How Do We Age? A Decomposition of Gompertz Law*

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Abstract. A strong regularity of human life is Gompertz's law, which predicts a near-perfect exponential increase in mortality with age. In this paper, we take into account that chronological age is not a cause of death and decompose Gompertz's law into two equally strong laws: (i) an exponential increase in health deficits as measured by the frailty index, and (ii) a power law association between the frailty index and the mortality rate. We show how the increase in the frailty index can be derived from the feature of self-productivity of health deficits. We explore the robustness of the Gompertz decomposition across countries, gender, and over time and show how information about mortality rates can be used to infer the state of health of an age-structured population. Finally, we use this method to infer the biological ages of past populations, such as Australian men in 1940 and Swedish men in 1770.

Keywords: mortality, morbidity, frailty index, health deficits

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1. Introduction

Gompertz's law of human mortality is soon celebrating its bicentennial but its popularity and relevance in fields as diverse as demography, biology, gerontology, economics, and actuarial science only increased with time (Gompertz, 1825; see Olshansky and Carnes, 1997, for a historical review). Gompertz law states that the mortality rate, i.e. the probability of dying in a certain age interval (usually a year) increases exponentially with age, implying that its logarithm increases linearly with age, as shown exemplarily for Swedish men in Figure B.1 in the Appendix. Mortality among children and young adults (under 30 years of age) is not accurately reflected in Gompertz's law because biological aging is dominated by accidental deaths. There is also a debate in the literature as to whether the fit of the data worsens for the oldest old (e.g. Gavrilov and Gavrilova, 2015; Barbi et al., 2018). However, over a long period of life, ranging from about 30 to 90 years of age, Gompertz law is estimated with great precision, usually with R^2s above 95%.

The estimated rate of increase in mortality (α) depends on the observed population and varies depending on the country, sex, and year of observation (usually between 7 and 11 percent). The main feature of a constant rate of increase, however, is very general and does not only apply to humans. Species as diverse as yeast, fruit flies, rats, dogs, and horses, have been shown to age also according to Gompertz law (Gavrilov and Gavrilova, 1991; Carnes et al., 1996).

Gompertz law is an excellent description of human aging, but it is not suitable as an explanatory model. Death is not caused by chronological age, but by the failure of essential functions in the human body. Specifically, aging is defined as "the time-independent series of cumulative, progressive, intrinsic, and deleterious functional and structural changes that culminate in death" (Arking, 2006, p. 11). In this paper, we propose a decomposition of Gompertz law into (i) a law of self-productivity of health deficits and (ii) an iso-elastic mapping from health deficits to the mortality rate. Self-productivity explains in reduced-form the accumulation of health deficits whereby the presence of existing health deficits leads to the emergence of new deficits.

In contrast to Gompertz law, which describes aging as an inevitable consequence of advancing time, the accumulation of health deficits is an idiosyncratic process that is malleable, for example, by healthy behavior, medical interventions, and health shocks such as accidents. The state of health, appropriately measured by an index of health deficits is thus a better predictor of death at the individual level (Mitnitski et al., 2002a; Rockwood and Mitnitski, 2007). Here, we exploit the feature that health deficits, appropriately measured, are not only an informative statistic at the individual level but also at the population level.

We measure health deficit accumulation by the frailty index. The frailty index proposed by Mitnitski et al. (2001) simply counts the number of aging-related health deficits of an individual relative to the number of potential health deficits that he or she may have. Health deficits include mild ones as well as serious disabilities. The exact choice of deficits is not crucial provided that sufficiently many indicators are present in the index (Searle et al., 2008). An explanation for this remarkable feature is that health deficits are interdependent and redundant in the sense that a given deficit represents a set of other deficits (Mitnitski et al., 2002a). For example, hypertension is associated with the risk of stroke, heart diseases, kidney diseases, dementia, and

problems of walking fast and sleeping well. This means that if a particular health deficit is missing from the list, its effect is picked up by a combination of other health deficits. It has been shown that this assumption has a thorough foundation in disease networks constructed from genetic or metabolic pathways (Barabasi et al., 2011).

The frailty index is a very good predictor of death at the individual level, outperforming not only chronological age but also other measures of health and aging such as biomarkers and biological clocks constructed from epigenetic information (Kim et al., 2017; Ji et al., 2021). Moreover, the literature has established as a robust result an exponential increase of the frailty index with age at a rate of around 2 to 4 percent per year (e.g. Mitnitski et al., 2002a,b; Mitnitski and Rockwood, 2016; Abeliansky and Strulik, 2018; Abeliansky et al., 2020; see Howlett et al., 2021 for a recent review of the literature.)

In this paper, we generalize the frailty index to a measure of health and biological age of nations. Specifically, following Dalgaard et al. (2022), we used data from the Global Burden of Disease (GBD) study (Vos et al., 2020) to construct the frailty index for the population of 40 countries, stratified by gender and age, and for the years from 1990 to 2019. With the data at hand, we estimate with great precision a law of health deficit accumulation, i.e. the rate of aging of nations (μ). Since health deficits and mortality both increase at constant rate with age, their logs most be linearly related, i.e. their levels are associated in form of a power law. Using data from HMD (2024) we estimate the power law parameter (γ). The decomposition of Gompertz law then suggests $\alpha = \gamma\mu$. In simple words, the mortality rate of a population increases at rate α because health deficits are accumulated at rate μ and because an increase of one percent health deficits causes an increase of the mortality rate by γ percent. We examine the stability of the estimated parameters across countries and over time and establish the predictive power of the Gompertz decomposition for actual mortality. Finally, we show how mortality data can be used to retrospectively predict the health of nations (for past periods for which health data is hardly available).

