Biological Age Across the Globe: 1990–2019*

Casper Worm Hansen†

Holger Strulik‡

Revised Version. March 24, 2025

Abstract. In this paper, we use data of the Global Burden and Disease Study to compute biological age across the world at the country–age-group–year level and separately for men and women. Biological age is the predicted age of a person determined by their health indicators. As health indicator, we use the frailty index, which is the proportion of age-related health deficits present in a person. We demonstrate that biological age varies significantly across the globe. For instance, the average biological age of chronologically 65-year old men varies between 61 to 74 years across countries. Given chronological age, biological age increased significantly from 1990-2019, in particular in age groups above 65. We also find evidence for conditional convergence of biological age. These trends are driven primarily by biologically young people in Africa who are becoming biologically older, and by biologically old people in rich countries who are becoming biologically younger. We find little evidence of absolute convergence, i.e. declining inequality in the global distribution of biological age.

Keywords: biological age, frailty index, health inequality, global health trends.

JEL: I10, I14, I15.

*We would like to thank Marc Luy, two anonymous reviewers, and the editors Andrew Scott and David Bloom for helpful comments.

† Department of Economics and CEPR, University of Copenhagen. Declarations of interest: none.

‡ University of Goettingen, Department of Economics, Platz der Goettinger Sieben 3, 37073 Goettingen, Germany; email: holger.strulik@wiwi.uni-goettingen.de. Declarations of interest: none.

1. Introduction

At the aggregate level of populations, chronological age is a very strong predictor of mortality among adults aged 30 to 90, a phenomenon known as Gompertz law (Gompertz, 1825; Olshansky, 1997). However, despite its excellent *predictive* power, chronological age lacks *explanatory* power since it does not explain a person's health status or death (Arking, 2008; Strulik, 2023). Chronologically speaking, every person on the planet ages by exactly one year every year while biologically speaking, a 70-year-old can be as healthy and as close to death as a 60-year-old. Moreover, chronological aging is inevitable while biological aging is malleable and influenced by the environment, individual behavior, and health policy.

A straightforward measure that takes into account this heterogeneity and plasticity of aging and mortality is biological age. In this paper, we use data from the Global Burden of Disease Study to calculate biological age worldwide, broken down by country, year, and age group, and separately for men and women. Biological age is the *predicted* chronological age of a person based on measured health indicators (and possibly additional information). It answers the question of how old we would estimate a person to be after examining his or her health indicators. Biological age, as a dynamic and personalized measure, better represents the health and aging process than chronological age and is therefore a more useful indicator for medical and longevity research. At the population level, it provides a more informative assessment of overall health than the chronological age distribution and offers valuable insights for policy-making.

While the early literature estimated biological age from sets of straightforward biomarkers of multiple organ systems (such as systolic heart rate, forced expiratory volume, urea nitrogen, hemoglubin concentration etc.), the recent literature proposed estimates based on genetic information (telomere length, DNA methylation), and blood levels of proteins (proteomic indicators) or metabolites (see Jia et al., 2017, and Jylävä et al., 2017, for a reviews of indicators and methods of biological age estimation). Here, we build on studies that estimated biological age using the frailty index (following the seminal work of Mitnitski et al., 2002a). The key advantage of the frailty index over other biological age measures is that it can be constructed for all age groups and nearly all countries worldwide, using a panel spanning 30 years of data. In contrast, other biological age indicators, such as DNA methylation clocks or telomere length, are typically derived from small population samples in specific countries at single points in time.

The frailty index, developed by Mitnitski and Rockwood, and several coauthors in a series of articles (based on Mitnitski et al., 2001), measures the proportion of a broad list of aging-related health conditions present in an individual. The list includes health deficits ranging from mild to nearly fatal. For a health deficit to be included in the frailty index, it must meet certain criteria: its prevalence should generally increase with age, it must be associated with overall health status, and it should not saturate too early while covering a range of organ systems (see Searle et al., 2008, for a review of the methodology). Research has shown that the specific deficits included in the index do not significantly affect its accuracy, as long as the list is sufficiently comprehensive (Searle et al., 2008; Theou et al., 2013). A higher index number means that people are seen as increasingly frail and in this sense as biologically older. Thus, the frailty index summarizes an individual's health status and biological aging in a single number (see Howlett et al., 2023, for a recent review).

On average, people accumulate health deficits at an exponential rate as they become chronologically older, with the frailty index increasing by about 3 percent from one year to the next. This pattern, similar to Gompertz law, was first observed in Canadians (Mitnitski et al., 2002a,b; Mitnitski and Rockwood, 2016) and has since been confirmed in populations across Europe, the United States, and several developing countries (Harttgen et al., 2013; Theou et al., 2014; Abeliansky and Strulik, 2018; Abeliansky et al., 2020). The exponential increase in health deficits suggests that biological aging is a self-productive process, where the development of new health deficits depends positively on the number of already existing ones. The self-productivity of health deficits can be explained by biological mechanisms at the cellular level (Belikov, 2019) and has received a scientific basis in reliability theory and in network theories of aging (Gavrilov and Gavrilova, 1991; Rutenberg et al., 2018).

According to the American Federation for Aging Research (2016, p. 2), a true biomarker of aging "must predict a person's physiological, cognitive, and physical function in an age-related way. In other words, it must predict the future onset of age-related conditions and diseases, and do so independently of chronological age." In practice, this criterion is assessed by the predictive power of biological age for mortality in regressions that control for chronological age (e.g. Levine, 2013; Mitnitski et al., 2013; Lu et al., 2019). The question is therefore whether knowing people's biological age provides important information about health and mortality beyond chronological age.

The predictive power of the frailty index for mortality has been demonstrated in many studies (e.g. Mitnitski et al., 2002a,b; Rockwood and Mitnitski, 2007; Mitnitski et al., 2013; Hosseini et al., 2022). A couple of recent studies compared the predictive power of alternative measures of biological age. In studies in which the frailty index was included, it was always found to be a significant predictor of mortality while telomere length, and some epigenetic clocks based on DNA methylation turned out to be insignificant (Kim et al., 2017; Li et al., 2020). The offered explanation is that insignificant regressors such as telomere length and the DNAmAge epigenetic clock are "too good" predictors of chronological age, i.e. their inclusion in the regression adds little further information beyond chronological age. Another advantage of the frailty index is that the pathway by which physiological function declines and the probability of death increases is obvious, while scientists have little idea about the mechanism by which telomere length or DNA methylation affect health deterioration and death (Jylävä et al., 2017).

So far, the computation of biological age was applied exclusively to individuals. Here, we extend the methodology and compute the biological age of populations at the global level. We build on Dalgaard et al. (2022) who constructed the frailty index from disease prevalence data from the Global Burden of Disease (GBD) study (Vos et al., 2020). Using data for 200 countries every fifth year for the period 1990–2019 and 5-year age groups from ages 30-34 to 80-84, we constructed a panel data set of the frailty index for populations at the country-year-sex-age-group level. We then proceeded as outlined above to estimate biological ages worldwide. We first regressed the frailty index on age, separately for men and women and by controlling for country and year fixed effects. We then used the estimated coefficients and the observed frailty index to compute for any given chronological age the biological age. This procedure generated for each age group and sex an "average world citizen" for whom chronological age and biological age coincide. It provides us with two dimensions through which we examine the global distribution of biological age: across countries and over time. In other words, we identify countries where people are relatively young or old given their chronological age and countries where people are aging particularly fast or slow.

Briefly, our results can be summarized as follows. Biological age varies substantially across the world. For example, the biological age of 65-year-old men ranges from 61 years to 74 years.[1]

[1]As mentioned, we use 5-year age groups and for simplicity we refer to the group by the youngest age in the group, such age 65 is the age group 65-69.

On average, the biological age for given chronological age group is increasing, and this trend is being driven by the chronologically older age groups. In rich countries (most of Europe, North America, Australia) people are biologically relatively old, but from 1990 to 2019 they became biologically younger by up to 2.5 years. The opposite is true in Africa: people are relatively young, biologically speaking, but age faster than in the rest of the world. Overall, we therefore observe conditional convergence (β–convergence) of biological age across the world. In absolute terms, we only observe a very slight (σ–) convergence, i.e. almost no change in the inequality of biological age distribution around the world.

Our paper is broadly related to studies of inequality and convergence in the global distribution of health and life expectancy. While many studies observed global convergence of life expectancy at birth, Becker et al. (2005) showed that convergence is mainly driven by declining inequality in mortality from communicable diseases while mortality from aging-related chronic diseases contributed to rising health inequality across countries. Edwards (2011) showed that strong global convergence in life expectancy at birth is accompanied by (slight) divergence in life expectancy at age 10. Aksan and Chakraborty (2023) decomposed life expectancy at birth into survival gains from specific age groups and showed that the age group 0–20 contributed 84% to the convergence of life expectancy at birth, while the age group 20–65 contributed to 29% and the age group 65+ contributed -13%. Bloom and Canning (2007) argued that the convergence process is more complex and showed the existence of two convergence clubs consisting of a group of countries with initially relatively high life expectancy at birth and strong convergence and a group of countries with initially low life expectancy and insignificant convergence.

Similarly to our approach, Chang et al. (2019) utilized GBD data to calculate an index of aging-related diseases, but focused on different outcomes. Specifically, they calculated the age-related disease burden for all countries and showed that it fell 23% worldwide from 1990 to 2017, with the greatest declines in high-income countries and smaller declines in Africa and Central and Eastern Europe. A decomposition analysis showed that the largest contributors to these trends were the increasing size of the adult population (leading to an increasing disease burden) and the decreasing case-fatality rate (leading to a declining disease burden). Prevalence rates (which form the basis for calculating biological age in our study) had a minimal and mostly negative impact on the age-related burden of disease, with the exception of Southeast Asia,

East Asia and Oceania, where the contribution was positive and large (17%). Trends in the worldwide distribution of age-related disease burden were not explicitly addressed.

The remainder of the paper is structured as follows. In Section 2 we introduce the data and explain the construction of the frailty index and biological age. The results are presented in Section 3. Section 4 discusses the results and concludes the paper.

