Life-Cycle Labor Supply and Physiological Aging across Countries*

Casper Worm Hansen†

Carl-Johan Dalgaard‡

Holger Strulik§

February 14, 2022

Abstract. We construct a cohort-based frailty index for 181 countries over the period 1990-2019. We use this macro measure of physiological aging to estimate the impact of deteriorating health on labor force participation. Our three-dimensional panel framework, in which the unit of observation is a cohort in a given country at a given age, allows us to control for a range of unobserved factors. Our identification strategy further exploits a compensating law of physiological aging to account for reverse causality. We find a strong negative effect of physiological aging on labor market participation: a one percent increase in the frailty index leads to a reduction of labor force participation of about 0.6 ($\pm$0.2) percentage points. Since health deficits are accumulated at a rate of about 3 percent per year of life, almost all of the age-related decline in labor force participation can be motivated by deteriorating health.

Keywords: Physiological Aging, Health, Life-Cycle Labor Supply.

JEL: I10, I15, J21, J26, E24.

* We would like to thank Jesus Crespo Cuaresma, Sebastian Vollmer, and participants of the ICAS Fellows Colloquium for discussion and comments. Holger Strulik conducted his research for this paper during a fellowship at the M.S. Merian – R. Tagore International Centre of Advanced Studies "Metamorphoses of the Political: Comparative Perspectives on the Long Twentieth Century" (ICAS:MP), an Indo-German research cooperation funded by the German Federal Ministry of Education and Research (BMBF). All views expressed here are solely that of the authors.

† Department of Economics, University of Copenhagen and CEPR (London), email: Casper.Worm.Hansen@econ.ku.dk

‡ Department of Economics, University of Copenhagen, CAGE (Warwick) and CEPR (London), email: carl.johan.dalgaard@econ.ku.dk

§ University of Göttingen, Department of Economics, Platz der Göttinger Sieben 3, 37073 Göttingen, Germany; email: holger.strulik@wiwi.uni-goettingen.de.

1. Introduction

The world is aging. This fact is an inevitable consequence of the demographic transition that has been sweeping most countries in the world since the 19th century and continuing throughout the 20th century with falling fertility and mortality rates (Reher, 2004). The process of population aging has later been fueled by a longevity transition in which mortality rates at later ages have been falling as well (e.g., Eggleston and Fuchs, 2012). Population aging is argued to be a key factor in shaping the current and future development of societies, often through its first order impact on labor markets (e.g, Gordon, 2016). The idea is that labor force participation rates follow a hump-shaped path over the life cycle, typically peaking in the 40s, and so if more people reach later ages, aging is mechanically reducing the size of the labor force.

This line of reasoning, however, implicitly assumes that labor force participation depends on chronological age and is not influenced by factors of physiologically aging that are naturally embedded in population aging, such as declining functionality of the human body. For example, according to the popular Mincer (1974)-wage equation, log-wages increase linearly with age and decline linearly with age-squared. The squared term eventually becomes dominating and causes individuals to retire. While it is intuitively plausible that the positive association with age captures increasing productivity through experience, it is harder to understand why productivity should decline because of "too much experience". The decline of productivity of elderly workers with advancing chronological age is more convincingly rationalized as a proxy of something else that causes productivity to decline as worker grow older, namely declining health.

The distinction between chronological age and physiological age (state of health) is important because chronological aging is inevitable while physiological aging is malleable; It can be slowed down and perhaps, eventually, abandoned (Jones and Vaupel, 2017; Lopez-Otín et al., 2013). Physiological aging can be explained by increasing loss of redundancy in the human body and therewith deteriorating reliability and increasing frailty (see Arking, 2006; Gavrilov and Gavrilova, 1991). According to this view, the increasing withdrawal of workers from the labor market is caused by declining productivity due to deteriorating muscle strength and motor skills, musculoskeletal pain, and decreasing cognitive abilities (Schaie, 1994; Nair, 2005; Skirbekk, 2004; Hedden and Gabrieli 2004; Strulik and Werner, 2016).

There exists a microeconomic literature that estimates the impact of health on labor supply using individual data. Depending on empirical approach and health measurements, results

differ from study to study (see French and Jones, 2017, for a review). The recent study by Blundell et al. (2021) uses alternative health measures, controls for individual fixed effects and initial conditions, and estimates that deteriorating health explains up to 15% of the decline in employment between ages 50 and 70 in England and the US. Vandenberghe (2021) estimates the association between health and employment at age 50 (and other labor market indicators) for 20 European countries and uses the estimates for counterfactual predictions of employment at age 70. He finds that deteriorating health explains at most 35 percent of the observed reduction in employment. Another literature uses calibrated models of the individual life cycle to asses how health shocks affect labor supply and other life cycle choices (e.g. French and Jones, 2011, Haan and Prowse, 2014, Capatina, 2015). This approach allows to identify several channels, aside from declining productivity, through which health could affect labor supply, such as utility gains from leisure, indirect effects through the impact of increasing life expectancy on savings and education decisions; disability benefits, the pension- and health-insurance system, and medical expenses. Capatina (2015) estimates that the removal of health shocks leads to an increase of labor supply of non-college educated individuals of 10.8 percent and that the largest part (7.4 percent) operates through changing labor productivity.[1]

In this paper, we propose an alternative approach to study at the aggregate level of countries how physiological aging influences age-specific labor force participation rates. In order to do this, we draw on research in the fields of biology and medicine to compute an empirical measure of physiological aging for most countries in the world from 1990 to 2019. This micro-founded variable, known at the frailty index, aggregates age-specific prevalence rates of 32 age-related diseases to a cohort-, age-, country-, and gender-specific measure of aging. The frailty index has been developed by Mitnitski et al. (2001, 2002) and is an established method to assess human aging, used by hundreds of studies in gerontology and medical science. The index simply records the fraction of a large set of aging-related health conditions that is present in an individual. It has been shown that it does not matter which particular health deficits are included in the index as long as there are sufficiently many (Searle et al., 2008). The intuition for this remarkable feature is that health deficits are connected to other health deficits. For example, hypertension

[1]A related literature uses life cycle models to explain how technological progress and perpetual wage growth contributed to the continuous rise of the length of the retirement period over the last century (see e.g. Bloom et al., 2014, and, in the context of the health deficit model, Dalgaard and Strulik, 2017). An extensive literature discusses how health affects the retirement decision through life expectancy and mortality (e.g. Kalemli-Ozcan and Weil, 2011; d'Albis et al., 2012; Kuhn et al., 2015, and, in the context of endogenous education, Hazan, 2009; Cervellati and Sunde, 2013; Hansen and Loenstrup, 2012; and Strulik and Werner, 2016).

is associated with the risk of stroke, heart diseases, kidney diseases, and dementia. The index thus captures in one number the biological aging process defined as the intrinsic, cumulative, progressive, and deleterious loss of function (Arking, 2006; Masoro, 2006).

The frailty index has a microfoundation in reliability theory (Gavrilov and Gavrilova, 1991), and in a network theory of human aging (Rutenberg et al., 2018). The quality of the frailty index has been demonstrated by its predictive power for death at the individual level and for mortality at the group level, as well as for other adverse health outcomes such as the risk of institutionalization in nursing homes and becoming a disability insurance recipient (Rockwood et al., 2006; Blodgett et al., 2016; Hosseini et al., 2021). Dalgaard and Strulik (2014) integrated the frailty index into an economic life cycle theory of health, aging, and death and provided a biologically founded framework to discuss health behavior and health outcomes.[2]

Most of the literature on the frailty index considers aging of individuals. Here, we built on the study of Dalgaard et al. (2022) who computed a frailty index for (subpopulations of) nations, constructed with with data from the Global Burden of Disease (GBD) study (Vos et al., 2020). In panel analysis, controlling for country- and time fixed effects, Dalgaard et al. (2022) showed that the frailty index at the population level replicates a number of regularities that were previously found at the level of individuals. Specifically, it was shown that the frailty index increases with age in exponential fashion, at a rate of 2.8 to 3.0 percent per year of age. This speed of aging was found to be very similar across continents and income groups. Moreover, for a subset of countries, for which mortality data was available, the study showed a strong association of health deficits and mortality. A one percent increase in the frailty index was found to be associated with an increase of the mortality rate by about 3 percent.

We extend this research by constructing the frailty index for cohorts. We then combine the frailty index with cohort-, age-, country-, and gender-specific labor force participation rates to estimate a cohort-based life-cycle model of labor supply for 181 countries over the period 1990-2019. The comparison of cohorts within the same country over the life cycle allows us to exploit fixed effects to control for a range of unobserved factors (e.g., cohort-gender-country fixed effects).

[2]Applications consider, for example, the gender gap in mortality (Schuenemann et al., 2017b) the health gain from marriage (Schuenemann et al., 2019a), fetal origins of late-life health (Dalgaard et al., 2019), and particular health behavior such as addiction (Strulik, 2018), self-control problems (Strulik, 2019), and adaptation to poor health (Schuenemann et al., 2017a).