Our study was inspired by Mitnitski et al. (2002b) who estimated a power law association between mortality and the frailty index with data for Canadian individuals. The usefulness of this result for a decomposition of Gompertz law, however, remained unnoticed. Here we estimate the power law association for a frailty index constructed for countries. Remarkably, Mitnitski et al.'s micro-level estimate of the mortality elasticity is close to our macro-level estimate. Mitnitski et al. estimated an elasticity of 2.74 for men and 3.80 for women, while we estimate an elasticity of 2.58 for men and 3.25 for women. The closeness of these estimates further supports the robustness of the power law association as a reduced-form description of the complex pathways from health deficit accumulation to death.

The law of health deficit accumulation can be derived from a simple production function for health deficits according to which the development of new health deficits is caused by already existing health deficits. Self-productivity of health deficits can be seen as a 'reduced-form' representation of biological theories of aging proposed to explain Gompertz law: reliability theory (Gavrilov and Gavrilova, 1991), network theory (Rutenberg et al., 2018), queuing-based models (Ledberg, 2020), and mean field models (Nielsen et al., 2024). A common feature of all

theories is the conceptualization of aging as accumulation of damage and failures in complex systems constructed of redundant elements, in which the failure of a component entails the failure of other components. Lopez-Otin et al. (2013) identify nine hallmarks of aging characterizing damage accumulation at the cellular level. Belikov (2019) provides a detailed description of the self-productive accumulation of damage and failure at the molecular level that explains the exponential increase of mortality from five age-related diseases (atherosclerosis, diabetes, hypertension, Alzheimer, and Parkinson). Tian et al. (2023) show how health deficits in one organ system are associated with faster aging of other organ systems. Howlett et al. (2021) review the literature explaining how age-related damage at the cellular level scales up to functional limitations and diseases expressed in the frailty index.

2. Theory and Empirical Model

2.1. **Derivation of Gompertz Law.** As explained in the Introduction, we use a reduced-form approach to micro-founded theories of aging as damage accumulation by implementing the feature that the presence of health deficits promotes the emergence of new deficits. Formally, the change of health deficits given by $\mathrm{d}D/\mathrm{d}x$, in which x is age, is a positive function of the already existing deficits $D(x)$:

$$\frac{\mathrm{d}D(x)}{\mathrm{d}x} = \mu D(x), \tag{1}$$

in which μ is the rate of aging. Equation (1) describes aging at the population level. At the level of individuals we would add a stochastic component that captures health shocks, thereby acknowledging that all individuals age in their own idiosyncratic ways even though they obey the same 'law of aging' at the population level. Notice that the law of aging meets the characteristics of aging as an intrinsic, cumulative, and progressive, and deleterious process.

The solution of the differential equation (1) is:

$$D(x) = \tilde{\lambda}\mathrm{e}^{\mu x}, \tag{2}$$

in which $\tilde{\lambda}$ is a constant depending on initial conditions. As a result of the progressive accumulation process, health deficits increase exponentially with chronological age. The left-hand side of Figure B.2 in the Appendix shows the deficit accumulation at an almost constant rate exemplary for Swedish men. The underlying data will be presented in the next section.

The presence of health deficits (and not chronological age) ultimately causes death and the probability to die increases with the number of health deficits that a person has. The observation that both health deficits and mortality increase exponentially with age suggests that there exists a power law association between health deficits and the mortality rate $m(x)$:

$$m(x) = \tilde{\eta} D(x)^{\gamma}. \tag{3}$$

Notice that the exponent γ is a dimensionless elasticity. It shows by what percentage the mortality rate increases if health deficits increase by one percent. The panel on the right-hand side of Figure B.2 in the Appendix illustrates the power law for the case of Swedish men, where the slope (γ) shows the elasticity of mortality with respect to health deficits.

Finally, we insert (2) into (3) to obtain Gompertz law:

$$m(x) = \tilde{\beta} \mathrm{e}^{\alpha x} \tag{4}$$

with Gompertz parameters $\tilde{\beta} \equiv \tilde{\eta}\tilde{\lambda}^{\gamma}$ and $\alpha \equiv \gamma\mu$. A simple aging model thus supports Gompertz's law as a measure of aging that correlates with chronological age, even though chronological age does not cause biological aging or death. The mortality rate increases at rate α because we age biologically at rate μ and because the mortality rate increases by γ percent when health deficits increase by one percent.

2.2. **Decomposition of Gompertz Law.** We begin by taking logs of (4):

$$\log m_{ct} = \beta + \alpha \cdot age_{ct} + \theta_t + \theta_c + \epsilon_{ct} \tag{5}$$

with $\beta = \log(\tilde{\beta})$. We estimate this using mortality rates for five-year age groups (30-34, 35-39, ..,90-94) separately for men and women and for the years 1990, 1995, 2000, 2005, 2010, 2015, 2019. In the most demanding specification, we control for time fixed effects and country fixed effects by θ_t and θ_c.

The theory predicts that the Gompertz parameter α can be decomposed into the product of the force of aging μ and the mortality elasticity γ. To verify this claim, we take logs of (2) and (3) and estimate:

$$\log D_{ct} = \lambda + \mu \cdot age_{ct} + \phi_t + \phi_c + \epsilon_{ct} \tag{6}$$

$$\log m_{ct} = \eta + \gamma \log D_{ct} + \psi_t + \psi_c + \epsilon_{ct} \tag{7}$$

with $\lambda = \log(\tilde{\lambda})$ and $\eta = \log(\tilde{\eta})$. The ϕ's and ψ's denote time and country fixed effects, and we apply the same sample structure in terms of age, time, and country. Combining equations (6) and (7), we again see that the coefficient α in Gompertz Law can be interpreted as the reduced-form product between the force of aging and the power-law relationship between mortality and deficits (i.e., $\alpha = \mu \times \gamma$). We are going to provide evidence of this interpretation by estimating all three equations and comparing $\hat{\alpha}$ to $\hat{\mu} \times \hat{\gamma}$.