2. Methodology and Empirical Model

2.1. **Data and Construction of the Frailty Index.** The national frailty (or deficit) index is constructed following the methodology of Dalgaard et al. (2022). Based on list of N health deficits, the frailty index of individual i from country c at time t is defined as:

$$D_{ict} = \frac{1}{N} \sum_{d=1}^{N} \mathbf{1}_{ict}(d), \tag{1}$$

where $\mathbf{1}_{ic}(d)$ is an indicator function that equals 1 if individual i has health deficit d. The individual deficits are included in the index without weights. While weighting can enhance the index's performance in specific samples, such as improving mortality prediction, unweighted indices are favored in the literature. This preference arises because weighting reduces generalizability, which is considered more valuable than higher predictive accuracy within a given sample (Rockwood and Mitnitski, 2007). Studies comparing weighted and unweighted indices have shown that weighting offers slight improvements in mortality prediction, but these gains are not significant enough to justify sacrificing generalizability (Theou et al., 2013).

For a population of size P_{ct} in country c, with individuals indexed by i, the average frailty index of the population, denoted as D_{ct} is then determined by:

$$D_{ct} = \frac{1}{P_{ct}} \sum_{i}^{P_{ct}} D_{ict}. \tag{2}$$

Using (1), this expression can be written as:

$$D_{ct} = \frac{1}{N} \sum_{d=1}^{N} \frac{P_{dct}}{P_{ct}}, \tag{3}$$

where P_{dct} represents the number of individuals in country c who suffer from deficit d. The aggregate frailty index of a country is therefore calculated as the average of N prevalence rates,

P_{dct}/P_{ct}. Similarly, the frailty index for an age group a in country c is determined as:

$$D_{act} = \frac{1}{N} \sum_{d=1}^{N} \frac{P_{dact}}{P_{act}}, \tag{4}$$

in which P_{dact}/P_{act} is the prevalence rate of health deficit d within age group a in country c at time t. Our empirical analysis is based on the age-group-specific frailty index in equation (4), which we compute separately for men and women.

We used disease prevalence rates from the Global Burden of Disease Study (GBD 2019; Vos et al., 2020) for the period 1990 to 2019 to construct the frailty index for the population between ages 30 and 90, stratified by 5-year age groups. The selection of prevalence rates followed the methodology of Searle et al. (2008) to select health deficits: they should generally increase with age, be related to overall health status, and should not be saturated too early and cover a range of organ systems. As in Dalgaard et al. (2022), the selection process resulted in a frailty index composed of 32 diseases for which global prevalence data are available. The list is shown in the Appendix.

Since the frailty index constructed from GBD data is based solely on aging-related diseases, there may be concerns that it differs methodologically from "conventional" frailty indices, which are typically constructed for individuals and incorporate both aging-related diseases and functional limitations. A recent study by O'Donovan et al. (2023) addresses these concerns. The authors analyze individual data from 15 countries using the SHARE database (Boersch-Supan et al., 2013) and construct two frailty indices: one based on 20 aging-related diseases for which corresponding prevalence rates are included in the GDB data set (the GBD-FI) and a conventional frailty index consisting of 70 items, comprising diseases, symptoms, and functional limitations. Their findings reveal a strong correlation between the two indices ($r = 0.67$ at the individual level and $r = 0.95$ or higher at the population level). They also demonstrate that both indices show a similar rate of increase in health deficits with age (0.030 vs. 0.036 per year), exhibit comparable distributions, and have similar predictive validity for mortality. The study concludes that the GBD-FI is a robust and externally valid measure to assess frailty across populations based on the deficit accumulation model (O'Donovan et al., p. 7).

In order to further corroborate the predictive quality of our GBD frailty index for mortality, we used data for overall mortality rates from UN (2024) and data for mortality from non-communicable diseases, which is also sourced from GBD (2019).

2.2. **Computation of Biological Age.** Following the methodology of Mitnitski et al. (2002a), we begin by estimating the log-linear association between chronological age CA and the frailty index D as:

$$\log D_{act} = \lambda + \mu \cdot CA_{act} + \phi_t + \phi_c + \epsilon_{ct}, \tag{5}$$

in which a is the age group, c is the country, t the year of observation, ϕ_t and ϕ_c's are period and country fixed effects and ϵ_{ct} is the error term. We estimate (5) separately for men and women because previous studies have shown that men and women age slightly differently. Women tend to have more health deficits for given age (higher λ) while men age faster (higher μ), see Mitnitski et al. (2002b), Abeliansky et al. (2020), Dalgaard et al. (2022).

We then used the estimated parameters and computed biological age by inverting equation (5):

$$BA_{act} = \frac{1}{\mu} \log \left(D_{act} - \lambda \right), \tag{6}$$

in which B_{act} is the biological age at chronological age a in country c and period t. For an intuitive interpretation, we can use the estimate $\log \widehat{D}_{act} = \lambda + \mu \cdot CA_{act}$ from equation (5) and eliminate λ in equation (6), which provides: $BA_{act} = CA_{act} + \log \left(D_{act} / \widehat{D_{act}} \right) / \mu$. By construction, $D_{act} = \widehat{D_{act}}$ for the average person at any given chronological age. Thus, biological age equals chronological age for the average person (Mitnitski and Rockwood, 2013). The equation also shows by how many years chronological age is modified for a given value of the individual frailty index. Since μ is about 3 percent (Mitnitski and Rockwood, 2013; Dalgaard et al., 2022; O'Donovan et al., 2023), a person who has, for example, 20 percent more health deficits than the average is estimated to be biologically 6 years older than the average ($\log(1.2)/0.03 = 6$). In other words, our estimates generate for each age group and sex an "average world citizen" for whom chronological age and biological age coincide. It provides us with two dimensions through which we examine the global distribution of biological age: across countries and over time.

In order to verify the predictive quality of the frailty index for mortality we estimated

$$\log m^j_{act} = \eta + \gamma \log D_{act} + \psi_t + \psi_c + \epsilon_{ct}, \quad j \in all, ncd, \tag{7}$$

where m^{all}_{act} denotes the all-cause mortality rate for age group a in country c and period t and m^{ncd}_{act} is the respective mortality rate for deaths from non-communicable diseases. Notice that

γ is the deficit elasticity of mortality, which shows the relative increase of mortality that is associated with a one percent increase of health deficits.

3. Results

3.1. **Frailty Index and Mortality.** We begin our analysis by demonstrating the predictive quality of the frailty index for mortality. Table A.1 in the Appendix shows the results from regressing (7) on all-cause mortality in the full sample. In columns (1) and (5), we see that health deficit accumulation explains more than 74 percent of the variance of mortality. Allowing for country and period fixed effects (as we will do in our biological age estimation), increases the R^2 to values above 0.95 with little change of the estimated age coefficients and constants. The deficit elasticity of mortality is about 3 percent and slightly larger for women than for men. The most interesting results with respect to the consideration of the frailty index for the computation of biological age are shown in columns (4) and (8). Following the methodology of biological age computation (as discussed in the Introduction), the frailty index is a useful health indicator for the computation of biological age if it contributes independently from chronological age to mortality. As shown in Table A.1, this is clearly the case. Controlling for chronological age, the frailty index remains a statistically and economically significant contributor to mortality.

Table A.2 in the Appendix shows results when the mortality regression including country and period fixed effects is carried out in samples split by continent. It shows that health deficits remain a very good predictor of mortality in all continents. A mild exception is Africa, where the R^2 drops to 0.94 for women and 0.90 for men. A potential explanation is that Africa is still in the midst of the epidemiological transition and aging-related (chronic) health deficits are an imperfect predictor of deaths from communicable diseases.

To explore this potential mechanism we repeated the exercise with the mortality rate from non-communicable diseases as the dependent variable. The results are shown in Panel B of Table A.2. We see that now the explained variation in mortality is as high in Africa as in the rest of the world. This is reassuring. Aging-related health deficits are globally an equally good predictor of aging-related deaths from non-communicable diseases, regardless of origin.

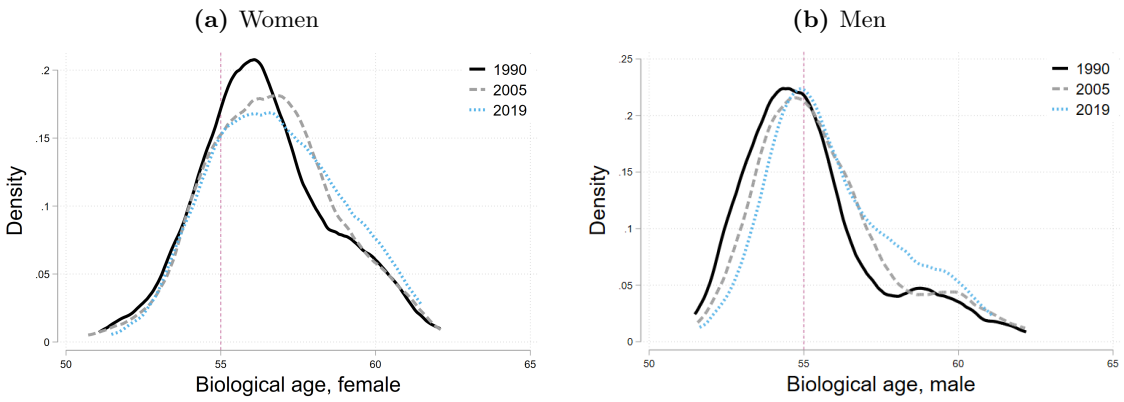
3.2. **The World Distribution of Biological Age: 1990–2019.** We next examined the distribution of biological age across the world and how it changed from 1990 to 2019. We first estimated equation (5) by sex, which provided the global estimates $\lambda_w = -3.970$ ($\pm 0.292 \times 10^{-2}$)

and $\mu_w = 0.0290$ ($\pm 0.510 \times 10^{-4}$) for women and $\lambda_m = -4.192$ ($\pm 0.328 \times 10^{-2}$) and $\mu_m = 0.0319$ ($\pm 0.574 \times 10^{-4}$) for men. We then fed the frailty index into equation (6) to predict biological age at the country-year-age-group level. In order to reduce the dimensionality for the presentation of results, we focus on 3 years (1990, 2005, and 2019) and 3 specific chronological ages: 55, 65, and 75.