There exists, however, also a problem of reverse causality i.e., that labor market participation could be influencing physiological aging. In a series of studies, Marmot argues that work affects health due to occupational stress, social position, and sense of being in control of one's life (e.g. Marmot et al., 1991, 1997). Another strand of literature argues that health status is negatively affected by blue collar and physical job burden (e.g. Fletcher et al., 2011; Morefield et al., 2011; Ravesteijn et al., 2016). Specifically, in the context of the frailty index, it has been shown that the exposure to physical or psychosocial job burden as well as employment in blue collar occupations is associated with a faster accumulation of health deficits during the work life (Abeliansky and Strulik, 2021). In order to address the issue of reverse causality; our identification strategy exploits a compensating regularity of the frailty index, implying that there exists a strong negative relationship between initial health deficits, measured at the beginning of working life, and the rate of health deficit accumulation. This pattern in the frailty index has been documented in previous research for samples of individuals (Mitnitski et al., 2002; Abeliansky and Strulik, 2018, Abeliansky et al., 2020) and it is also present in our global study.

We find that physiological aging has a negative effect on labor force participation rates. Our baseline estimate suggests that, if the frailty index increases by one percent, the labor force participation rate decreases by 0.25 percentage points. In 2SLS estimations the point estimates increases in absolute value to more than 0.6 percentage points. Noticing that the frailty index increases, on average, by 2.6 to 3.0 percent per year over the life cycle, these estimates indicate that there is a substantial drag on labor supply of deteriorating health due to physiological aging.

Our results provide an empirical reason as to why the relationship between age and labor force participation eventually becomes negative. When health deficits are missing in regressions of labor markets participation, the age coefficients suggest that the positive effect of chronological age (experience) on labor supply is reverted around age 35–39. When we control for health deficits, chronological age exerts a positive impact until age 55 and chronological age alone would not be able to explain deteriorating participation rates. The decline by almost 30 percentage points from ages 30-40 to ages 60-64 is almost fully accounted for by deteriorating health.

The paper proceeds as follows. In the next section, we introduce the frailty index and its measurement at the macro level, explain the compensation law of morbidity, and set up a simple life cycle model to derive the implications of physiological aging on labor force participation. In

Section 3, we introduce our data and in Section 4 we explain our estimation strategy. In Section 5 we present the results. Section 6 discusses implications and concludes.

2. Measurement and Theory

2.1. **The Frailty Index.** A widely used empirical measure for human aging has been developed by Mitnitski and Rockwood and various coauthors in a series of articles in the form of the *frailty index* (Mitnitski et al., 2002; Mitnitski et al., 2013; Mitnitski et al., 2016; Rockwood and Mitnitski, 2007). As humans age, they develop an increasing number of health deficits. Some of these deficits may be viewed as relatively mild nuisances while others are more serious in nature. The notion is that when the number of deficits rises the body becomes more frail. A frailty index can then be constructed for an individual as the proportion of the total potential deficits (**a**ilments) $a = 1, ..., A$ that an individual has. That is, the frailty index of individual j with gender g living in country i is:

$$d_{jgi} = \frac{1}{A} \sum_{a=1}^{A} \mathbf{1}_{jgi}(a), \tag{1}$$

where $\mathbf{1}_{ji}(a)$ is an indicator function that takes on the value 1 if individual j suffers from deficit a. The criteria for the selection of health deficits are outlined in Searle et al. (2008): they need to be aging-related (prevalence increasing in age), associated with health status, not saturate too early, and cover a broad range of deficits. No specific deficit is required to enter into the index, since results appear to be unaffected by the specific list of deficits as long as a sufficient number of deficits – 30 to 40 – are included (Rockwood and Mitnitski, 2007; Searle et al., 2008). As explained in the Introduction, the intuition for the remarkable feature that the appearance of specific deficits is not decisive lies in the micro-foundation of the frailty index in reliability theory (Gavrilov and Gavrilova, 1991) and in a network theory of human aging (Rutenberg et al., 2018); theories that emphasize that health deficits are connected.[3]

In light of its simplicity and intuitive nature, it is perhaps unsurprising that the frailty index has been applied in hundreds of studies until now. However, in most of these studies the index is computed for samples of individuals. Here, we follow the methodology developed in Dalgaard et al. (2022) and compute the frailty index for populations. Specifically, given the measurement

[3]The large literature of micro studies using the frailty index typically uses an unweighted index because the weighting of the items limits generalizability across populations and studies. Studies comparing weighted and unweighted indices found that weighting improves prediction quality (for mortality) slightly, but that these gains are not large enough to give up generalizability (e.g. Theou et al., 2013).

of the frailty index at the individual level in equation (1), the average frailty index of cohort c, gender g, in country i is:

$$D_{cgi} = \frac{1}{P_{cgi}} \sum_{j}^{P_{cgi}} d_{jgi}, \tag{2}$$

where P_{cgi} is the number of individuals belonging to cohort c, gender g, living in country i. Inserting equation (1) and rearranging, allows us to write equation (2) as:

$$D_{cgi} = \frac{1}{A} \sum_{a=1}^{A} \frac{P_{acgi}}{P_{cgi}}, \tag{3}$$

where P_{acgi}/P_{cgi} is the prevalence rate of age-related (disease) condition a in cohort c, gender g, country i. Therefore, in order to work out the aggregate frailty index for this particular age cohort, we simply need to calculate the average of A prevalence rates, P_{acgi}/P_{cgi}. These prevalence rates are available for most countries in the world in the GBD database (Vos et al., 2020).

2.2. **Compensation Law of Deficit Accumulation.** Research on the dynamics of health deficits accumulation has established that health deficits grow, on average, at a constant rate with advancing age (Mitnitski et al., 2002a; Mitnitski and Rockwood, 2016; Abeliansky and Strulik, 2018) such that a person j at age t displays $D_j(t)$ health deficits:

$$D_j(t) = D_j(0)\mathrm{e}^{\mu_j t} \quad \Leftrightarrow \quad \log D_j(t) = \log D_j(0) + \mu_j t. \tag{4}$$

This research has also shown that there exists a strong negative relationship between initial health deficits and the rate of health deficit accumulation (Mitnitski et al., 2002a, Abeliansky and Strulik, 2018). The relationship is akin to the compensation law in mortality, also known as Strehler-Mildvan correlation (Strehler and Mildvan, 1960; Gavrilov and Gavrilova, 1991). It implies that there exists an age $t = T$ at which all individuals from a population are predicted to display the same frailty index, $D_j(T) = D(T)$ for all j.

Figure 2 displays the compensation law for two individuals $j = 1, 2$. Individual 1 starts with less health deficits but ages faster such that at age T both individuals display the same frailty index. Formally the compensation law states that

$$\log D_j(0) = \lambda - \mu_j T. \tag{5}$$

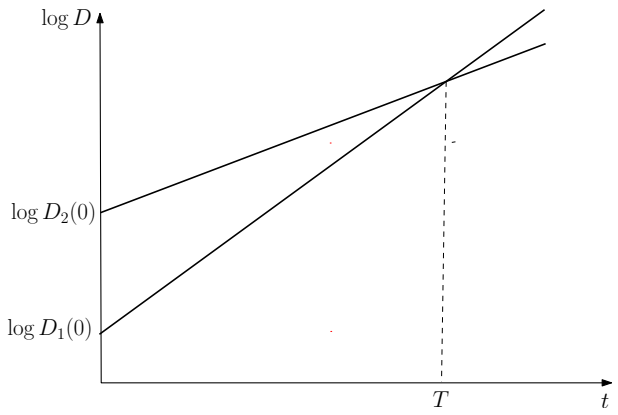
The parameters T and $\lambda \equiv \log D(T)$ have been estimated with great precision and the estimates suggest that T is about 95 to 105 years, depending on the population investigated

(Mitnitski et al, 2002; Abeliansky and Strulik, 2018; Abeliansky et al., 2019). Since T is independent from j, it is considered a population-specific constant and it has been associated with the life span of a population (Gavrilov and Gavrilova, 1991). Substituting $D_j(0)$ in (4) by (5), we obtain:

$$\log D_j(t) = \lambda - \mu_j(T - t). \tag{6}$$

Recall, that due to (4), initially healthier individuals (with less health deficits) display a higher rate of aging μ_j such that, for any $t < T$, they display less health deficits $D_j(t)$ than initially less healthy individuals, see Figure 1.

FIGURE 1. Compensation Law of Health Deficits



We will exploit the compensation law in our empirical strategy. In particular, taking age differences of (4), we obtain $\Delta \log D_j = \mu_j$ and inserting (5) we obtain the association between the growth rate of deficits and the initial level of deficits, $\Delta \log D_j \equiv \log D_j(t) - \log D_j(t-1) = (\lambda/T) - (1/T) \log D_j(0)$. Considering individuals j drawn from different countries i, cohorts c, and gender g, and allowing for country- cohort, and gender-fixed-effect FE, we obtain:

$$\Delta \log D_{cgi} = \alpha \log D_{cgi}(0) + FE, \tag{7}$$

in which $\alpha = -1/T$, $\Delta \log D_{cgi} = \mu_{cgi}$ is the growth rate of health deficits and $D_{cgi}(0)$ are initial health deficits of an individual from cohort c, gender g, and country i. Equation (7) is our theoretical motivation for the first-stage regression given in equation (14) below.