Finally, we are going to use the estimated age coefficients and regression constants in equations (5) and (6) to make deficit predictions:

$$\log D_{ct}^{Predict} = \hat{\lambda} + \frac{\hat{\mu}}{\hat{\alpha}}(\log m_{ct} - \hat{\beta}). \tag{8}$$

The advantage with the approach is that we only need to apply the estimated parameters from the equations that contain age terms (i.e., equations 5 and 6). We are going to show that this procedure works well within our sample and then apply it during periods of time where data on health deficits are not available, but age-specific mortality rates are available. We demonstrate that the same insights are obtained when using the estimated coefficients from the inverse of equation (7) to make the predictions instead.

3. Data and Construction of the Frailty Index

The construction of the national frailty (or deficit) index follows Dalgaard et al. (2022). Consider a population of size P_c in country c consisting of individuals indexed by i. The average

frailty index of the population d_c, is then computed as:

$$D_c = \frac{1}{P_c} \sum_{i}^{P_c} D_{ic}. \tag{9}$$

The frailty index of individual i from country c is defined as:

$$D_{ic} = \frac{1}{N} \sum_{d=1}^{N} \mathbf{1}_{ic}(d), \tag{10}$$

where $\mathbf{1}_{ic}(d)$ is an indicator function that takes on the value 1 if individual i suffers from deficit d. Using (10), we can rewrite (9) as:

$$D_c = \frac{1}{N} \sum_{d=1}^{N} \frac{P_{dc}}{P_c}, \tag{11}$$

where P_{dc} is the number of people in country c that suffer from deficit d. The aggregate frailty in a country is thus simply the average of N prevalence rates, P_{dc}/P_c. Similarly, the frailty index for an age group a in country c is computed as:

$$D_{ac} = \frac{1}{N} \sum_{d=1}^{N} \frac{P_{dac}}{P_{ac}}, \tag{12}$$

where P_{dac}/P_{ac} is the prevalence rate of health deficit d within age group a in country c.

We constructed the frailty index separately for men and women using data on disease prevalence rates from the Global Burden of Disease Study (GBD 2019; Vos et al., 2020) for the period 1990 to 2019. The prevalence rates are available for men and women separately, as well as for five year age-groups. For construction of the frailty index we focused on the population between ages 30 and 80 and followed the methodology laid out in Searle et al. (2008) requiring that (i) a deficit needs to be associated with health status; (ii) a deficit's prevalence must generally increase with age; (iii) a deficit must not saturate too early; (iv) the deficits that make up a frailty index must cover a range of systems and (v) must not change from one observation period to the next. As in our earlier study, this selection procedure led to a frailty index constructed from 32 diseases for which there exists data on prevalence at the global level (listed in Appendix A).

We use the Human Mortality Database (HMD, 2024) to obtain age-specific mortality rates for 40 countries by sex. These so-called period mortality rates are measured in 1990, 1995 up to 2019 to match the deficit data. The countries included in the sample are European, Western offshoots, and a couple Asian ones (Japan, Korea). The sample consists of relatively economically developed countries, which has the advantage that the disease prevalence data from GBD (2019) are actual disease data and are not otherwise derived from mortality rates.

4. Results

The coefficients from estimating Gompertz Law are reported in Table 1. The female estimates, shown in columns 1-3, are 9.5 percent for the age parameter, and the males estimates, shown in columns 4-6, are 8.7 percent. The age parameter ($\hat{\alpha}$) is thus higher for males than females,

but is otherwise extremely stable across specifications, unsurprisingly showing that age rather the differences across country and time explain variation in mortality rates, and in terms of magnitude in line with the previous literature (see e.g. Strulik and Vollmer, 2013).

TABLE 1. **Gompertz Law: Mortality and Age** ($\hat{\alpha}$ and $\hat{\beta}$)

	(1) $\log m_{ct}$	(2) $\log m_{ct}$	(3) $\log m_{ct}$	(4) $\log m_{ct}$	(5) $\log m_{ct}$	(6) $\log m_{ct}$
age_{ct}	0.0949***	0.0949***	0.0949***	0.0874***	0.0874***	0.0874***
	(0.000828)	(0.000828)	(0.000828)	(0.00136)	(0.00136)	(0.00136)
β	-10.50***	-10.50***	-10.50***	-9.407***	-9.407***	-9.407***
	(0.0752)	(0.0455)	(0.0456)	(0.133)	(0.0750)	(0.0750)
R^2	0.938	0.973	0.988	0.885	0.961	0.981
Country FE	No	Yes	Yes	No	Yes	Yes
Year FE	No	No	Yes	No	No	Yes
Sex	Female	Female	Female	Male	Male	Male
Obs	2937	2937	2937	2937	2937	2937
Countries	40	40	40	40	40	40

This table reports parameter estimates of equation (5) for women (columns 1-3) and men (columns 4-6). The sample period is 1990, 1995, 2000, 2005, 2010, 2015, and 2019. The fixed effects included are indicated in the bottom rows. Standard error are clustered at the country level. *** p<0.01, ** p<0.05, * p<0.1.

The estimated rates of aging in terms of deficits are reported in Table 2. Consistent with the results in Dalgaard et al. (2022), which were obtained for a larger sample of countries, we find that female deficits grow by 2.9 percent per age-year, while male deficits grow by 3.3 percent. These magnitudes are the same across the three different type of specifications. Again, we clearly see the pattern that possible heterogeneities across country and time have no influence on the relationship between deficits and age.

In Table 3, we report the mortality-deficit relationship by estimating equation (7), which provides somewhat larger estimates for females compared to males, but otherwise very stables estimates across specifications. A one percent increase in health deficits is associated with a 2.6 percent increase in mortality among men and a 3.3 percent increase in mortality among women.