The distribution for 55-year-olds is shown in Figure 1. There is a substantial spread of biological age at 55 across the world, ranging from about 52 to 62. The distribution is almost symmetrical and only slightly skewed to the right for men. Most importantly, the distributions are nearly stable over time, in particular those for men. This suggests that 55-year-olds around the world have neither gotten biologically older nor younger over time. Similar observations apply to people chronologically younger than 55 years (results available on request).

Figure 2 shows that these observations change significantly for 65-year-olds. The range of biologically ages becomes wider and the distributions more skewed to the right, in particular for men. Also, the distributions visibly shift to the right over time, in particular over the 1990-2005 period. On average, the 65-year-olds are about 3 years biologically older in 2019 than in 1990. Figure 3 shows that these trends also apply for the 75-year-olds where we now also observe a significant rightward shift of the distributions for the 2005–2019 period.

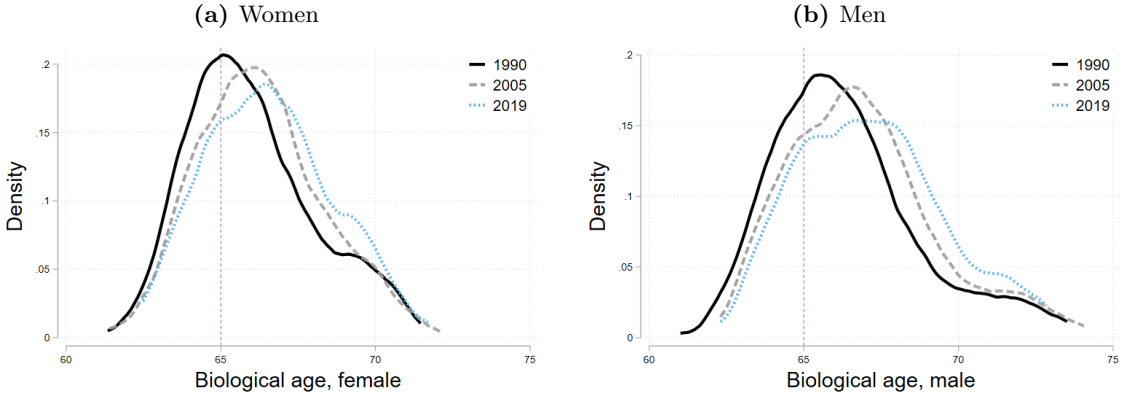
Figure 1. Distribution of Biological Age over Time: 55-year Olds



This figure shows the distribution of biological age for women (Panel A) and men (Panel B) at chronological age 55 for the years 1990, 2005, and 2019, using kernel density estimation. Notice that age 55 refers to age group 55-59.

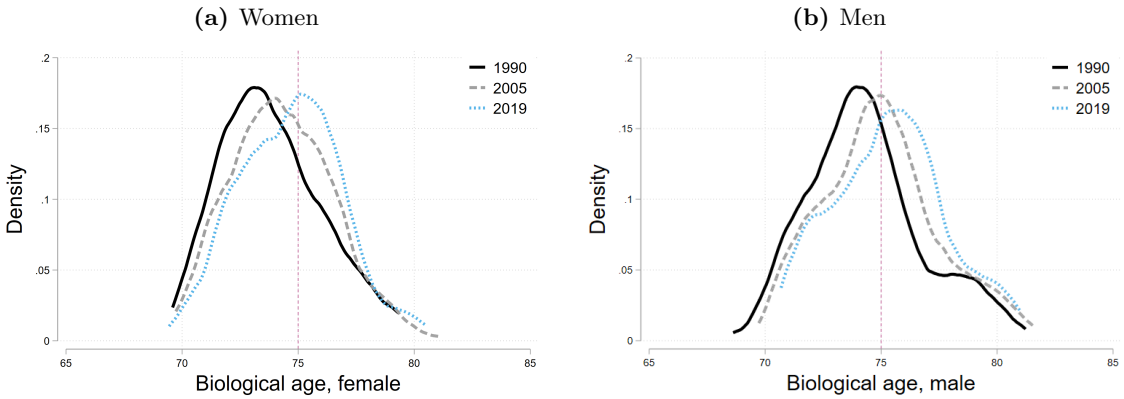
3.3. **Robustness Checks.** The conclusions have so far been drawn visually, by comparing distributions. We next report results of statistical tests to determine whether biological age

Figure 2. Distribution of Biological Age over Time: 65-year Olds



This figure shows the distribution of biological age for women (Panel A) and men (Panel B) at chronological age 65 for the years 1990, 2005, and 2019, using kernel density estimation. Notice that age 65 refers to age group 65-69.

Figure 3. Distribution of Biological Age over Time: 75-year Olds



This figure shows the distribution of biological age for women (Panel A) and men (Panel B) at chronological age 65 for the years 1990, 2005, and 2019, using kernel density estimation. Notice that age 75 refers to age group 75-79

has changed significantly over time. For that, we regressed biological age of our three main age groups (55-59, 65-69, 75-79) on year dummies, with 1990 as the omitted year. Results are shown in Appendix Table A.3 for females (Panel A) and males (Panel B). Columns (1)–(3) report estimates for the full sample. This statistical test suggests, for example, that 75-year-old men are, on average, biologically 1.02 years older than men of the same age in 1990, a difference that is statistically significant at the one-percent level (see Panel B in column (3)). The same pattern holds for all other years and age groups, as well as for women.

We next address the potential concern that these results are driven by a large number of small countries where the data might be unreliable. In columns (4)–(6), we exclude countries

in the first tercile of population size in 1990, meaning that countries with fewer than 900,000 inhabitants are not included in the sample. In columns (7)–(9), we raise this threshold to the median, which is close to 5 million. The overall patterns remain similar, although the effect of biological aging is less pronounced, particularly for 55-year-old women (see Panel B in columns (4) and (7)).

Until now, we have focused on unweighted averages of biological age. Next, we compare the results with and without population size weighting. When applying population weights (e.g., for ages 55–59, the population size of that age group serves as the weight), the unweighted averages reflect the biological age of "the average country", whereas the weighted averages represent the biological age of "the average world citizen". Appendix Figures A.1 and A.2 show the average biological age and its 95 percent confidence interval for our three baseline age groups and both genders. Results for unweighted averages reiterate the already documented fact: biological age increases from 1990 to 2019. In contrast, weighted averages are slightly higher in absolute terms, but do not show a clear time trend. This can be explained by a compositional effect: if population growth is higher in countries where people are initially biologically younger – as observed in our data – then the weighted biological age may remain stable or even decline, even as the unweighted biological age increases. The choice of the appropriate measure depends on the focus of the study. Since our research examines the biological age of specific age groups within countries, we prefer unweighted averages.

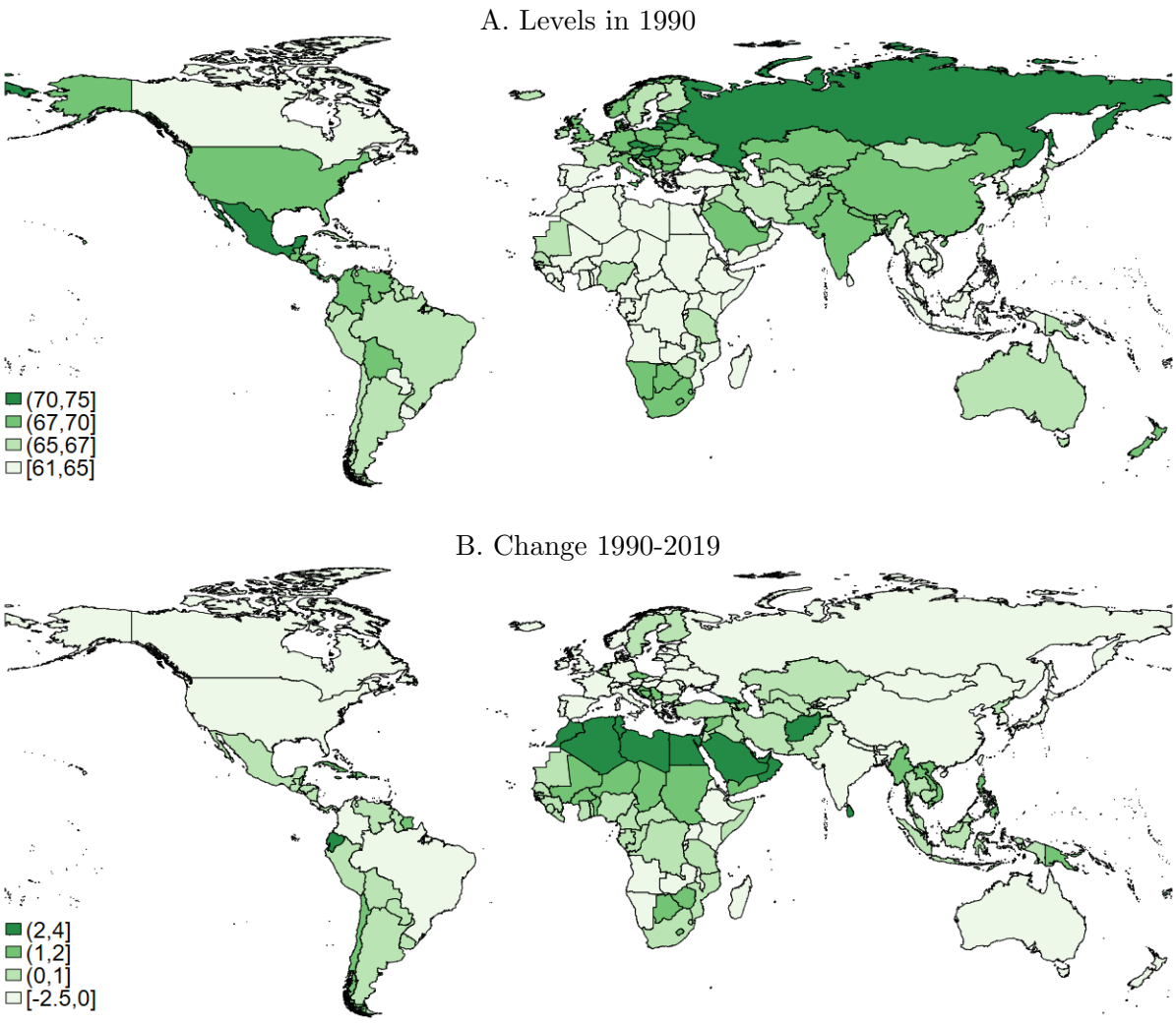
As outlined above, the selection of aging-related diseases included in the frailty index follows an established methodology, and previous research has shown that results remain robust as long as a sufficient number of conditions are considered (Searle et al., 2008; Theou et al., 2013). However, concerns may still remain as to whether our results are influenced by certain diseases included in the index. To address this, we conducted a robustness check by recalculating the frailty index while systematically excluding one item at a time. Appendix Figure A.3 shows that the average biological age for both men and women at chronological age 75 remains consistent in 1990 and 2019. This pattern holds for all other chronological ages, which are available upon request. These findings suggest that no single item in the frailty index is solely responsible for the observed variation in biological age.

