho - \dot{\lambda}_D \quad \text{(A.6)}$$

$$\left[\frac{\partial\mathcal{H}}{\partial S} =\right] u(c) - \omega p - \lambda_S\left[\xi D^{\psi} + (1-af(p))bD^{\nu}\right] = \lambda_S\rho - \dot{\lambda}_S. \quad \text{(A.7)}$$

Inserting (A.2) and (A.3) in (A.4) we obtain the solution for infectious disease protection (7). From log-differentiating (A.2), substituting $\dot{S}/S = -m$ and using (A.5), we obtain the Euler equation (8). Log-differentiating (A.3) by noting that $-\lambda_D > 0$ and substituting (A.5) confirms the equation of motion for health investments to prevent and treat chronic diseases (9). Sorting terms in (A.6) implies

$$\frac{\dot{\lambda}_D}{\lambda_D} = \rho - \mu + \frac{\mu\eta\delta Ah^{\gamma}}{(1+\eta D)^{1-\delta}} - \mu\beta(1-af(p))b\nu D^{\nu-1} + \frac{\lambda_S S\left[\xi\psi D^{\psi-1} + (1-af(p))b\nu D^{\nu-1}\right]}{\lambda_D}. \quad \text{(A.8)}$$

Substituting λ_D from (A.3) and λ_k from (A.2) confirms the equation of motion for the shadow price of health deficits (10). Finally, sorting terms in (A.7) we obtain the law of motion for the shadow price of survival (11). This concludes the proof of Lemma 1.

Appendix B: Calibrating the Epidemiological Transition

Figures 2 and 3 replicate our calibration of the epidemiological transition (Strulik and Grossmann, 2024) for the model structure of the present paper. The results are very similar.

FIGURE A.1 The Epidemiological Transition

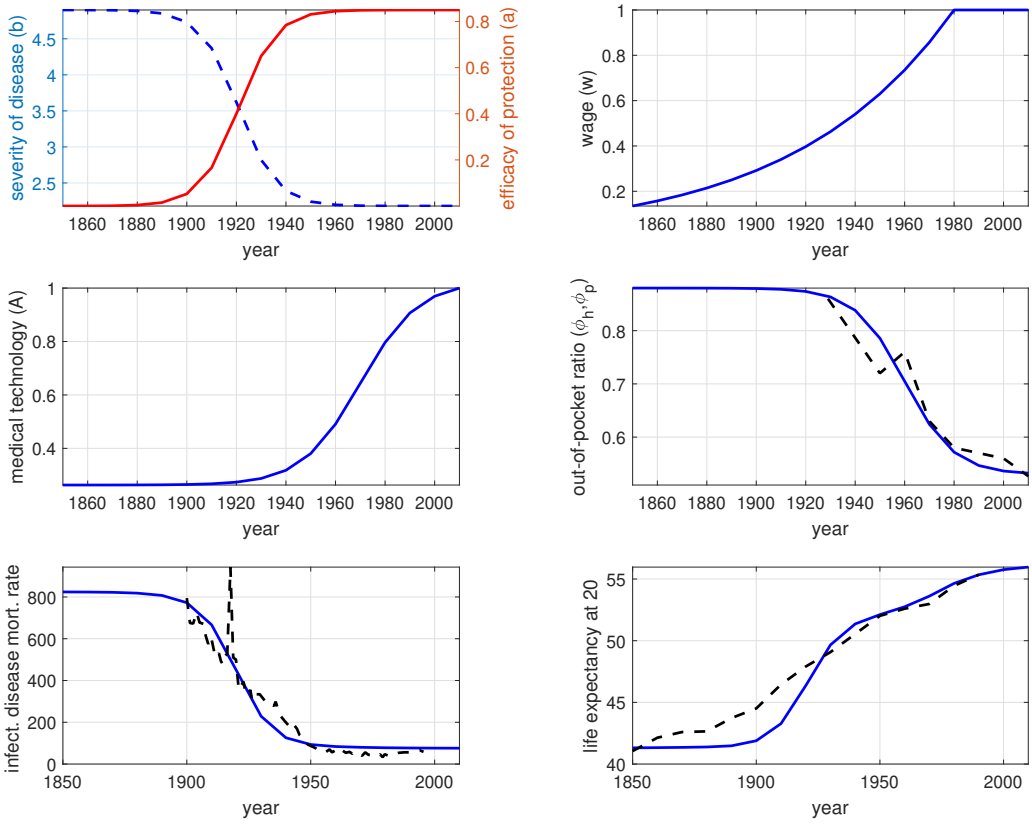


Table A.1: Parameter Values

Parameter	Meaning	Value
μ	natural rate of aging	0.034
A	medical technology (scale)	0.0008
γ	medical technology (curvature)	0.25
δ	medical technology (curvature)	1.6
η	medical technology (curvature)	5.1
β	inflammaging coefficient	7.5
ν	immunosenescence coefficient	3.2
b	infectious disease prevalence	2.2
a	infectious disease technology	0.85
ϵ	aging residual	-0.0004
ω	disutility from protection	0.03
σ	inverse of the IES	1.01
ρ	time preference rate	0.06
r	interest rate	0.06
w	wage income	27,928
ϕ_h,ϕ_p	coinsurance ratio	0.53
π_h	unit price health care (chronic)	1.88
π_p	unit price health care (infection)	115
ψ	survival function (chronic)	2.8
ξ	survival function (chronic)	3.8
θ	share annuitized wealth	1.0
$D(0)$	initial health deficits	0.036
$D(T)$	final health deficits	0.55
$k(0) = k(T)$	initial and final wealth	0.0

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