We can now provide evidence on the main point of the paper: at the bottom of Table 3 we report the implied Gompertz parameters α, obtained by multiplying the estimated coefficients μ and γ from the Table 2 and 3. The predicted Gompertz parameters are very similar in magnitude compared to the estimated ones from Table 1. For example, using the specification that controls for country and year fixed effects (i.e., columns 3 and 6 across the three tables), we find for the female sample $\hat{\alpha}^F = 0.0949$ vs. $\hat{\mu}^F \times \hat{\gamma}^F = 0.0940$, and the male sample $\hat{\alpha}^M = 0.08742$ vs. $\hat{\mu}^M \times \hat{\gamma}^M = 0.0867$.

In Figure 1, we report estimates of equations (5)-(7) for each country, year, and sex. The reduced-form Gompertz Law of mortality coefficients ($\hat{\alpha}$) are depicted in panel A, the age deficits ($\hat{\mu}$) in panel B, the directly estimated deficit-mortality power-law coefficients ($\hat{\gamma}$) in panel C, and

TABLE 2. **The Speed of Aging** ($\hat{\mu}$ and $\hat{\lambda}$)

	(1) $\log D_{ct}$	(2) $\log D_{ct}$	(3) $\log D_{ct}$	(4) $\log D_{ct}$	(5) $\log D_{ct}$	(6) $\log D_{ct}$
age_{ct}	0.0289***	0.0289***	0.0289***	0.0335***	0.0335***	0.0335***
	(0.000202)	(0.000202)	(0.000203)	(0.000204)	(0.000204)	(0.000205)
λ	-3.932***	-3.932***	-3.932***	-4.239***	-4.239***	-4.239***
	(0.0167)	(0.0111)	(0.0111)	(0.0159)	(0.0112)	(0.0113)
R^2	0.978	0.993	0.993	0.969	0.990	0.990
Country FE	No	Yes	Yes	No	Yes	Yes
Year FE	No	No	Yes	No	No	Yes
Sex	Female	Female	Female	Male	Male	Male
Obs	2937	2937	2937	2937	2937	2937
Countries	40	40	40	40	40	40

This table reports parameter estimates of equation (6) for women (columns 1-3) and men (columns 4-6). The sample period is 1990, 1995, 2000, 2005, 2010, 2015, and 2019. The fixed effects included are indicated in the bottom rows. Standard error are clustered at the country level. *** p<0.01, ** p<0.05, * p<0.1.

TABLE 3. **Mortality and Deficits** ($\hat{\gamma}$ and $\hat{\eta}$)

	(1) $\log m_{ct}$	(2) $\log m_{ct}$	(3) $\log m_{ct}$	(4) $\log m_{ct}$	(5) $\log m_{ct}$	(6) $\log m_{ct}$
$\log D_{ct}$	3.247***	3.257***	3.254***	2.606***	2.592***	2.589***
	(0.0380)	(0.0377)	(0.0376)	(0.0415)	(0.0457)	(0.0457)
η	2.323***	2.348***	2.341***	1.651***	1.618***	1.609***
	(0.0796)	(0.0883)	(0.0880)	(0.0808)	(0.110)	(0.110)
R^2	0.937	0.964	0.977	0.909	0.956	0.974
Country FE	No	Yes	Yes	No	Yes	Yes
Year FE	No	No	Yes	No	No	Yes
Sex	Female	Female	Female	Male	Male	Male
Obs	2937	2937	2937	2937	2937	2937
Countries	40	40	40	40	40	40
Implied α	0.0938	0.0941	0.0940	0.0872	0.0868	0.0867

This table reports parameter estimates of equation (7) for women (columns 1-3) and men (columns 4-6). The sample period is 1990, 1995, 2000, 2005, 2010, 2015, and 2019. The fixed effects included are indicated in the bottom rows. Implied α is $\hat{\mu} \times \hat{\gamma}$ using estimates from this table ($\hat{\gamma}$) and estimates from the previous table ($\hat{\mu}$). Standard error are clustered at the country level *** p<0.01, ** p<0.05, * p<0.1.

the implied values for power law from $\gamma = \alpha/\mu$ in panel C.[1] Across all 640 regressions (2 sexes ×

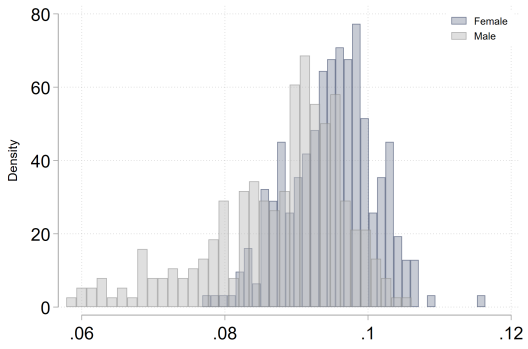
[1]Thus, instead of computing implied values for α as we did in Table 3, one can of course, as an alternative, calculate implied values for γ, as we do in panel D of Figure 1 for all countries, years and sexes.

8 years × 40 countries), the estimated α's are in the range 6 to 11 percent while the estimated μ's are in the range 2.3 to 3.5 percent. The directly estimated values for the power law and the implied values for the γ's, based on the coefficients in panels A and B, are in the range 1.8 to 4.2. This can be compared to the "global parameters" reported in Table 3, which were 2.6 for males and 3.3 for females; or be compared to the directly estimated γ's in panel C. We note that the reason that the estimates in panels C and D and are not exactly alike is that they obtained in two different ways: estimated directly in panel C, and in panel D derived indirectly from the estimated parameters show in panels A and B.