2.3. **Labor Force Participation.** In this section, we integrate health deficits in a standard model of optimal labor force participation and derive the structural model for the regression

analysis. Consider an individual j experiencing instantaneous utility $u(c_j) - \phi_j \ell_j$, in which c_j is consumption, ℓ_j is labor supply and ϕ_j is disutility from work. The utility function u exhibits positive and declining marginal utility $u' > 0$, $u'' < 0$. Here we focus on labor supply at the extensive margin such that $\ell_j \in \{0,\ 1\}$, i.e. individuals are in or out of the labor force. Individuals maximizes lifetime utility:

$$\int_0^T \left[u(c_j(t)) - \phi_j \ell_j(t)\right] \mathrm{e}^{-\rho t} \mathrm{d}t, \tag{8}$$

with time preference rate ρ, subject to the budget constraint $\dot{k}_j(t) = r k_j(t) + \mathbf{1}_{\ell_j(t)=1} w_j(t) - c_j(t)$, in which t is age, k_j are assets, r is the interest rate, w_j is the wage rate and $\mathbf{1}_{\ell_j=1}$ is an indicator function that assumes the value of one if the individual is in the labor force (and zero otherwise). Parameters depend potentially on gender-, cohort-, and country-specific characteristics. When possible without loss of information, the respective indices are suppressed to avoid notational clutter.

The wage at age t is a function of age and health. Let initial age, i.e. the age at which individuals enter the workforce be normalized to zero. The wage-per-age function is then given by:

$$w_j(t) = \omega_j e_j(t) g(D_j(t)), \tag{9}$$

in which the initial wage ω_j summarizes education and other parameters given at the point of entry in the workforce. Allowing for pension income, the left-hand side of (9) would be rewritten as $w_j(t)(1 - \xi(t))$, in which $\xi(t)$ is the replacement rate. The structure of the problem would be preserved. The term $e_j(t)$ captures experience, which grows with increasing age (duration of stay in the workforce). As discussed in the Introduction, according to the original Mincer (1974)-wage equation, a negative term of "experience squared" causes wages to decline in old age such that individuals withdraw from the labor force. Here, we consider instead that productivity is reduced by the presence of health deficits, $g(D)$. The function g is declining in deficits and concave such that $g' < 0$ and $g'' \leq 0$. Experience is assumed to be a positive and concave function of age with $e_j' > 0$ and $\lim_{t\to\infty} e_j' = 0$. This means that, eventually, with increasing age, health deficits become the dominating force on productivity, which causes individuals to exit the labor force.

Individuals maximize (8) subject to (9) and (10), the asset accumulation function and potentially other dynamic constraints. Irrespective of the complexity of the underlying dynamic

problem, the first order condition for labor supply is straightforward since it does not reflect intertemporal trade-offs but the intra-temporal trade-off between working and not working. Specifically, the first order condition for labor supply reads:

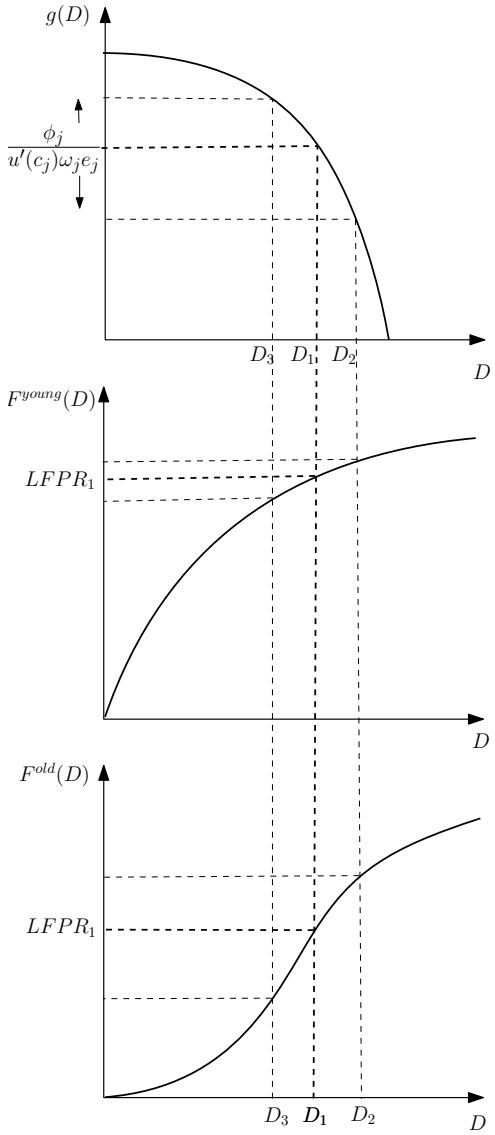
$$\phi_j \leq u'(c_j(t))\omega_j e_j(t) g(D_j(t)), \tag{10}$$

which requires that the marginal disutility from work is not larger than the marginal utility from work, consisting of earned income evaluated at the marginal utility $u'(c)$ that a unit of income can buy. At the point of optimal retirement, (10) holds with equality. Inspection of (10) shows that, ceteris paribus, i.e. for given parameters and functional forms, individuals who have accumulated more health deficits withdraw earlier (at smaller t) from labor force participation.[4] The top panel of Figure 3 illustrates the participation constraint for labor supply. In the benchmark case, only individuals with less than D_1 health deficits supply labor.

In order to make inferences from individual choice on the labor force participation rate of a cohort at a given age, we exploit the result from Rockwood et al. (2004) that health deficits at any age are approximately Gamma-distributed with cumulative distribution function $F(D) = 1/\Gamma(\kappa)\gamma(\kappa, D/\theta)$. Rockwood et al. (2004) show that the distribution function is age specific and that the shape parameter κ increases with age while the scale parameter θ declines. Diagrammatically this means that $F(D)$ is strictly concave at young ages and becomes s-shaped for the elderly, as shown in the center- and bottom-panel of Figure 3. This feature expresses the fact that most young individuals display a small frailty index while among the elderly only few individuals exhibit a small frailty index and many display a health deficits in the intermediate range. The labor force participation rate (LFPR) can be read off directly from Figure 1 since only individuals with deficits below the $F(D)$–curve supply labor. Notice that a variation in individual characteristics, i.e. a movement of the $\phi_j/[\omega_j u'(c(t))e(t)]$, elicits a greater variation of the LFPR for the elderly than for young workers. This feature simply expresses the fact that among elderly workers there are more individuals with health deficits in the vicinity of the LFPR threshold. Figure 3 illustrates this feature for a given variation of the threshold indicated by dashed lines. Ceteris paribus, the model thus predicts that labor force participation of older

[4]We could arrive at a condition isomorph to (10) via an alternative pathway. To see this, assume that health deficits leave productivity unaffected but increase the disutility from work, such that ϕ_j is replaced by $\phi_j f(D)$ with $f' > 0$, $f'' \geq 0$. In reality both mechanisms are likely operative and we can imagine that one function $g(D)$ captures their joint effect in reduced-form.

FIGURE 2. Health Deficits and Labor Force Participation



The upper panel shows the association between health deficits and productivity and the optimal deficit level at entry into retirement (the threshold). The center (bottom) panel shows the distribution of health deficits in the young (old) population and the implied rate of labor force participation.

cohort depends more strongly (with greater elasticity) on health deficits than that of young cohorts.

The threshold also moves with advancing chronological age. As individuals gather more experience, $e_j(t)$ rises, and the threshold moves down. This feature reflects the fact that withdrawing from the labor force causes larger income losses due to experience as individuals grow older. In particular, for young and middle-aged workers this effect could be the dominating force such

that labor supply increases with age. For the elderly, however, it is likely that the gains from experience have asymptotically reached its limit such that the physiological aging effect (strongly) dominates. In summary, the theory predicts a strong negative impact of health deficits on LFPR for the elderly while the effect is smaller for the young and middle aged. The reason is that, on average, elderly workers have developed more health deficits such that a greater share of them has frailty index in the range where workers consider retirement.

Finally, we can use the theory to discuss the evolution of the LFPR of cohorts over time. The $F(D)$–curve of later-born cohorts is first order stochastically dominated by $F(D)$ of earlier cohorts if later-born cohorts are healthier at any age (as the studies for European countries and the U.S., suggest; Abeliansky and Strulik, 2018; Abeliansky et al., 2020). Furthermore, the LFPR may shift over time due to changing education or changing preferences for LFP (of, for example, women) as well as due to technological progress and income growth. With growing income, ω_j increases, shifting the threshold upwards. However, consumption increases as well, implying that $u'(c)$ goes down and that the movement of the LFPR is ambiguous. For the special case of log-utility and a constant savings rate, the threshold stays constant under technological progress and income growth. Generally, however, the LFPR may shift for various non-health-related reasons from one cohort to the next, a feature that highlights the importance of a cohort-based analysis.