3.4. **The Spatial Distribution of Biological Age.** Summarizing, old people becoming on average biologically older, i.e. less healthy around the world. We next explore in more detail the

driving forces behind the shift of the distribution. Figure 4 shows in Panel A the distribution of biological age of 65-year-old women in 1990. Panel B shows the respective change in biological age from 1990 to 2019. Figure 5 shows the same results for men.

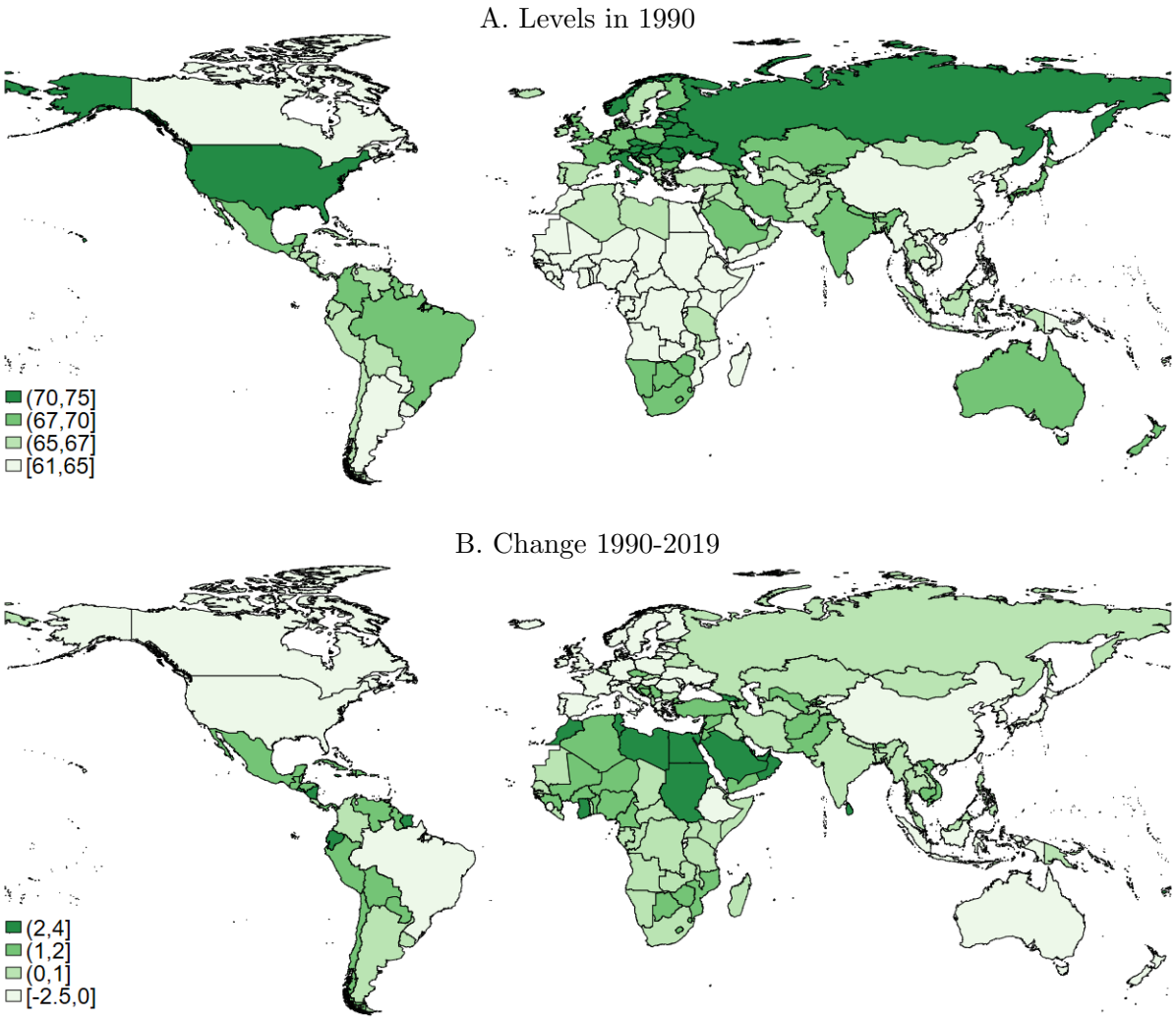
As a stylized fact, these maps show that in rich countries and many middle-income countries, 65-year-olds are biologically relatively old in 1990 and are getting biologically younger from 1990 to 2019. The opposite is true in low-income countries and especially in Africa where the 65-year-olds are relatively young in 1990 and are getting biologically older over the 1990-2019 period. There are also several outliers from the general rule. Most notably, Russian men who are were biologically very old in 1990 and still became older over the 1990-2019 period.

Figure 4. Spatial Distribution of Biological Age for chronological age 65: **Women**



This figure shows the biological age at chronological age 65 for women in 1990 (Panel A) and the corresponding absolute change in biological age from 1990 to 2019 (Panel B). Notice that age 65 refers to age group 65-69.

Figure 5. Spatial Distribution of Biological Age for chronological age 65: **Men**



This figure shows the biological age at chronological age 65 for men in 1990 (Panel A) and the corresponding absolute change in biological age from 1990 to 2019 (Panel B). Notice that age 65 refers to age group 65-69.

3.5. **Convergence and Inequality of Biological Age.** In this section we assess convergence and inequality by two metrics developed in growth economics, β–convergence and σ–convergence (Barro et al., 1991). Simply put, β convergence means that regions with initially low levels of income grow faster. Applied to our context, β convergence means that populations that start out relatively young age faster, in biological terms. Figure 6 shows for biological age at 65 in 1990 versus the change in biological age from 1990–2019. From visual inspection and the negative trend line, we conclude β–convergence: countries where 65-year-olds are initially young are aging faster in biological terms (similar figures can be drawn for other chronological ages). The β-coefficient for men is estimated at -0.139 ($\pm$0.054), indicating that one additional year of

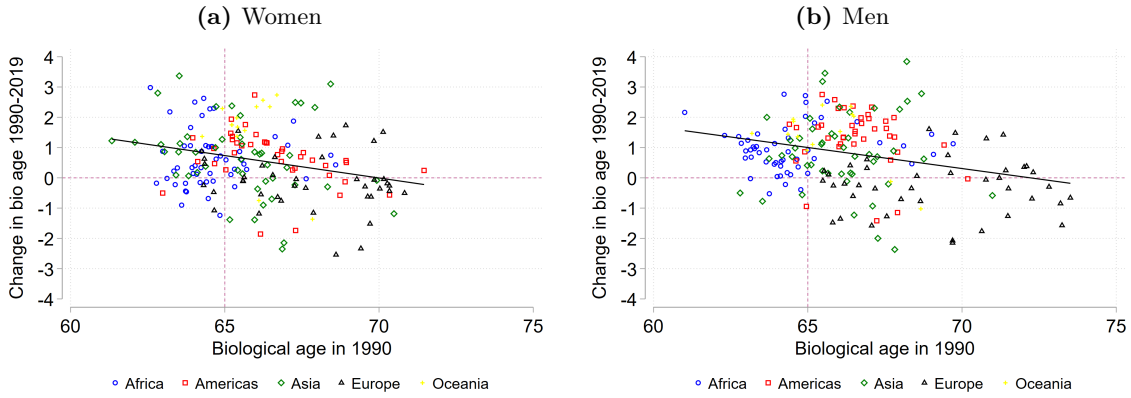
biological age in 1990 is associated with a reduction of 0.139 years in biological age over a period of 29 years. The corresponding estimate for women is -0.148 ($\pm$0.069). This suggests that the observed β-convergence is relatively slow. This can be further confirmed by translating these results into growth regressions, a common approach in the income growth literature. Here, we estimate annual convergence rates of approximately 0.5 percent per year. As already concluded from Figures 4 and 5, the convergence is largely driven by the (mostly European) rich countries, where relatively old people are getting younger, and the African countries, where relatively young people are getting older. Similar convergence results are obtained for the 55-year-olds and 75-year-olds (see Figures A.4 and A.5 in the Appendix) and for other ages (available on request).

The observation of β–convergence, although slow, seemingly suggests a decline in the world inequality of biological age. This, however, is not the case. Figure 7 considers σ–convergence by plotting the coefficient of variation in biological age at 65 over time. We see a very mild trend of declining inequality for men, but mostly the figure conveys the impression of constant inequality of biological age (similar figures can be drawn for other chronological ages). In order to understand the result, notice that β–convergence implies σ–convergence only if the countries share a common steady state for biological age. This is clearly not the case. Diagrammatically, Figure 6 seemingly suggests a steady state at about ages 70 for women and 72 for men, i.e. where the trend line hits zero, implying no change in biological age. However, most countries that are getting younger are situated at ages below 70 or 72, implying that they are moving away from the zero-change line.

4. Discussion and Conclusion

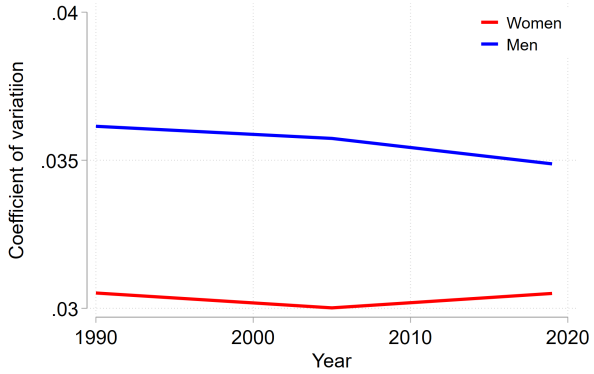
In this paper, we used an established metric from gerontology, the frailty index, to assess health and aging around the world. We used GBD data to compute the frailty index by sex-year-age-groups of countries from which we then inferred the biological age of the population of the world. We found that, on average, people became biologically older for given chronological age, and that these trends were concentrated among the chronologically old people. These trends were driven primarily, but not exclusively, by African countries where biologically relatively young people became older. In rich countries, in contrast, biologically relatively old people became younger by up to two years in the period 1990–2019.