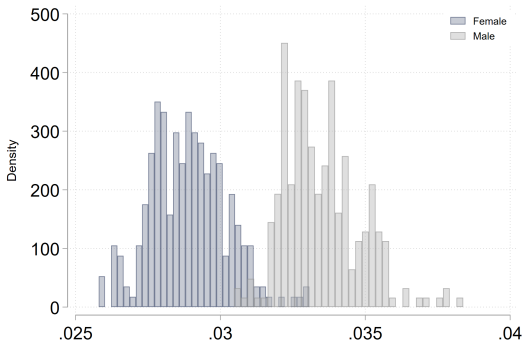
In Figure 2, we show estimates for each year and sex, while controlling for country fixed effects. We see that the speed of aging parameter ($\hat{\mu}$) is extremely stable across the years (panel B). The estimated α's are also quite stable for women and slightly increasing for men (panel A). An increasing rate of mortality α (together with a declining shift parameter β) is an expression of the compensation law of mortality (Gavrilov and Gavrilova, 1991; Strulik and Vollmer, 2013).

FIGURE 1. **Regression Coefficients by Country, Sex, and Year**

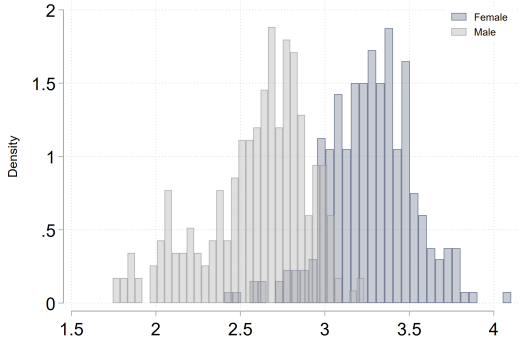
(A) Gompertz Law of mortality estimates ($\hat{\alpha}$)



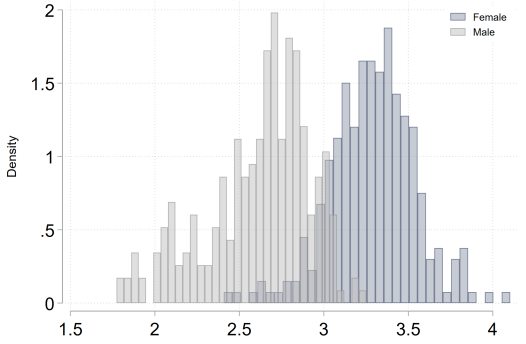
(B) Deficits-age estimates ($\hat{\mu}$)



(C) Mortality-deficit relation estimated directly ($\hat{\gamma}$)



(D) Implied estimates for the mortality-deficit relation ($\frac{\hat{\alpha}}{\hat{\mu}}$)

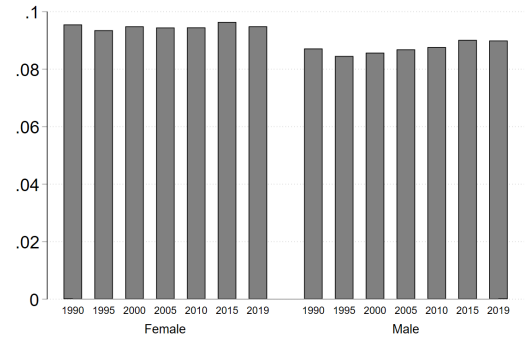


This figure shows estimates from equations (5)-(7) for each country, year, and sex in the sample. These are reported without confidence bands. In panel D, we combine the estimates in panels A and B to obtain and implied values for γ, which can be be compared to the ones estimated directly in panel C.

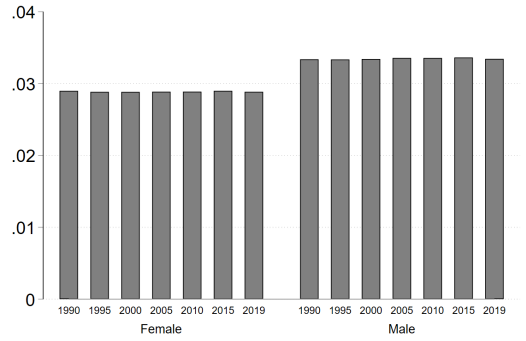
In Figure 3, we compare our in-sample predictions of deficits using equation (8) to predict deficits. We use different specifications to obtain the required parameter coefficients: Panel A uses country-year-sex specific estimates to make the predictions, panel B uses country-sex

FIGURE 2. **Regression Coefficients by Sex and Year**

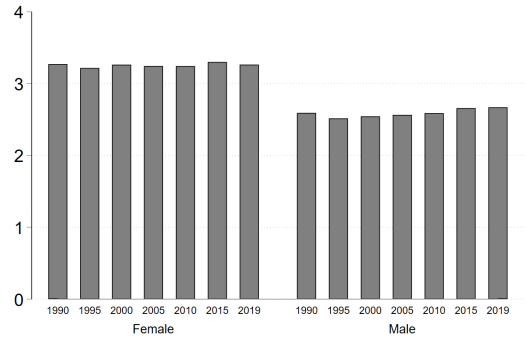
(A) Gompertz Law of mortality estimates ($\hat{\alpha}$)



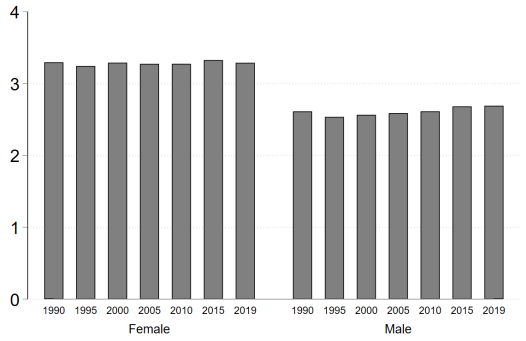
(B) Deficits-age estimates ($\hat{\mu}$)



(C) Mortality-deficit relation estimated directly ($\hat{\gamma}$)



(D) Implied estimates for the mortality-deficit relation ($\frac{\hat{\alpha}}{\hat{\mu}}$)



This figure shows regression coefficients from equations (5)-(7) in panels A-C and then implied values for γ in panel C for each year and sex, for all 40 countries in our sample and controlling for country fixed effects. Standard errors are nor reported, but all estimates are statistically significant at the one percent level