In order to eliminate the non-linearities, we approximate the $F(D)$–curve by a logarithmic function. Allowing the parameters of the labor supply model to be country -, gender-, age-, and cohort-specific, the theory predicts that

$$LFPR_{cgit} = \beta \log D_{cgit} + FE, \tag{11}$$

in which subindex c, g, i, and t identifies the cohort, gender, country, and age of the considered individuals and FE are group-specific fixed effects. This provides the structural model for our regression analysis. The theory predicts that $\beta < 0$, which our analysis outlined below will be empirically testing.

3. Data and Sampling

3.1. **Disease Prevalence Data.** Data on disease prevalence rates are taken from the GBD database (Vos et al., 2020), which covers the period 1990 to 2019. The prevalence rates are available for men and women by five year age-groups. Keeping with conventions in the literature

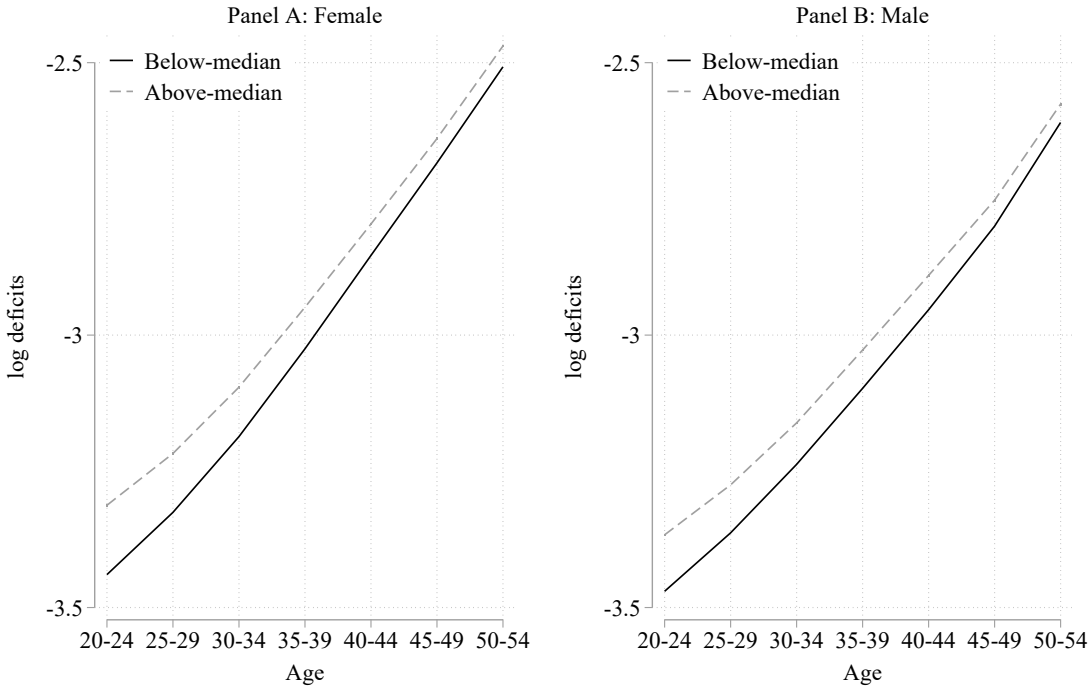
on health deficits, the youngest cohort included in our analysis is age 20 to 24 when sampled. In the construction of the frailty index, we abide by the criteria listed in Searle et al. (2008) and we strictly follow O'Donovan et al. (2020) and Dalgaard et al. (2020) in the selection of disease items. This leaves us with 32 aging-related health conditions, which are listed in the Appendix. The prevalence rates of these diseases are aggregated into the frailty index as suggested by equation (3).

Figure 3 provides empirical evidence for the compensating effect of deficits across countries. We split our sample of cohorts into above and below median initial deficits (measured at age 20-24) and and display average deficits of these two groups over the life cycle by gender. The estimates are reported for women in Panel A and men in Panel B. This shows clearly that the compensating law of deficits is also present in our sample in that cohorts with different levels of initial deficits are converging in terms of deficits over the life cycle. The initial log level of deficits is inversely related to the growth rate of deficits, reflecting the compensation law of health deficit accumulation (see Section 2.2).

3.2. **Labor Supply Data.** This subsection presents our data on life-cycle labor supply. Data on life-cycle labor market participation rates are drawn from the International Labour Organization's (ILO) database (ILO, 2021). Labor force participation rates (LFPRs) by gender, age, and country are, in principle, available annually at five-year age intervals. As our baseline sample consists of birth cohorts of five-year intervals observed from 1990 to 2029, we only need data on LFPRs (by gender, age, and country) every fifth year (1990, 1995,.., 2019).[5] The age groups available from the ILO database are: 20-24, 25-29, .., 60-64 and 65+, but since we use five-year birth cohorts, the latter age-group (65+) cannot be used in our analysis and is accordingly dropped. Figure 4 shows binned scatter plots of the LFPR over the life cycle by gender for our cohorts, while controlling for country fixed effects. For both men and women, we observe the well-known hump-shaped pattern of LFPRs in age. In addition, we see an upward movement of the labor supply curve for women (Panel A). After being merged together with our health deficits data, our dataset ends up consisting of 120 age-cohort-gender observations for 181 countries amounting to 22,806 in total.

[5]The latter age interval is only four years as we do not have data on deficits nor labor force participation from 2020.

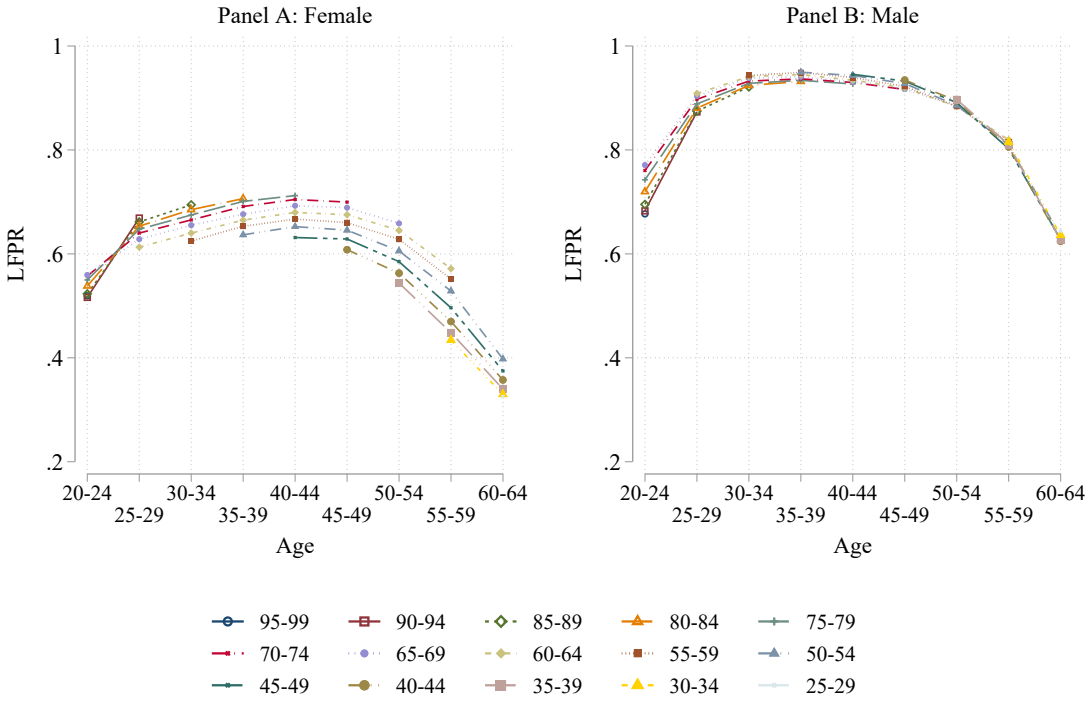
FIGURE 3. Compensation Law of Health Deficits



Notes: This figure shows the development of the average frailty index over the life cycle from age 20-24 to 50-54 by splitting the sample into above and below median initial health deficits, measured at age 20-24. Panel A reports estimates for females, while Panel B reports estimates for males. We can only report these developments until age 50-54 since initial deficits (at age 20-24) are only observed for a sub-sample of our cohorts. See also Section 3.3.

3.3. **Cohort Sample Structure.** We consider five-year (birth) cohorts, which in our baseline sample, includes cohorts born: 1925-1929, 1930-1934, .., 1995-1998. As the Lexis diagram in Figure 5 illustrates, our data allow us to observe both physiological aging and labor participation for these cohorts from 1990 to 2019 up to the age category 60-64, corresponding to the so-called diagonal "life lines". The yellow stars indicate when we measure initial deficits for the different cohorts. Because of data availability on deficits, we are not able to measure deficits at the beginning of the life cycle (age 20-24) for cohorts born before 1965, so initial deficits are measured at older ages for these cohorts. We account for this problem empirically by additionally controlling for age-cohort and age-country fixed effects in the 2SLS regressions. In addition, we cannot use the youngest cohort born 1995-1998 in the regressions, as we do not observe any age changes in deficits and labor force participation for this cohort.