Figure 6. β–**Convergence in Biological Age for chronological age 65**



This figure shows the relationship between biological age in 1990 and the absolute change in biological age from 1990 to 2019. The colors indicate the geographical locations of the observation in terms of continent. The red vertical dashed marks the difference between decreases and increases in biological age, while the red dashed horizontal line indicates that we are looking at chronological age 65. Notice that age 65 refers to age group 65-69.

Figure 7. σ–**Convergence in Biological Age for chronological age 65**



This figure shows the coefficient of variation of biological age at chronological age 65 from 1990 to 2019. Notice that age 65 refers to age group 65-69.

A potential explanation for these trends can be derived from the epidemiological transition and trends in medical progress. Many African countries are still in the midst of the epidemiological transition and a large share of deaths are due to malnutrition and communicable diseases. In indirect support of this mechanism, we showed in the Appendix that the frailty index explains deaths from all causes somewhat less well in Africa than in the rest of the world while deaths from non-communicable diseases are about equally well explained everywhere in the world. A consequence of high exposure to communicable diseases is likely a selection- or survivorship effect: the survivors of the communicable diseases are relatively healthy, i.e., biologically young people.

With ongoing epidemiological transition the survivor effect vanishes, implying that we see it in particular in old people who had a greater lifetime exposure to communicable diseases. This is what we observe when we compare the African countries (identified by blue circles in Figure 6 and in Figure A.1 and A.2 in the Appendix). At age 75, Africans are biologically up to 7 years younger than the world average. At age 55, this biological age advantage is much smaller and for women no longer visually discernable. With the ongoing epidemiological transition, later-born generations experienced lower lifetime disease exposure at any given age. As the survivorship effect diminishes, a greater number of relatively frail individuals survive, leading to a biologically older population – an effect observed primarily among those who are chronologically older (see Krenz and Strulik, 2023, for a theoretical model and supporting evidence for the survivorship effect of the epidemiological transition in India).

An alternative explanation for the increasing biological age in Africa and other low-income regions could be measurement error. A well-known limitation of the GBD study is that, in poorer countries, data often come from small, non-representative samples, and the prevalence of non-communicable diseases may be underestimated due to limited diagnostic capacity. To address missing or incomplete data, GBD relies on statistical models to estimate disease prevalence and mortality. This could result in a systematic underestimation of aging-related diseases in low-income countries, leading to an underestimation of their biological age. If this measurement error declines over time, it may falsely suggest that people are becoming biologically older. However, the GBD study provides no evidence of a systematic measurement error that would misrepresent actual health trends.

Another possible explanation for the rise in biological age is structural change and urbanization, which may have exposed later-born generations to increased pollution, stress, and unhealthy lifestyle changes, such as higher rates of smoking, alcohol consumption, and dietary shifts toward processed foods. However, this factor appears to be more relevant for the explanation of increasing biological age in Russia and other former USSR countries. The selection effect is less convincing for these countries, as biological age rose despite the relatively early completion of the second stage of the epidemiological transition in the mid-20th century.

In the rich countries, medical progress, increasing education and income, and the associated institutional and behavioral changes (e.g. better nutrition, less smoking, access to health care) are likely drivers of the decline of chronic diseases for given chronological age (Hall and Jones,

2007; Dalgaard and Strulik, 2014; Frankovic and Kuhn, 2019, 2023; Strulik, 2022). Our results are consistent with micro studies that found a persistent trend in declining health deficits for given age of elderly individuals in Europe and the U.S. (Abeliansky and Strulik, 2019; Abeliansky et al., 2020; Old and Scott, 2023). They are also consistent with Levine and Crimmins (2018) who showed in a study based on biomarkers that biological age declined in the U.S. over the period 1988-2010 with the greatest improvements for chronologically old people.

Biological age offers a novel approach to measuring global health, fundamentally differing from other metrics like Disability-Adjusted Life Years (DALYs) and Health-Adjusted Life Expectancy (HALE) even when these indicators are based exclusively on non-communicable (i.e. aging-related) diseases. While biological age evaluates the average health of specific groups of individuals that are alive at a particular point in time (e.g., 60-year-old American men in the year 2000), DALYs and HALE are population-specific indicators of healthy survival prospects. To illustrate the difference, imagine a population where half are affected by a chronic disease. Initially, individuals with the disease die at age 65, but in the new scenario, they survive until age 70. As a result, the biological age of the 65-69 age group would clearly increase. However, since life expectancy has also risen, DALYs for this age group could decrease, increase, or remain unchanged, depending on whether the gains in life expectancy outweigh the additional Years Lived with Disability (YLD) due to the higher disease prevalence in this age group. This distinction clarifies why we observe an increase in the biological age of elderly individuals in some regions, such as Northern Africa and South Asia, for which other studies report no increase or even a decline in DALYs due to non-communicable diseases (e.g., Chang et al., 2019, Ferrari et al., 2024).

The findings of this paper are important for understanding global health inequalities, aging trends, and the effects of public health policies. A recent review has highlighted the limitations of relying solely on chronological age in economic modeling and policymaking and argued that incorporating biological age measures can provide more precise insights for shaping policies on labor markets, pensions, and healthcare systems (Kotschy et al., 2025). Building on these insights, our study is the first to calculate biological age on a global scale. Our findings underscore the need for health and labor market policies to account for biological age alongside chronological age when evaluating population health. For instance, a more equitable public

pension system should integrate mechanisms that reflect biological age rather than relying on a fixed chronological threshold (see Grossmann et al., 2024 for recent proposals on this approach).

Biological age serves as a valuable complement to other aggregate indicators of population health. Instead of focusing solely on healthy survival prospects, such as DALYs and HALE, it offers a clear, group-specific measure of the health status of the living population at a given time. The fact that the biological age of, say, 70-year-olds can worsen even as their healthy life expectancy remains stable or improves, reveals a distinct target for policy interventions that might be obscured by metrics that merge health indicators with life expectancy measures. More generally, the fact that biological aging is modifiable highlights the potential for policies to slow its progression. This suggests that emphasizing preventive healthcare and healthy aging initiatives over reactive treatments could be a more effective approach for improving public health outcomes.

Our study has several limitations. The macro analysis based on prevalence rates prevents drawing conclusions about the distribution of biological age within age groups within countries. A decomposition analysis of global lifespan inequality showed that it is mostly driven by within-country inequality (Permeyer and Scholl, 2019). Likewise, Chang et al. (2019) showed that the evolution of age-related disease burden is predominantly explained by demographic changes in age structure and changes in disease-specific mortality rates. However, we found that a significant variation in biological age persists at the country-year-age-group level, which is intriguing to compare across countries and over time. We share with Chang et al. (2019) the limitations of the original GBD-study. Observed data may be sparse and subject to measurement error, in particular for low- and middle-income countries and estimated data are subject to uncertainty and potential bias (for details, see Vos et al., 2020). Finally, we cannot draw conclusions about future distributions of biological age around the world. This is not only because our analysis indicates the absence of a common steady state for biological age, but also because the long-term effects of recent health trends, such as the opioid crisis (Case and Deaton, 2019) and the Covid-19 pandemic (Strulik and Grossmann, 2024) and their impact on the accumulation of chronic health deficits are not yet reflected in the aging-related health deficit data of the GBD study.

References

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

Abeliansky, A. L., 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.

Aksan, A. M., and Chakraborty, S. (2023). Life expectancy across countries: Convergence, divergence and fluctuations. *World Development* 168, 106263.

American Federation for Aging Research (2016). Biomarkers of Aging. `https://www.afar.org/imported/AFAR_BIOMARKERS_OF_AGING_2016.pdf`

Argentieri, M. A., Xiao, S., Bennett, D., Winchester, L., Nevado-Holgado, A. J., Ghose, U., ... and van Duijn, C. M. (2024). Proteomic aging clock predicts mortality and risk of common age-related diseases in diverse populations. *Nature Medicine*, 1-11.

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

Barro, R. J., Sala-i-Martin, X., Blanchard, O. J., and Hall, R. E. (1991). Convergence across states and regions. *Brookings Papers on Economic Activity* Vol. 1991(1), 107-182.

Becker, G. S., Philipson, T. J., and Soares, R. R. (2005). The quantity and quality of life and the evolution of world inequality. *American Economic Review* 95(1), 277-291.

Belikov, A. V. (2019). Age-related diseases as vicious cycles. *Ageing Research Reviews* 49, 11-26.

Bloom, D. E., and Canning, D. (2007). Mortality traps and the dynamics of health transitions. *Proceedings of the National Academy of Sciences* 104(41), 16044-16049.

Boersch-Supan, A., Brandt, M., Hunkler, C., Kneip, T., Korbmacher, J., Malter, F., ... and Zuber, S. (2013). Data resource profile: the Survey of Health, Ageing and Retirement in Europe (SHARE). *International Journal of Epidemiology* 42(4), 992-1001.

Case, A., and Deaton, A. (2017). Mortality and morbidity in the 21st century. *Brookings Papers on Economic Activity* 2017 Spring; 2017: 397-476.

Chang, A. Y., Skirbekk, V. F., Tyrovolas, S., Kassebaum, N. J., and Dieleman, J. L. (2019). Measuring population ageing: an analysis of the Global Burden of Disease Study 2017. *The Lancet Public Health* 4(3), e159-e167.

Dalgaard, C. J., and Strulik, H. (2014). Optimal aging and death: understanding the Preston curve. *Journal of the European Economic Association* 12(3): 672–701.

Dalgaard, C.-J., Hansen, C., and Strulik, H. (2022). Physiological aging around the world. *PLOS ONE* 17(6), e0268276.

Edwards, R. D. (2011). Changes in world inequality in length of life: 1970–2000. *Population and Development Review* 37(3), 499-528.