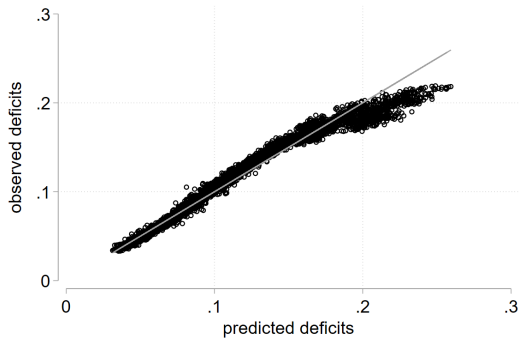
specific estimates, while panel c is based on global estimates for each sex, controlling for country and year fixed effects, as reported in columns 3 and 6 of Tables 1 and 2. In each panel, a 45-degree line indicates the locus of perfect alignment of prediction and observation. We conclude that our procedure to predict health works fairly well in all three cases. However, we tend to over-predict at high levels of health deficits, above a frailty index of 0.2, which is reached on average at about age 85. For the oldest old, health deficits tend to increase slower than exponentially (cf. Figure B.2), perhaps indicating a selection (or survivor) effect. This means that the use of mortality rates to predict health deficits leads to an over-prediction of health deficits at high levels of deficits.

5. How was health?

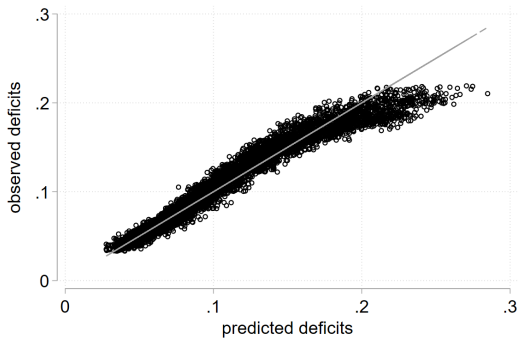
In many countries, mortality statistics go back further than health statistics. Next, we show how this information can be used in terms of out-of-sample predictions to assess the health status of populations from previous time periods. Specifically, we use HMD (2024) data available for earlier periods to estimate Gompertz law of mortality. We then take the predicted historical

FIGURE 3. **Observed vs. Predicted Deficits**

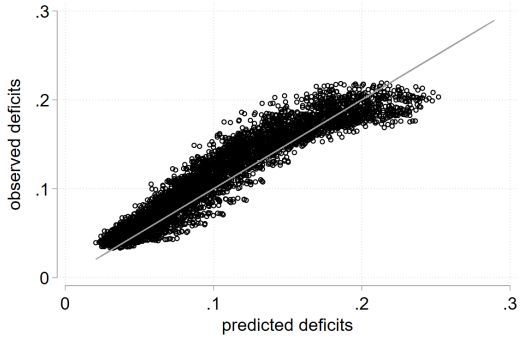
(A) Based on estimated parameters by country, year, and sex



(B) Based on estimated parameters by country and sex



(C) Based on estimated parameters by sex



This figure shows the relationship between observed deficits and predicted deficits from equation 8. Panel A uses country-sex-year specific estimates; panel B uses country-sex specific estimates, while panel C uses common estimates across countries, controlling for country and year fixed effects.

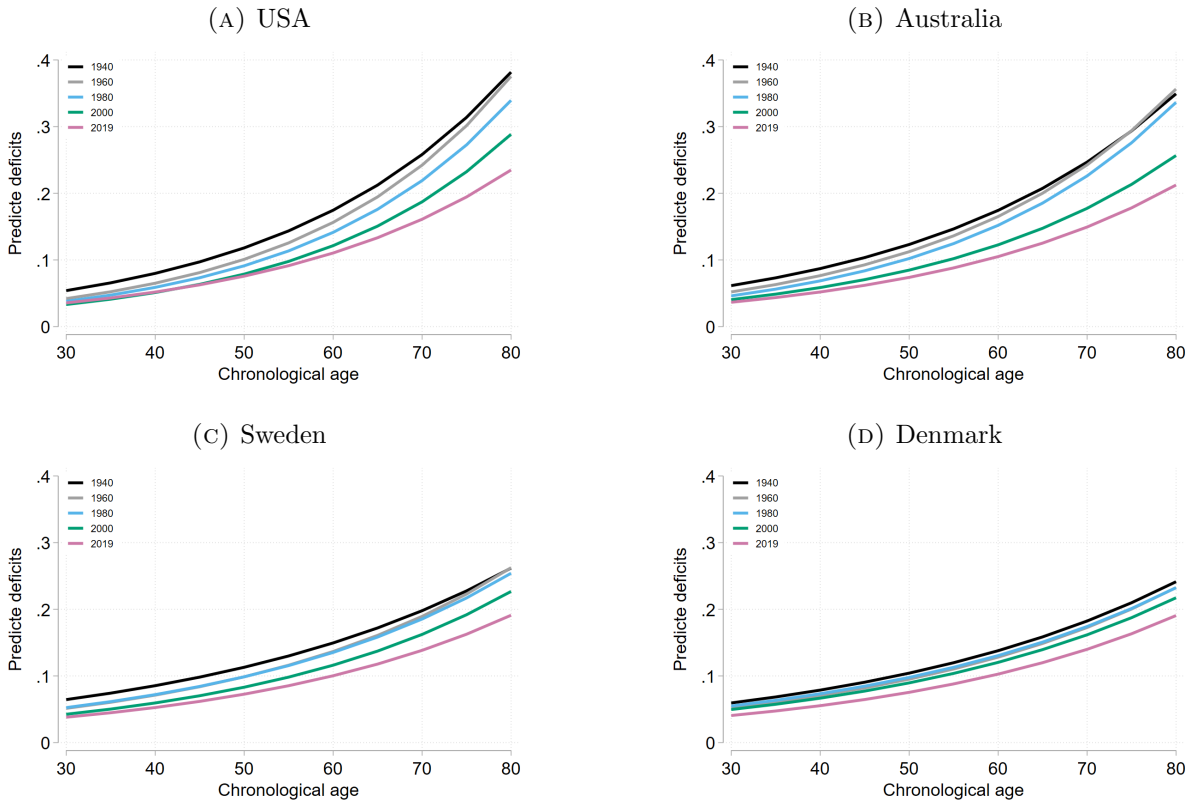
morality rates $\log \hat{m}_{ct}$ and feed them into the prediction equation (8):

$$\log D_{ct}^{Predict} = \hat{\lambda} + \frac{\hat{\mu}}{\hat{\alpha}}(\log \hat{m}_{ct} - \hat{\beta}). \tag{13}$$