FIGURE 4. Labor Force Participation by Gender and Cohort



Notes: This figure shows the binned scatter plot of LFPRs from age 20 to 64 by gender and cohort. Before showing the LFPRs, we control for country fixed effects in the full sample. The sample here includes 15 different cohorts in 181 countries observed from 1990 to 2019. The numbers in the legend indicate birth years for the different cohorts; the oldest cohort being born from 1925 to 1929 (25-29), for example.

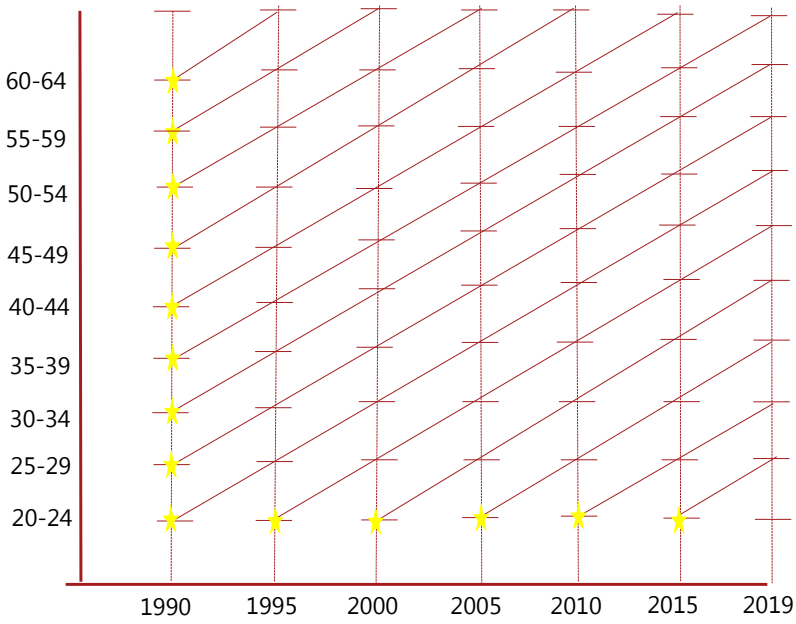
4. Estimation Strategy

This section explains how we plan to empirically estimate the effect of physiological aging on labor supply. Motivated by our theoretical model for health deficits and labor supply, the structural equation takes on the following form:

$$LFPR_{cgit} = \beta \log D_{cgit} + \theta_{gt} + \delta_{cgi} + \varepsilon_{cgit}, \tag{12}$$

where $LFPR_{cgit}$ is the labor force participation rate of birth cohort c with gender g, living in country i, observed at age t. The corresponding frailty index (in natural logarithm) is given by $\log D_{cgit}$. We include gender-age fixed effects (θ_{gt}) and cohort-gender-country fixed effects (δ_{cgi}) in the baseline, but in 2SLS estimation, we also control for country-age fixed effects (θ_{ct}) and cohort-age (θ_{ct}) fixed effects in order to take into account that initial deficits are measured later in life for older cohorts, as explained above.

FIGURE 5. Lexis Diagram for Sampled Cohorts from 1990 to 2019



Notes: This figure shows the structure of our cohort sample across. The yellow stars indicate when initial deficits are measured for the different cohort. It is evident that our limited sample window prevents us from measuring initial deficits for all cohorts at age 20-24 and for these cohorts we instead measure them when entering the sample window in 1990.

We estimate the coefficient of interest, β, by taking first differences in age, which differences out the cohort-gender-country fixed effect. This gives the following estimation equation:

$$\Delta LFPR_{cgit} = \beta \Delta \log D_{cgit} + \Delta\theta_{gt} + \Delta\varepsilon_{cgit}, \tag{13}$$

where $\Delta LFPR_{cgit}$ is the change in the labor force participation rate from age t to t+1, $\Delta \log D_{cgit}$ is the growth rate of deficits, $\Delta\theta_{gt}$ are gender-age fixed effects, and $\Delta\varepsilon_{cgit}$ is the error term, which is clustered at the country level.

Compared to the "level model" in equation (12), this age-stacked, first-differences specification is easier to connect with our 2SLS strategy, which is going to exploit differences in initial deficits between cohorts as an IV for the growth rate of deficits (i.e., the change in logged deficits) as theoretically motivated by equation (7). Based on this compensation law, the first stage is constructed as:

$$\Delta \log D_{cgit} = \alpha \log D_{cgi}^{Initial} + \Delta\tilde{\theta}_{gt} + \Delta\tilde{\varepsilon}_{cgit}, \tag{14}$$

where $\log D_{cgi}^{Initial}$ is deficits measured at the beginning of the work-life cycle (age 20-24), which can only be measured for cohorts born later than 1965 given the GBD data on deficits. Therefore, we measure initial deficits in 1990 for cohorts born before 1965 when they enter our sample window (cf. Figure 5). In order not to compare older cohorts, where initial deficits are thus measured later in life, we follow two related approaches. First, we restrict the sample only to cohorts born after 1965, for which it is possible to measure initial deficit at age 20-24. Alternatively, we include all cohorts in the sample, but then control for cohort-age and country-age fixed effects in order not to make any misleading comparisons. Finally, note that the excluded instrument (initial deficits, $\log D_{cgi}^{Initial}$) does not vary by age, and so our 2SLS estimation should essentially be thought of a number of stacked first differences (in age), and motivated by the law of compensating deficits, initial deficits are assumed to have the same impact on the change in logged deficits from one age to the next. Our identifying assumption, in terms of solving the inherent problem of reverse causality, is that labor market participation later in wor life (e.g., at age 50-54) does not affect health deficits at beginning of the work life (age 20-24).

5. Results

Table 1 reports the results from estimating different variants of equation (13) by OLS. When not including any fixed effects, in column 1, we find that $\hat{\beta} = -1.5$ with a standard error of 0.07, implying that if deficits increase by one percent, labor force participation decreases by 1.5 percentage points. In the subsequent columns, when age and gender FE are included, we observed that the estimated effect reduces to about 0.25 to 0.28 percentage points.[6] Noting that health deficits increase, on average, by around 3 percent per year over the life cycle, even this magnitude implies a substantial drag on labor supply of physiological aging. The point estimate from specification (4) in Table 1 implies that a one-standard deviation of the health indicator (the standard deviation of the logged frailty index is 0.49) is associated with a decline in labor force participation by 12 percentage points. For comparison, using micro data, Vandenberghe (2021) estimates for a sample of workers aged 50–54 from 20 European countries that a one standard deviation in health is associated with a decline of labor force participation between 12 and 30 percentage points. Our OLS estimates based on macro data are thus in line with estimates from the lower bound of a recent study using micro data.

[6]Including gender fixed effects in equation (13) corresponds to controlling for gender-specific linear trends in equation (12).

TABLE 1. OLS Estimates

	(1) $\Delta LFPR$	(2) $\Delta LFPR$	(3) $\Delta LFPR$	(4) $\Delta LFPR$
$\Delta \log D$	-1.493***	-0.282***	-0.279***	-0.255***
	(0.067)	(0.049)	(0.049)	(0.056)
Observations	17,014	17,014	17,014	17,014
Age FE	No	Yes	Yes	Yes
Gender FE	No	No	Yes	Yes
Gender-Age FE	No	No	No	Yes

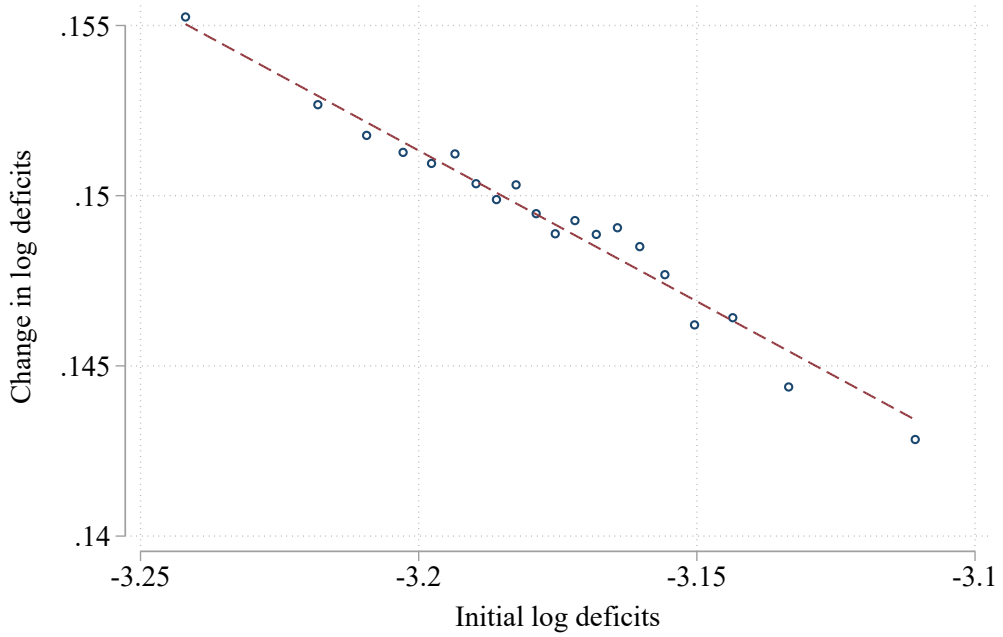
Notes: This table reports OLS estimates of equation (13). The dependent variable ($\Delta LFPR$) is the change in labor force participation from one age to the next, while the main explanatory variable ($\Delta \ln D$) is the change in logged deficits. Standard errors are robust and clustered at the country level. *** p<0.01, ** p<0.05, * p<0.1

While the estimates reported in Table 1 hold constant a host of unobserved country-, age-, and cohort-factors, such as years of schooling, which is largely determined before entering work-life, they are unlikely to yield the causal effect of physiological aging on labor supply, since labor supply influences physiological aging (see the discussion of the related literature in the Introduction). For this reason, we expect the OLS coefficient of health deficits to be downward biased (in absolute value) and now turn our attention to the 2SLS strategy, which exploits the compensating law of health deficits accumulation to construct an IV.