Ferrari, A. J., Santomauro, D. F., Aali, A., Abate, Y. H., Abbafati, C., Abbastabar, H., ... and Murray, C.J.L. (2024). Global incidence, prevalence, years lived with disability (YLDs), disability-adjusted life-years (DALYs), and healthy life expectancy (HALE) for 371 diseases and injuries in 204 countries and territories and 811 subnational locations, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. The Lancet, 403(10440), 2133-2161.

Frankovic, I., and Kuhn, M. (2019). Access to health care, medical progress and the emergence of the longevity gap: A general equilibrium analysis. *Journal of the Economics of Ageing* 14, 100188.

Frankovic, I., and Kuhn, M. (2023). Health insurance, endogenous medical progress, health expenditure growth, and welfare. *Journal of Health Economics* 87, 102717.

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

GBD (2019). Global Burden of Disease Study. `https://www.healthdata.org/research-analysis/gbd`

Gompertz, B. (1825). On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies, *Philosophical Transactions of the Royal Society of London* 115: 513–583.

Grossmann, V., Schünemann, J., and Strulik, H. (2024). Fair Pension Policies with Occupation-Specific Ageing. *Economic Journal* 134, 2835-2875.

Hall, R. E., and Jones, C. I. (2007). The value of life and the rise in health spending. *Quarterly Journal of Economics* 122(1), 39-72.

Hosseini, R., Kopecky, K. A., and Zhao, K. (2022). The Evolution of Health over the Life Cycle. *Review of Economic Dynamics* 45, 237-263.

Hansen, C.W., Dalgaard, C.-J. and Strulik, H. (2023). Life-Cycle labor supply and physiological aging across countries. *PLOS ONE* 18(11): e0294952.

Howlett, S. E., Rutenberg, A. D., and Rockwood, K. (2021). The degree of frailty as a translational measure of health in aging. *Nature Aging* 1(8), 651-665.

HMD (2024). Human Mortality Database. Max Planck Institute for Demographic Research (Germany), University of California, Berkeley (USA), and French Institute for Demographic Studies (France). Available at www.mortality.org (data downloaded on January 2024).

Jia, L., Zhang, W., and Chen, X. (2017). Common methods of biological age estimation. *Clinical Interventions in Aging* 12, 759-772.

Kim, S., Myers, L., Wyckoff, J., Cherry, K. E., and Jazwinski, S. M. (2017). The frailty index outperforms DNA methylation age and its derivatives as an indicator of biological age. *Geroscience* 39, 83-92.

Hamczyk, M. R., Nevado, R. M., Barettino, A., Fuster, V., and Andres, V. (2020). Biological versus chronological aging: JACC focus seminar. *Journal of the American College of Cardiology* 75(8), 919-930.

Kotschy, R., Bloom, D. E., and Scott, A. J. (2025). On the Limits of Chronological Age. *Annual Review of Economics*, forthcoming

Krenz, A., and Strulik, H. (2023). Physiological aging in India: The role of the epidemiological transition. *PLoS One* 18(7), e0287259.

Levine, M. E., and Crimmins, E. M. (2018). Is 60 the new 50? Examining changes in biological age over the past two decades. *Demography* 55, 387-402.

Li, X., Ploner, A., Wang, Y., Magnusson, P. K., Reynolds, C., Finkel, D., ... and Haegg, S. (2020). Longitudinal trajectories, correlations and mortality associations of nine biological ages across 20-years follow-up. *Elife* 9, e51507.

Lu, A. T., Quach, A., Wilson, J. G., Reiner, A. P., Aviv, A., Raj, K., ... and Horvath, S. (2019). DNA methylation GrimAge strongly predicts lifespan and healthspan. *Aging* 11(2), 303.

Ledberg, A. (2020). Exponential increase in mortality with age is a generic property of a simple model system of damage accumulation and death. *PloS One* 15(6), e0233384.

Levine, M. E. (2013). Modeling the rate of senescence: can estimated biological age predict mortality more accurately than chronological age?. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences* 68(6), 667-674.

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., Graham, J. E., Mogilner, A. J., and Rockwood, K. (2002a). Frailty, fitness and late-life mortality in relation to chronological and biological age. *BMC Geriatrics* 2(1), 1-8.

Mitnitski, A. B., Mogilner, A. J., MacKnight, C., and Rockwood, K. (2002b). The accumulation of deficits with age and possible invariants of aging. *The Scientific World Journal* 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.

O'Donovan, M. R., Devleesschauwer, B., Sezgin, D., Liew, A., Kabir, Z., and O'Caoimh, R. (2023). Comparing frailty prevalence between countries: validation of the Global Burden

of Disease study Frailty Index (GBD-FI) in the survey of health, ageing and retirement in Europe. Age and Ageing, 52(11), afad214.

Old, J., and Scott, A. (2023). Healthy ageing trends in England between 2002 to 2018: Improving but slowing and unequal. *Journal of the Economics of Ageing* 26, 100470.

Olshansky, S. J., and Carnes, B. A. (1997). Ever since Gompertz. *Demography* 34(1): 1–15.

Permanyer, I., and Scholl, N. (2019). Global trends in lifespan inequality: 1950-2015. *PloS one* 14(5), e0215742.

Rockwood, K., and Mitnitski, A. (2007). Frailty in relation to the accumulation of deficits. *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.

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.

Sprott, R. L. (2010). Biomarkers of aging and disease: introduction and definitions. *Experimental Gerontology*, 45(1), 2-4.

Strulik, H. (2022). Medical progress and life cycle choices. *Journal of the Economics of Ageing* 23, 100415.

Strulik, H., and Grossmann, V. (2024). Beyond Covid: Pandemics and the Economics of Aging and Longevity CESifo Discussion Paper No. 11104.

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.

Theou, O., Brothers, T. D., Pena, F. G., Mitnitski, A., and Rockwood, K. (2014). Identifying common characteristics of frailty across seven scales. *Journal of the American Geriatrics Society* 62(5), 901-906.

Tian, Y. E., Cropley, V., Maier, A. B., Lautenschlager, N. T., Breakspear, M., and Zalesky, A. (2023). Heterogeneous aging across multiple organ systems and prediction of chronic disease and mortality. *Nature Medicine* 29(5), 1221-1231.

UN (2024). World Population Projections. `https://population.un.org/wpp/`.

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.

Appendix A.1: 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.

Appendix A.2: Additional Results

Table A.1: Estimated Frailty-Mortality Relationship

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
deficits	2.439***	2.473***	2.477***	2.941***	2.797***	2.874***	2.877***	2.060***
	(0.0244)	(0.0241)	(0.0242)	(0.251)	(0.0328)	(0.0313)	(0.0314)	(0.290)
Constant	1.787***	1.869***	1.880***	3.010***	2.040***	2.222***	2.229***	0.284
	(0.0457)	(0.0587)	(0.0590)	(0.612)	(0.0482)	(0.0746)	(0.0748)	(0.690)
R^2	0.816	0.944	0.958	0.969	0.747	0.932	0.944	0.967
Country FE	no	yes	yes	yes	no	yes	yes	yes
Period FE	no	no	yes	yes	no	no	yes	yes
Age FE	no	no	no	yes	no	no	no	yes
Sex	Women	Women	Women	Women	Men	Men	Men	Men
Obs	15400	15400	15400	15400	15400	15400	15400	15400
Countries	200	200	200	200	200	200	200	200

This table reports the estimated coefficients from regressing the logged all-cause age-specific mortality rate on the logged deficit rate by sex (women in columns 1-4 and men in columns 5-8). The fixed effects included are indicated in the bottom rows. Robust standard errors clustered at the country in parenthesis. ***,**,* significant at, respectively, the 10, 5, and 1 percent level.

Table A.2: Frailty-Mortality Relationship Across Continents

Panel A: All-cause age-specific mortality rates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
deficits	2.477***	2.285***	2.358***	2.573***	2.653***	2.513***	2.877***	2.394***	2.857***	3.007***	3.346***	2.911***
	(0.0242)	(0.0517)	(0.0413)	(0.0457)	(0.0497)	(0.0427)	(0.0314)	(0.0581)	(0.0369)	(0.0468)	(0.0348)	(0.0618)
Constant	1.880***	1.879***	1.508***	2.067***	1.787***	2.034***	2.229***	1.788***	2.031***	2.487***	2.574***	2.345***
	(0.0590)	(0.127)	(0.101)	(0.112)	(0.118)	(0.104)	(0.0748)	(0.140)	(0.0876)	(0.112)	(0.0813)	(0.146)
R^2	0.958	0.937	0.962	0.958	0.971	0.986	0.944	0.901	0.958	0.955	0.977	0.965
Continent	all	Africa	Americas	Asia	Europe	Oceania	all	Africa	Americas	Asia	Europe	Oceania
sex	female	female	female	female	female	female	male	male	male	male	male	male
Obs	15400	4004	3003	3619	3157	1078	15400	4004	3003	3619	3157	1078
Countries	200	52	39	47	41	14	200	52	39	47	41	14

Panel B: Non-communicable age-specific mortality rates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
deficits	2.977***	3.163***	2.850***	2.971***	3.030***	2.664***	3.366***	3.425***	3.123***	3.397***	3.586***	3.146***
	(0.0199)	(0.0308)	(0.0436)	(0.0381)	(0.0372)	(0.0625)	(0.0188)	(0.0276)	(0.0322)	(0.0370)	(0.0291)	(0.0355)
Constant	2.636***	3.273***	2.176***	2.678***	2.444***	2.205***	3.023***	3.489***	2.338***	3.170***	3.004***	2.804***
	(0.0486)	(0.0757)	(0.106)	(0.0938)	(0.0888)	(0.152)	(0.0447)	(0.0665)	(0.0765)	(0.0888)	(0.0679)	(0.0840)
R^2	0.977	0.985	0.977	0.978	0.980	0.980	0.980	0.984	0.980	0.980	0.984	0.990
Continent	all	Africa	Americas	Asia	Europe	Oceania	all	Africa	Americas	Asia	Europe	Oceania
sex	female	female	female	female	female	female	male	male	male	female	male	male
Obs	15400	4004	3003	3619	3157	1078	15400	4004	3003	3619	3157	1078
Countries	200	52	39	47	41	14	200	52	39	47	41	14

This table reports the relationship between the logged deficit rate and the logged all-cause mortality rate (Panel A) and the logged non-communicable mortality rate for 5-year age groups from age 20-24 to 80-84. The table demonstrates that in terms of explanatory power and the estimated coefficient the deficit variable performs as well for African countries (columns 2 and 8) as for countries in other continents. Robust standard errors clustered at the country in parenthesis. ***,**,* significant at, respectively, the 10, 5, and 1 percent level.