We use predicted mortality rates rather than observed values in order to smooth out measurement error. The parameters in (13) are based on country-sex specific estimates for the year 2019. The prediction thus answers the question of how we would assess the health of historical populations today when we only knew their mortality rates.

Figure 4 shows the predicted health deficits of men from four selected countries (USA, Australia, Sweden, and Denmark) for the period 1940 to 2019. We see that, for example, Swedish men are predicted to have accumulated a frailty index of about 0.1 at age 60 in 2019, at age 50 in 1980, and at age 45 in 1940. In the two Scandinavian countries we see smaller health gains among older people than in the US and Australia, largely because older Scandinavians were already relatively healthy by mid-century. In the U.S., on the other hand, we observe very small health gains for young people since the 1960s and even a bit of health deterioration from 2000 to 2019.

FIGURE 4. **Predicted Historical Deficits 1940 – 2019**



This figure shows predicted deficits age, using equation 13 with specific estimated parameters for each country and men for the year 2019. We use the estimated age-specific mortality rates in the years 1940, 1960,.., 2010, 2019 to make the historical predictions of deficits.

To further facilitate the interpretation of the predicted historical health data, we follow Mitnitski et al. (2002a) and transform them into predicted biological age using the following equation:

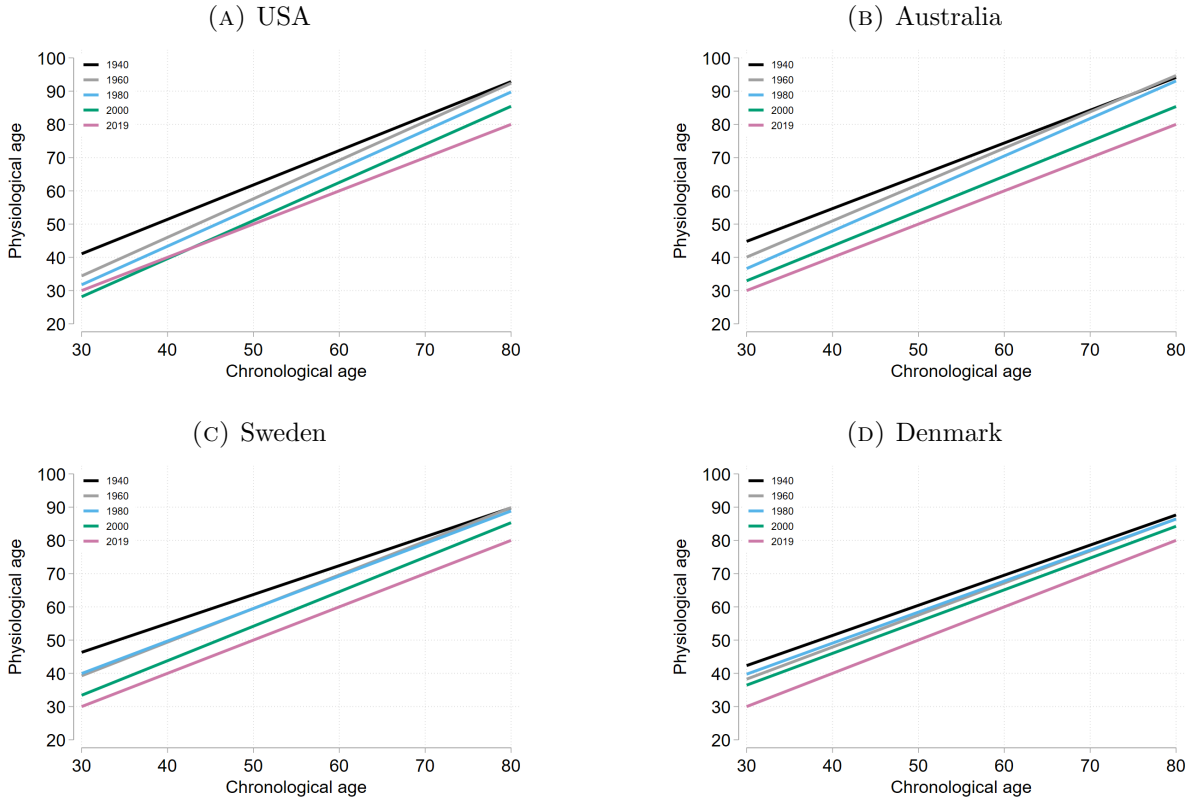
$$bioage_{ct}^{Predict} = (\log D_{ct}^{Predict} - \hat{\lambda})/\hat{\mu}, \tag{14}$$

where the estimated parameters used in equations (13) and (14) are based on country-sex specific estimates for the year 2019. Biological age is thus defined as the age at which the average individual of country c in 2019 has $D_{ct}^{Predict}$ deficits. According to this normalization biological age coincides with chronological age for people observed in 2019. For other periods, biological age is the estimated age based on their health deficits, i.e. it answers the question "Based on their predicted health deficits, how old would we estimate today people who were xx years old in the year $yyyy$?