In Figure 6, we depict the first-stage relationship as a binned scatter plot for our full sample of cohorts, while controlling for gender-age, county-age and cohort-age fixed effects. This first-stage coefficient is estimated as $\hat{\alpha} = -0.09$, which is statistically significant at the one percent level. This magnitude implies that if initial deficits decrease by a one standard-deviation of a natural log point (0.3), the growth rate of deficit increases by 3 percentage points. Given that we took differences of five year intervals, it implies a change of the annual growth rate of deficits by 0.6 percentage points. As can be seen from the bottom of Table 3, the Kleibergen-Paap F statistics ranges from 72 to 172 in all 2SLS specifications, indicating that the first-stage fit is strong, which reduces any concerns about weak-instrument biases.

Table 2 reports the resulting 2SLS estimates. In Columns 1-4, we include only cohorts born later than 1965, where (given our data on deficits) it is possible to measure initial deficits at the beginning of the work-life cycle. In these models, with the same fixed effects as in Table 1, the first-stage relationship is a little stronger compared to the model where all cohorts are included

FIGURE 6. First-stage Relationship between Initial Deficits and Changes in Deficits



Notes: The figure shows a binned scatter plot of the first-stage relationship, in which we include all cohorts and control for gender-age fixed effects, country-age fixed effects, and cohort-age fixed effects. $\hat{\alpha} = -0.09$ and standard error = 0.01.

along with additional interaction fixed effects, reported in Figure 6 and Column 5 of Table 2. However, in all the reported models, the 2SLS estimate is about -0.6 (standard error 0.2), which is more than twice the numerical magnitude of the OLS estimates, reported in Columns 2-4 of Table 1. This pattern is consistent with the OLS estimate being downward biased due to reverse causality. The estimate from specification (4) in Table 2 implies that a one-standard deviation of the health indicator is associated with a decline in labor force participation by 30 percentage points. This estimate is in line with the upper bound of estimates from micro data in the Vandenberghe (2021) study.

5.1. **Age and Labor Force Participation: Simulations.** In this section, we aim to assess the explanatory power of physiological aging for labor force participation rates (LFPRs) by way of simulation. The simulations are based on the level estimates shown in Table 3 and the evolution of health deficits by age. We first consider labor supply of an average world citizen in the panel on the left-hand side of Figure 7. The blue (solid) line shows the age-coefficients when the LFPR is regressed on age fixed effects and gender-country-sex fixed effects (reported

Table 2. 2SLS Estimates

	(1) $\Delta LFPR$	(2) $\Delta LFPR$	(3) $\Delta LFPR$	(4) $\Delta LFPR$	(5) $\Delta LFPR$
$\Delta \log D$	-0.600***	-0.594**	-0.630***	-0.615***	-0.636***
	(0.230)	(0.229)	(0.190)	(0.189)	(0.243)
Observations	7,602	7,602	7,602	7,602	17,014
Age FE	No	Yes	Yes	Yes	Yes
Gender FE	No	No	Yes	Yes	Yes
Gender-Age FE	No	No	No	Yes	Yes
Country-Age FE	No	No	No	No	Yes
Cohort-Age FE	No	No	No	No	Yes
Cohorts	>1965	>1965	>1965	>1965	all
First-stage F stat.	110.6	110.4	171.7	172.3	71.74

Notes: This table reports 2LS estimates of Eq. 13, using Eq. 14 as the first stage. The dependent variable ($\Delta LFPR$) is the change in labor force participation from one age to the next, while the main explanatory variable ($\Delta \ln D$) is the change in logged deficits. In Columns 1-4 only cohorts born before 1965 are include, while in Column 5 all cohorts are included. Standard errors are robust and clustered at the country level. *** p<0.01, ** p<0.05, * p<0.1

in column 1 of Table 3). According to this view, increasing as well as declining LFPRs is "explained" by chronological age. This model suggest that labor supply starts declining from about age 40 because workers are getting older. It could have been derived from a standard Mincer model, according to which worker productivity and wages decline with chronological age as elderly workers grow older.

The red (dashed) line shows the predicted LFPR by age when logged deficits are added in the regression and age-group-specific health deficit are fed into the model. Estimates are taken from column 2 of Table 3. We assume that new health deficits are accumulated at a rate of 2.9 percent per year of age. This average speed of physiological aging has been estimated by Dalgaard et al. (2022) using the same data. It implies that the frailty index increases from about 0.03 at age 20-24 to 0.09 at age 60-64 (see also Figure 3). The predicted LPFR by age closely traces the age-specific LFPR but the explanation differs. Now, deteriorating health is the main driver of declining LFPRs. This can be seen by the black (dash-dotted line), which reports predicted LFP assuming constant health. Controlling for health status, labor force participation is predicted to increase until age 50-54 and the LPFR at age 60-64 is as high as at age 25-29. The area between the red and the black line provides an estimate of the increase in LFPR that

TABLE 3. Level Estimates

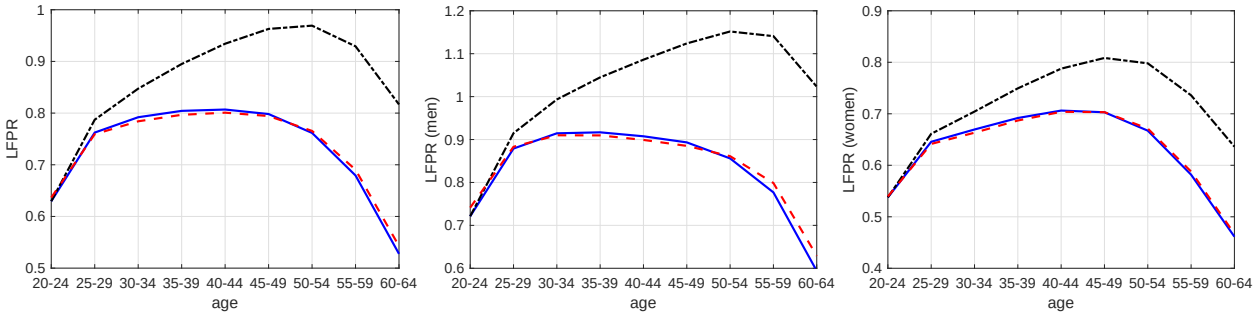
	(1) $LFPR$	(2) $LFPR$	(3) $LFPR$	(4) $LFPR$	(5) $LFPR$	(6) $LFPR$
$\log D$		-0.242***		-0.148**		-0.357***
		(0.051)		(0.073)		(0.071)
age 25-29	0.133***	0.158***	0.108***	0.123***	0.158***	0.194***
	(0.005)	(0.008)	(0.006)	(0.010)	(0.006)	(0.010)
age 30-34	0.163***	0.218***	0.132***	0.167***	0.193***	0.272***
	(0.006)	(0.014)	(0.007)	(0.019)	(0.007)	(0.018)
age 35-39	0.175***	0.266***	0.154***	0.212***	0.196***	0.324***
	(0.007)	(0.020)	(0.008)	(0.029)	(0.008)	(0.028)
age 40-44	0.177***	0.305***	0.169***	0.250***	0.186***	0.365***
	(0.007)	(0.028)	(0.008)	(0.041)	(0.008)	(0.038)
age 45-49	0.169***	0.333***	0.165***	0.271***	0.172***	0.403***
	(0.007)	(0.035)	(0.009)	(0.052)	(0.008)	(0.048)
age 50-54	0.132***	0.340***	0.130***	0.260***	0.135***	0.430***
	(0.008)	(0.044)	(0.009)	(0.065)	(0.008)	(0.061)
age 55-59	0.050***	0.300***	0.044***	0.198***	0.056***	0.420***
	(0.008)	(0.052)	(0.010)	(0.076)	(0.009)	(0.074)
age 60-64	-0.102***	0.187***	-0.076***	0.099	-0.127***	0.302***
	(0.013)	(0.061)	(0.013)	(0.086)	(0.015)	(0.087)
Observations	21,720	21,720	10,860	10,860	10,860	10,860
Cohort-Country-Sex FE	Yes	Yes	Yes	Yes	Yes	Yes
Sample	All	All	Female	Female	Male	Male

Notes: This table reports OLS estimates of Eq. 12. The dependent variable ($LFPR$) is the labor force participation rate, while the explanatory variable ($\log D$) is logged deficits. Age 20-24 is the reference age group for the age dummies. columns 1 and 2 include only all cohorts, while Columns 3-6 split the sample by gender, which means that these specifications only absorb country-cohort fixed effects. Standard errors are robust and clustered at the country level. *** p<0.01, ** p<0.05, * p<0.1

could be achieved by abolishing physiological aging. The estimates suggest a gain in LFPR of more than 15 percent at age 44-49 and of about 25 percent at age 60-64.