Table A.3: Changes in average biological age from 1990

Panel A: Female

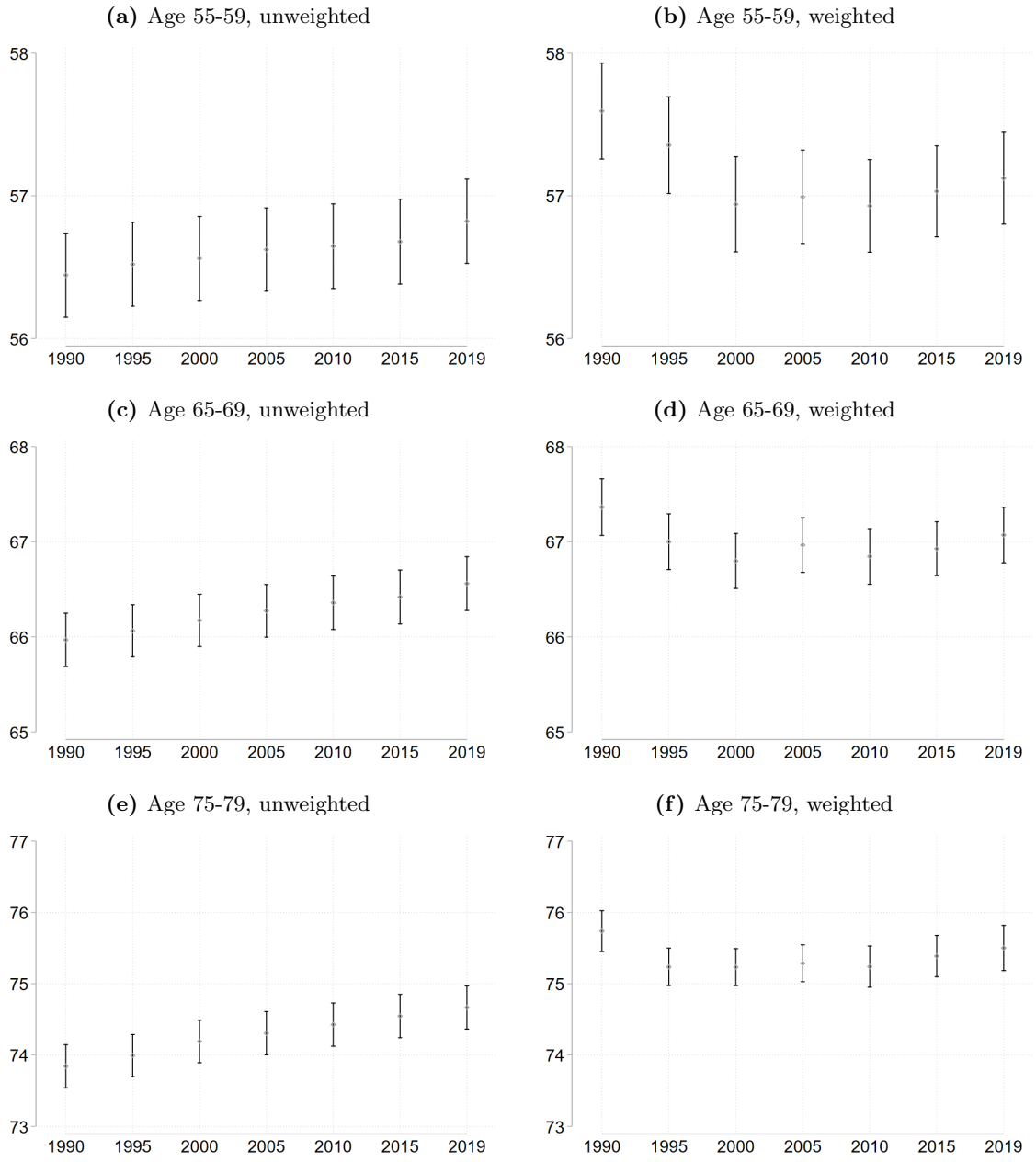
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
1995	0.077***	0.095***	0.150***	0.023	0.036	0.086***	0.019	0.019	0.081*
	(0.028)	(0.026)	(0.028)	(0.034)	(0.031)	(0.032)	(0.047)	(0.041)	(0.041)
2000	0.118**	0.205***	0.348***	-0.002	0.083	0.225***	-0.031	0.039	0.209***
	(0.045)	(0.047)	(0.048)	(0.051)	(0.052)	(0.055)	(0.067)	(0.064)	(0.067)
2005	0.179***	0.306***	0.464***	0.014	0.165**	0.342***	-0.005	0.120	0.319***
	(0.060)	(0.060)	(0.063)	(0.064)	(0.067)	(0.073)	(0.083)	(0.081)	(0.089)
2010	0.203***	0.391***	0.584***	0.026	0.229***	0.436***	-0.003	0.169*	0.398***
	(0.068)	(0.072)	(0.076)	(0.074)	(0.080)	(0.089)	(0.096)	(0.098)	(0.109)
2015	0.235***	0.451***	0.704***	0.047	0.293***	0.568***	0.030	0.247**	0.534***
	(0.073)	(0.077)	(0.084)	(0.078)	(0.087)	(0.099)	(0.102)	(0.109)	(0.124)
2019	0.378***	0.592***	0.824***	0.160**	0.403***	0.673***	0.125	0.351***	0.640***
	(0.076)	(0.080)	(0.085)	(0.078)	(0.088)	(0.100)	(0.101)	(0.110)	(0.126)
age	55-59	65-69	75-79	55-59	65-69	75-79	55-59	65-69	75-79
sample	full	full	full	w.o small	w.o small	w.o small	w.o median	w.o median	w.o median
sex	female	female	female	female	female	female	female	female	female
observations	1400	1400	1400	1050	1050	1050	700	700	700
countries	200	200	200	150	150	150	100	100	100

Panel B: Male

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
1995	0.131***	0.135***	0.153***	0.094***	0.094***	0.101***	0.092**	0.083**	0.091**
	(0.024)	(0.024)	(0.025)	(0.029)	(0.029)	(0.029)	(0.039)	(0.039)	(0.039)
2000	0.304***	0.340***	0.424***	0.207***	0.257***	0.334***	0.178***	0.223***	0.306***
	(0.039)	(0.040)	(0.040)	(0.043)	(0.044)	(0.044)	(0.056)	(0.056)	(0.057)
2005	0.440***	0.526***	0.642***	0.295***	0.400***	0.533***	0.276***	0.380***	0.507***
	(0.055)	(0.056)	(0.055)	(0.060)	(0.062)	(0.061)	(0.074)	(0.077)	(0.077)
2010	0.479***	0.568***	0.734***	0.307***	0.429***	0.612***	0.281***	0.392***	0.568***
	(0.067)	(0.068)	(0.067)	(0.075)	(0.079)	(0.078)	(0.093)	(0.098)	(0.099)
2015	0.576***	0.699***	0.878***	0.380***	0.535***	0.748***	0.358***	0.498***	0.697***
	(0.074)	(0.076)	(0.077)	(0.083)	(0.089)	(0.091)	(0.101)	(0.109)	(0.116)
2019	0.717***	0.811***	1.022***	0.497***	0.648***	0.907***	0.458***	0.584***	0.843***
	(0.078)	(0.082)	(0.084)	(0.086)	(0.097)	(0.101)	(0.103)	(0.118)	(0.127)
age	55-59	65-69	75-79	55-59	65-69	75-79	55-59	65-69	75-79
sample	full	full	full	w.o small	w.o small	w.o small	w.o median	w.o median	w.o median
sex	male	male	male	male	male	male	male	male	male
observations	1400	1400	1400	1050	1050	1050	700	700	700
countries	200	200	200	150	150	150	100	100	100