The middle panel of Figure 7 shows results when the simulation exercise is repeated in a sample restricted to men (estimates from columns 3 and 4 of Table 3). The predictions with and without consideration of physiological aging (the red and blue line) trace the actual LFP quite well, as a comparison with Figure 4 show. The black (dash-dotted) line, showing LFP with constant health, reveals that the potential LFP gain from slowing down physiological aging is even greater than in the full sample. Of course, an LFPR above 1 makes no sense. Assuming an upper boundary of potential LFP at or below 1, the estimates thus suggest that there is no reduction in LFPR due to chronological aging.

FIGURE 7. Age and Labor Force Participation: Simulations



Blue (solid) lines: predictions when health deficits are omitted as explanatory variable. Red (dashed) lines: predictions when log deficits are included as regressor and deficits increase by 2.9 percent per year. Black (dash-dotted) line: predictions when log deficits are included as regressor but deficits are held constant for prediction.

The panel on the right-hand side of Figure 7 shows the estimated and simulated results for women. Again, the predictions trace the actual LFPR quite well (cf. Figure 4). The estimated coefficient of log deficits is less than half in absolute size for women (0.15 vs. 0.36) and thus the impact of deteriorating health on labor supply is smaller. The potential gain in LFP from abolishing physiological aging, however, is still substantial. It is about 10 percentage points at age 44-49 and almost 20 percentage points at age 60-64. If we assume an upper bound for LFP at or below 0.7, for example because of pregnancy and child rearing, we would arrive at the same conclusion as for men: advancing chronological age does not contribute to declining LFPR when physiological aging is held constant.

6. Conclusion

In this paper, we contributed to the literature on the employment effects of health with a novel approach using macro data. Instead of using individuals as subjects of investigation, we considered cohorts born between 1925 and 1995 from 181 countries and estimated the impact of deteriorating health on labor force participation. Drawing on research in the fields of biology and medicine and aging-related health data on prevalence rates of health deficits from the GBD study, we computed the frailty index as a measure of physiological aging over the life cycle. The panel structure of the data over 1990-2020 allowed us to follow cohorts over time and to control for a host of potential confounders by country, gender, age, and cohort fixed effects. We exploited the association between initial health deficits and the growth rate of health deficits

(the compensation law of morbidity) as an instrument in order to eliminate the problem of reverse causality.

The results suggest a strong negative effect of physiological aging on labor market participation. According to the OLS results, a one standard deviation increase of health deficits is associated with a decline in labor force participation by about 12 percentage points, an estimate that aligns with the lower bound of recent estimates from European micro data. In IV regressions, we found a substantially larger effect, according to which a one percent increase in the frailty index is associated with an 0.6 percentage point decline of the labor force participation rate. This means that an increase of health deficits by one standard deviation leads to a decrease of labor force participation by 30 percentage points, an estimate that aligns with the upper bound of recent micro estimates. Simulations, in which we feed the actual (average) increase of health deficits into an estimated model of life cycle labor supply, we find that almost all decline in labor supply of the elderly can be motivated by deteriorating health and that advancing chronological age (i.e. experience) exerts a positive impact on labor supply until age 55.

The main policy conclusion is thus that, if health of the workforce could be improved, population aging would much less of a concern for labor force participation rates of the population below age 65. Historical studies by Costa (2000, 2002) show that the deterioration of the human body slowed down since the beginning of the 20th century such that later born cohorts of elderly US American men (50-64 years old) experience less impairments of bodily function. Costa shows that a host of health conditions improved quite strongly, some, such as joint problems, back problems, and heart and circulatory conditions, improved at a rate more than twice as fast as the improvement of life expectancy. In a sample of European countries as well as in the US, average health deficits at any age above 50 declined by 1.0-1.4 percent per year of later birth (Abeliansky and Strulik, 2019; Abeliansky et al., 2019). These observations suggest that productivity and labor force participation, in particular of elderly individuals, could be stimulated by improving health.

These trends, however, are not yet visible at the global level. Inspection of our GBD-based data reveals that that younger cohorts in non-western and poor countries are less healthy at all working ages. Physiological aging thus operate against the "demographic dividend", that could be derived from the relatively young work force in these countries (Bloom et al., 2003). Later

born workers in western and rich countries, in contrast, do not experience these negative trends and benefit from (mildly) improved health, in particular at later working ages, in line with the results from the micro studies cited above. These trends, however, are not (yet) sufficiently strong to offset the deleterious effects of population aging on labor supply.

References

Abeliansky, A., and Prettner, K. (2017). Automation and demographic change. Discussion Paper. University of Goettingen.

Abeliansky, A. and Strulik, H. (2018). How we fall apart: Similarities of human aging in 10 European countries. *Demography* 55, 341-359.

Abeliansky, A. and Strulik, H. (2019). Long-Run Improvements in Human Health: Steady But Unequal. *Journal of the Economics of Ageing* 14, 100189.

Abeliansky, A., Erel, D., and Strulik, H. (2020). Aging in the USA: similarities and disparities across time and space. *Scientific Reports* 10(1), 14309.

Abeliansky, A. and Strulik, H. (2021). Health and aging before and after retirement. Discussion Paper, University of Goettingen.

Acemoglu, D., and Restrepo, P. (2018). Demographics and automation (No. w24421). National Bureau of Economic Research.

Aksoy, Y., Basso, H. S., Smith, R. P., and Grasl, T. (2019). Demographic structure and macroeconomic trends. *American Economic Journal: Macroeconomics* 11(1), 193-222.

Arking, R. (2006). *The Biology of Aging: Observations and Principles*, Oxford University Press, Oxford.

Blodgett, J. M., Theou, O., Howlett, S. E., Wu, F. C., and Rockwood, K. (2016). A frailty index based on laboratory deficits in community-dwelling men predicted their risk of adverse health outcomes. *Age and Ageing* 45(4), 463-468.

Bloom, D., Canning, D., and Sevilla, J. (2003). The demographic dividend: A new perspective on the economic consequences of population change. Rand Corporation.

Bloom, D.E., Canning, D. and Moore, M. (2014). Optimal retirement with increasing longevity. *Scandinavian Journal of Economics*, 116(3), 838–858.

Blundell, R., Britton, J., Dias, M. C., and French, E. (2021). The impact of health on labor supply near retirement. *Journal of Human Resources* 1217-9240R4.

Boersch-Supan, A. H. (2008). The impact of global aging on labor, product, and capital markets. *Population and Development Review* 34, 52-77.

Burtless, G. (2013). The Impact of Population Aging and Delayed Retirement on Workforce Productivity. Center for Retirement Research, Working Paper 2013-11.

Capatina, E. (2015). Life-cycle effects of health risk. *Journal of Monetary Economics* 74, 67-88.

Cervellati, M. and Sunde, U., 2013, Life expectancy, schooling, and lifetime labor supply: Theory and evidence revisited. *Econometrica*, 81, 2055–2086.

Cooley, T., and Henriksen, E. (2018). The demographic deficit. *Journal of Monetary Economics* 93, 45-62.

Costa, D.L. (2000). Understanding the twentieth-century decline in chronic conditions among older men. *Demography*, 37, 53–72.

Costa, D.L. (2002). Changing chronic disease rates and long-term declines in functional limitation among older men. *Demography*, 39, 119–137.

d'Albis, H., Lau, S.P., and Sánchez-Romero, M. (2012). Mortality transition and differential incentives for early retirement. *Journal of Economic Theory*, 147, 261–283.

Dalgaard, C.-J. and Strulik, H. (2017). The genesis of the golden age: Accounting for the rise in health and leisure. *Review of Economic Dynamics*, 24, 132-151.

Dalgaard, C.-J., Hansen, C., and Strulik, H. (2022). Physiological aging around the world. Discussion Paper.

Dalgaard, C. J., Hansen, C. W., and Strulik, H. (2021). Fetal origins – A life cycle model of health and aging from conception to death. *Health Economics* 30(6), 1276-1290.

O'Donovan, M., Sezgin, D., Kabir, Z., Liew, A., and O'Caoimh, R. (2020). Assessing Global Frailty Scores: Development of a Global Burden of Disease-Frailty Index (GBD-FI). *International Journal of Environmental Research and Public Health* 17(16), 5695.

Eggleston, K.N., Fuchs, V.R. (2012). The new demographic transition: most gains in life expectancy now realized late in life. *Journal of Economic Perspectives* 26(3), 137-56.

Fletcher, J.M., Sindelar, J.L., and Yamaguchi, S. (2011). Cumulative effects of job characteristics on health. *Health Economics* 20(5), 553-570.

French, E. (2005). The effects of health, wealth, and wages on labour supply and retirement behaviour. *Review of Economic Studies* 72(2), 395-427

French, E., and Jones, J. B. (2017). Health, health insurance, and retirement: a survey. *Annual Review of Economics* 9, 383-409.

Fries, J.F. (1980). Aging, natural death, and the compression of morbidity. *New England Journal of Medicine* 303(3), 130-135.

Gavrilov, L.A. and Gavrilova, N.S. (1991), *The Biology of Human Life Span: A Quantitative Approach*. Harwood Academic Publishers, London.

Goldin, C., Katz, L. F., and Kuziemko, I. (2006). The homecoming of American college women: The reversal of the college gender gap. *Journal of Economic Perspectives* 20(4), 133-156.

Gordon, R.J. (2016). *The rise and fall of American growth: The US standard of living since the civil war*. Princeton University Press.

Haan, P., and Prowse, V. (2014). Longevity, life-cycle behavior and pension reform. *Journal of Econometrics* 178, 582-601.

Hansen, C.W. and Lønstrup, L. (2012). Can higher life expectancy induce more schooling and earlier retirement? *Journal of Population Economics*, 25(4), 1249–1264.

Hazan, M. (2009). Longevity and lifetime labor supply: Evidence and implications. *Econometrica*, 77, 1829–1863.

Hedden, T., and Gabrieli, J. D. (2004). Insights into the ageing mind: a view from cognitive neuroscience. Nature Reviews Neuroscience 5(2), 87-95.

Hosseini, R., Kopecky, K. A., and Zhao, K. (2021). The Evolution of Health over the Life Cycle. *Review of Economic Dynamics*, forthcoming.

ILO (2021). ILOSTAT Data. International Labour Organization. `https://ilostat.ilo.org/data` [accessed on June 2021].

Jones, O.R., and Vaupel, J.W. (2017). Senescence is not inevitable. *Biogerontology* 18(6), 965-971.

Kalemli-Ozcan, S., Weil, D.N. (2010). Mortality change, the uncertainty effect, and retirement. *Journal of Economic Growth*, 15, 65-91.

Kotschy, R., and Sunde, U. (2018). Can education compensate the effect of population ageing on macroeconomic performance?. *Economic Policy* 33(96), 587-634.

Kuhn M., Wrzaczek, S., Prskawetz, A. and Feichtinger, G., 2012. Optimal choice of health and retirement in a life-cycle model. *Journal of Economic Theory*, 158, 186–212.

Lopez-Otín, C., Blasco, M.A., Partridge, L., Serrano, M., and Kroemer, G. (2013). The hallmarks of aging. *Cell* 153(6), 1194-1217.

Maestas, N., Mullen, K. J., and Powell, D. (2016). The effect of population aging on economic growth, the labor force and productivity (No. w22452). National Bureau of Economic Research.

Marmot, M. G., Stansfeld, S., Patel, C., North, F., Head, J., White, I., ... and Smith, G. D. (1991). Health inequalities among British civil servants: the Whitehall II study. *The Lancet* 337(8754), 1387-1393.

Marmot, M., Ryff, C. D., Bumpass, L.L., Shipley, M., and Marks, N. F. (1997). Social inequalities in health: next questions and converging evidence. Social Science & Medicine, 44(6), 901-910.

Mincer, J. (1974). *Schooling, Experience and Earnings*. New York: Columbia University Press.

Mitnitski, A.B., Mogilner, A.J., and Rockwood, K. (2001). Accumulation of deficits as a proxy measure of aging. *Scientific World* 1, 323-336.

Mitnitski, A.B., Mogilner, A.J., MacKnight, C., and Rockwood, K. (2002a). The accumulation of deficits with age and possible invariants of aging. *Scientific World* 2, 1816-1822.

Mitnitski, A.B, Song, X., and Rockwood, K. (2013). Assessing biological aging: the origin of deficit accumulation. *Biogerontology* 14(6), 709-717.

Mitnitski, A.B, and K. Rockwood. (2016). The rate of aging: the rate of deficit accumulation does not change over the adult life span. *Biogerontology*, 17(1), 199–204.

Morefield, B., Ribar, D. C., and Ruhm, C. J. (2012). Occupational status and health transitions. *The B.E. Journal of Economic Analysis & Policy* 11(3).

Nair, K.S. (2005). Aging muscle. *American Journal of Clinical Nutrition*, 81, 953–963.

Ravesteijn, B., Kippersluis, H.V., and Doorslaer, E.V. (2018). The wear and tear on health: What is the role of occupation? *Health Economics* 27(2), e69-e86.

Reher, D.S. (2004). The demographic transition revisited as a global process. *Population, Space and Place* 10(1), 19-41.

Rockwood, K., Mogilner, A., and Mitnitski, A. (2004). Changes with age in the distribution of a frailty index. *Mechanisms of Ageing and Development* 125(7), 517-519.

Rockwood, K., and Mitnitski, A. (2007). Frailty in relation to the accumulation of deficits. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 62(7), 722-727.

Rutenberg, A. D., Mitnitski, A. B., Farrell, S. G., and Rockwood, K. (2018). Unifying aging and frailty through complex dynamical networks. *Experimental Gerontology* 107, 126-129.

Schaie, K.W. (1994). The course of adult intellectual development. *American Psychologist*, 49, 304–313.

Schünemann,, J., Strulik, H., and Trimborn, T. (2017a). Going from bad to worse: Adaptation to poor health, health spending, longevity, and the value of life. *Journal of Economic Behavior and Organization* 140, 130-146.

Schünemann, J., Strulik, H., Trimborn, T. (2017b). The gender gap in mortality: How much is explained by behavior? *Journal of Health Economics* 54, 79-90.

Schünemann, J., Strulik, H., Trimborn, T. (2020). The marriage gap: Optimal aging and death in partnerships, *Review of Economic Dynamics* 36, 158-176.

Searle, S. D., Mitnitski, A., Gahbauer, E. A., Gill, T. M., and Rockwood, K. (2008). A standard procedure for creating a frailty index. *BMC geriatrics*, 8(1), 24.

Strehler, B.L. and Mildvan, A.S. (1960). General theory of mortality and aging, *Science* 132, 14-21.

Skirbekk, V. (2004). Age and individual productivity: A literature survey, *Vienna Yearbook of Population Research*, 133–153.

Strulik, H., and Werner, K. (2016). 50 is the new 30 – long-run trends of schooling and retirement explained by human aging. *Journal of Economic Growth* 21(2), 165-187.

Strulik, H. (2018). Smoking Kills: An Economic Theory of Addiction, Health Deficit Accumulation, and Longevity. *Journal of Health Economics* 62, 1-12.

Strulik, H. (2019). Limited self-control and longevity. *Health Economics* 28(1), 57-64.

Theou, O., Brothers, T. D., Mitnitski, A., and Rockwood, K. (2013). Operationalization of frailty using eight commonly used scales and comparison of their ability to predict all-cause mortality. *Journal of the American Geriatrics Society* 61(9), 1537-1551.

UN (2019). United Nations Population Division. World Population Prospects 2019. `https://population.un.org/wpp/Download/Standard/Population/`

Vandenberghe, V. (2021). Health, cognition and work capacity beyond the age of 50: International evidence on the extensive and intensive margins of work. *International Labour Review* 160(2), 271-310.

Vos, T., Lim, S. S., Abbafati, C., Abbas, K. M., Abbasi, M., Abbasifard, M., ... and Bhutta, Z. A. (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet, 396(10258), 1204-1222.

WHO (2012), Life Expectancy and Healthy Life Expectancy. Global Health Observatory Data Repository, `http://apps.who.int/ghodata/`

7. Appendix

A. Items in the Frailty Index

The frailty index is based on prevalence rates for the following diseases (32 in total):

Diarrheal diseases; Protein-energy malnutrition; Neoplasms; Ischemic heart disease; Stroke; Non-rheumatic valvular heart disease; Cardiomyopathy and myocarditis; Atrial fibrillation and flutter; Peripheral artery disease; Other cardiovascular and circulatory diseases; Chronic respiratory diseases; Peptic ulcer disease; Gallbladder and biliary diseases; Alzheimer's disease and other dementias; Parkinson's disease; Depressive disorders; Diabetes mellitus; Chronic kidney disease; Skin and subcutaneous diseases; Other sense organ diseases; Rheumatoid arthritis; Osteoarthritis; Low back pain; Gout; Urinary diseases and male infertility; Genital prolapse; Endocrine, metabolic, blood, and immune disorders; Oral disorders; Falls; Hearing loss; Heart failure; Blindness and vision loss.