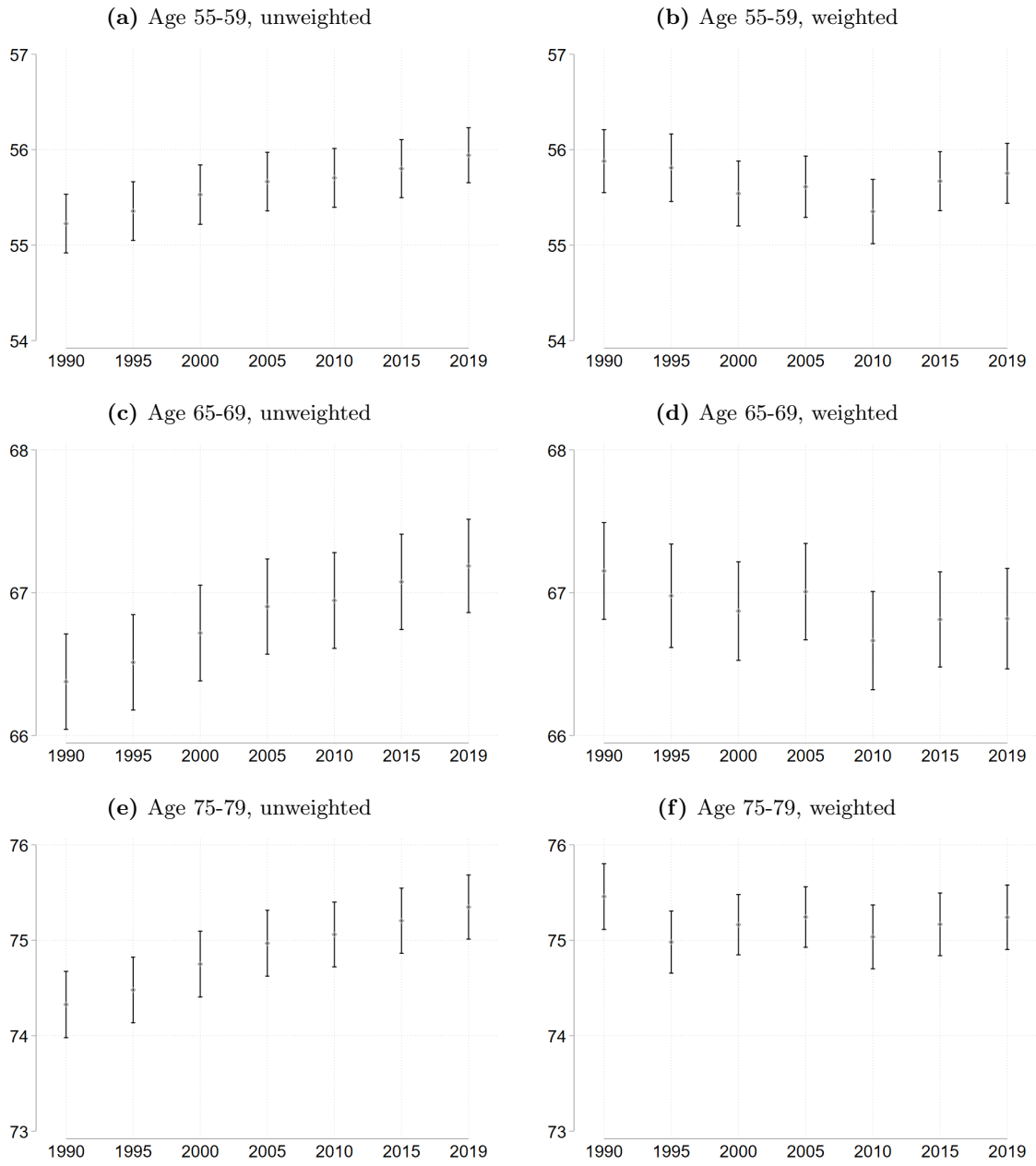
This table reports the change in average biological age from 1990 (the omitted comparison year in the regressions) to the year indicated, separately by sex: females in Panel A and males in Panel B. The specific age group (55-59, 65-69, 75-79) is indicated in the fifth row from the bottom. Columns 1?3 include the full sample. Columns 4-6 exclude the 10% smallest countries by population size in 1990 (i.e, "w.o small"), while Columns 7-9 exclude the 50% smallest countries (i.e, "w.o median"). Robust standard errors clustered at the country in parenthesis. ***,**,* significant at, respectively, the 10, 5, and 1 percent level.

Figure A.1 Average biological age for women, weighted and unweighted



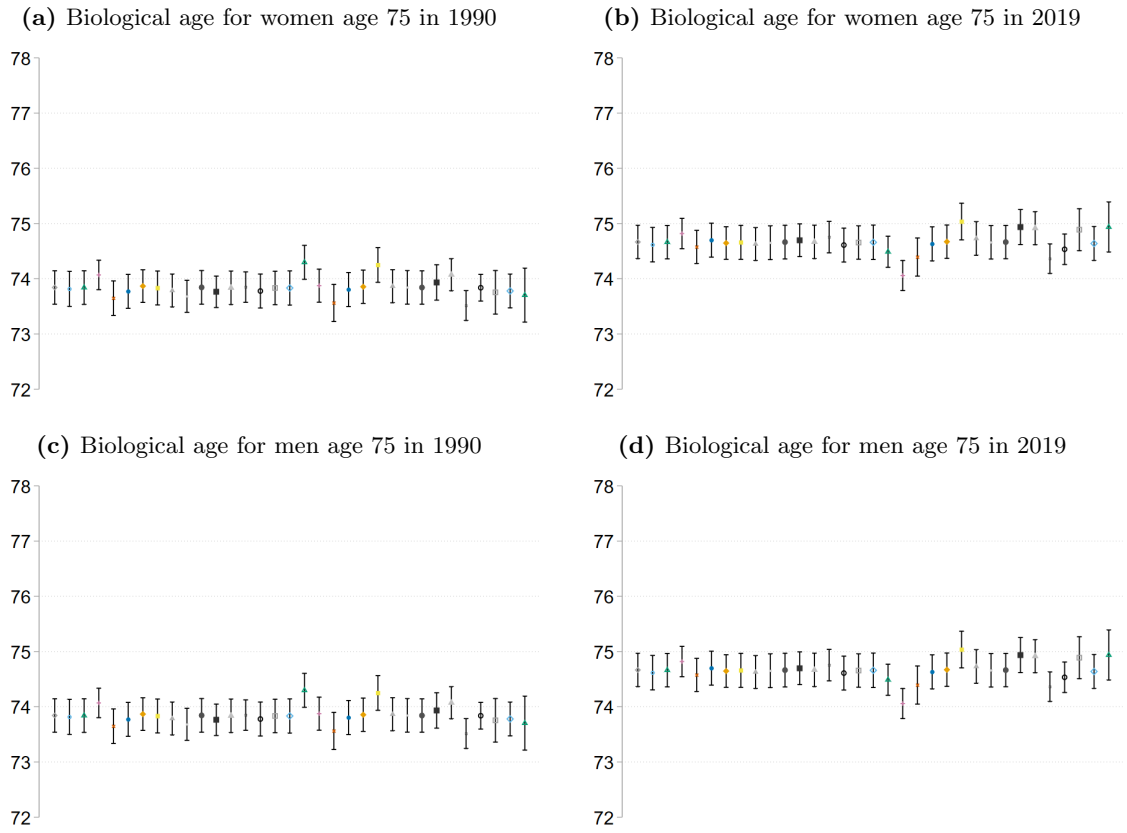
This figure shows the average biological age for women at ages 55, 65, and 75 from 1990 to 2010. The left-hand side figures are weighted by population size of the age groups, while the right-hand side figures are unweighted. Vertical lines indicate 95% confidence bands.

Figure A.2 Average Biological Age of Men: Unweighted (left) and Weighted (right)



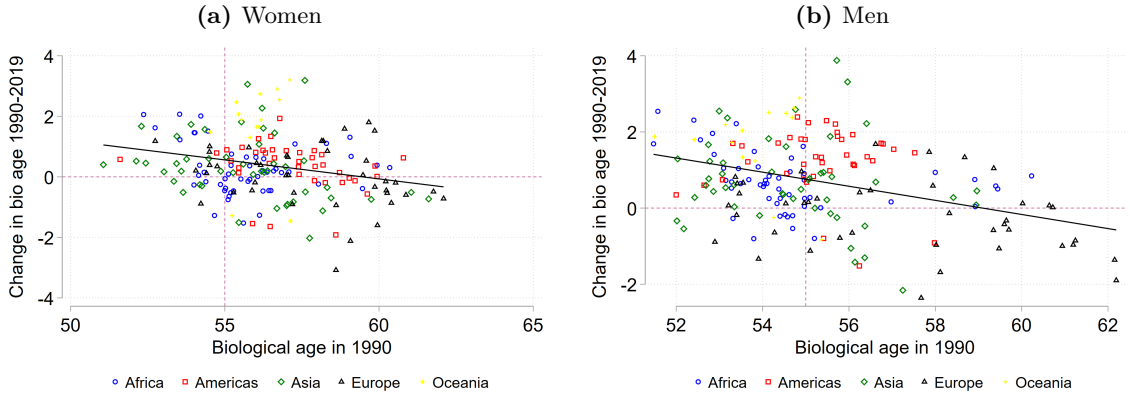
This figure shows the average biological age for men at ages 55, 65, and 75 from 1990 to 2010. The right-hand side figures are weighted by population size of the age groups, while the left-hand side figures are unweighted. Vertical lines indicate 95% confidence bands.

Figure A.3 Robustness to items included in the frailty index



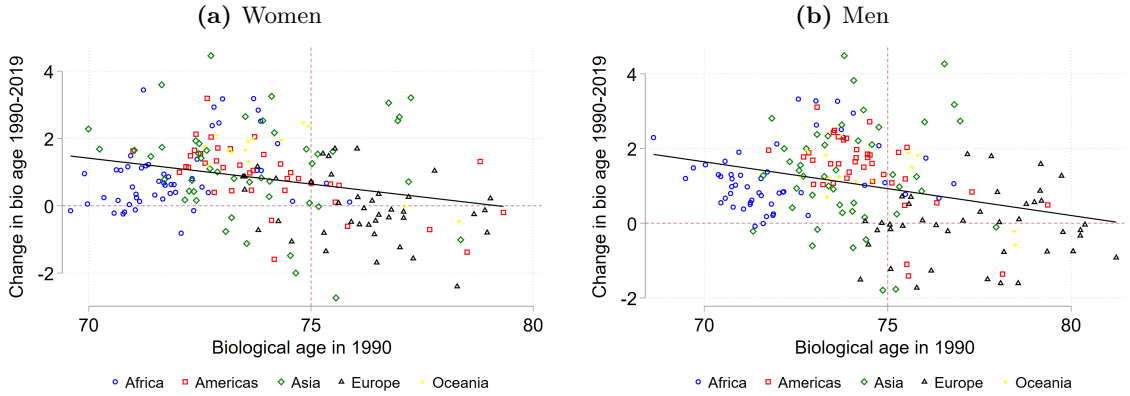
This figure presents the biological age of women and men at age 75 in 1990 and 2019, using different versions of the frailty index. The first confidence interval in each sub-figure corresponds to the baseline measure, which includes all 32 items. The subsequent 32 confidence intervals represent versions of the frailty index where one item is omitted at a time. The consistent patterns suggest that no single item is solely driving the variation in biological age

Figure A.4 β–Convergence in Biological Age chronological age 55



This figure shows the relationship between biological age in 1990 and the absolute change in biological age from 1990 to 2019. The colors indicate the geographical locations of the observation in terms of continent. The red vertical dashed marks the difference between decreases and increases in biological age, while the red dashed horizontal line indicates that we are looking at chronological age 55. Notice that age 65 refers to age group 55-59.

Figure A.5. β–Convergence in Biological Age for chronological age 75



This figure shows the relationship between biological age in 1990 and the absolute change in biological age from 1990 to 2019. The colors indicate the geographical locations of the observation in terms of continent. The red vertical dashed marks the difference between decreases and increases in biological age, while the red dashed horizontal line indicates that we are looking at chronological age 75. Notice that age 75 refers to age group 75-79.