In Figure 5, we plot chronological age against predicted biological age, again for men from the four selected countries (USA, Australia, Sweden, and Denmark) for 1940 to 2019. Returning to our previous example, we see that Swedish men who were 45 years old in 1940 and 50 years old in 1980 are predicted to be 60 years old in 2019. Or vice versa, a 60-year-old men in 2019 would be predicted to be 69 in 1980 and 72 in 1940. For the Scandinavian countries, the decline in biological age 1940–2019 is larger for the young than for the old. For the U.S. and

Australia, people at all ages experienced about the same decline in biological age of about 10 to 12 years. These results corroborate earlier studies arguing that "60 is the new 50" (Levine and Crimmins, 2018) while the claim that "50 is the new 30" (Strulik and Werner, 2016) appears to be exaggerated by a couple of years. In contrast to these generally positive health trends, people under 40 in the United States were biologically *younger* in 2000 than in 2019, consistent with the "death of despair" hypothesis (Case and Deaton, 2022).

FIGURE 5. **Biological Age 1940 – 2019**



This figure shows predicted biological age, using equation 8 with specific estimated parameters for each country and men for the year 2019. We use the estimated age-specific mortality rates in the years 1940, 1960,.., 2010, 2019 to make the historical predictions of deficits which allows us to calculate the biological age using $bioage_{ct} = (\log D_{ct}^{predict} - \hat{\lambda})/\hat{\mu}$, where $\hat{\lambda}$ and $\hat{\mu}$ also are male-specific to each country in 2019.

In principle this procedure can be applied to any historical mortality data. However, when going further back in time, the model needs a refinement to take into account non-aging-related (accidental) deaths. Specifically, we estimate the Gompertz-Makeham law of mortality (Makeham, 1860):

$$m(x) = A + \tilde{\beta} \exp(\alpha x), \tag{15}$$

using a non-linear regression method. The Makeham term captures accidental deaths. For recent periods, the Makeham term is usually very small and differs insignificantly from zero. It can be safely ignored such that (15) collapses to Gompertz law. For earlier periods, however, it has be shown that the Makeham term can be substantial (Gavrilov and Gavrilova, 1991) and its omission could lead to biased estimates of health deficits and biological age.

To show the importance of including the Makeham term, we use HMD (2024) data for Sweden 1770-2019. We first ignore the Makeham term and follow the same procedure as for Figure 5. Results are shown in Panel A of Figure B.4 in the Appendix B.4. We see that the predicted biological age of a Swedish man in 1770 is very high, 30-year-olds are predicted to be of biological age 60.

We next obtain A from regressing (15) and use the predicted *aging-related* mortality rate $\tilde{m}(x) = \hat{m}(x) - A$ in (13). In line with the theory, we therefore use the mortality rate that is caused by health deficit accumulation (rather than accidents). Results are shown in Panel B of Figure B.4. Comparing biological age from both panels, we see no significant differences for the year 2000, corroborating the claim that the Makeham term can be safely ignored. For 1950 and earlier periods, we see significant differences, in particular at young ages. Purging the mortality rate from accidental deaths and deaths from infectious diseases with non-aging mortality reduces biological age predicted for the 19th century by about 10 years. Biological age, however, is still very high. In 1770, 30 year-olds have a biological age of about 50. The health differences across periods are largest for young people. The convergence at older ages is akin to the compensation law of mortality and the feature that initially more healthy people age faster (Gavrilov and Gavrilova, 1991; Abeliansky et al., 2018). The greatest health improvements were achieved over the period 1850 to 1900.

6. Conclusion

While Gompertz law is an excellent description of human mortality, it does not explain how we age. In this paper, we proposed an explanation that is derived from a decomposition of Gompertz law into an exponential increase in health deficits, measured by the frailty index, and a power law relationship between health deficits and mortality. We showed how the increase in health deficits can be derived from the self-productivity of health deficits, which in turn has a foundation in the theoretical biology of aging. In short, we die at a rate of about 9 percent because we accumulate health deficits at a rate with 3 percent and any one percent increase in health deficits increases mortality by 3 percent. Using data from the Global Burden of Disease study and the Human Mortality Database, we showed that the parameters underlying the Gompertz decomposition can be estimated with great precision and mild but measurable differences for men and women.

We then showed how the decomposition can be used to retrospectively predict the health status of past populations. This procedure is useful since in many countries, mortality statistics go back further than health statistics. For better interpretation, we performed a further transformation of the data that provides the biological age of individuals. This is an estimate of the chronological age of people from past populations based on their predicted health deficits. According to this metric, people from past populations were biologically much older than they are today. An average 60-year-old Swede in 2020, for example, has the same biological age as a 50-year-old in 1980 and a 45-year-old in 1940.

Health predictions going further back in time, when non-aging related deaths played a greater role, were obtained with non-linear regression and the Makeham amendment. They showed that

the health gap between past and present population groups continues to widen with increasing distance in time. For example, we predict that 40-year-old Swedes in 1770 had the same health deficits as 60-year-olds today, meaning that 40-year-olds today have about 50 percent fewer health deficits than in 1770. These results are comparable with those of the few other studies on chronic health deficits in past populations. Costa (2000) found that an index of severe chronic health problems in American men aged 50–74 declined by 66 percent from the early 1900s to the 1970s. Strulik and Grossmann (2024) estimate that the frailty index of 65-year-old Americans declined by 40 percent from 1860 to 2020.

As with mortality, individual productivity decline can be well described by chronological age, for example as in Mincer regressions, although chronological age is not causal (Mincer, 1974; Hansen et al., 2023). The insight that productivity and life cycle decisions, such as retirement, depend on biological age gives policymakers an edge. While chronological aging proceeds inevitably by one year every year, biological aging is malleable. Forecasts of future productivity and fiscal sustainability are typically based on populations structured by chronological age. However, if the biological aging trends uncovered in our study continue, the future prospects for population aging are less bleak than often assumed.

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Appendix A. Appendix: Items in the Frailty Index

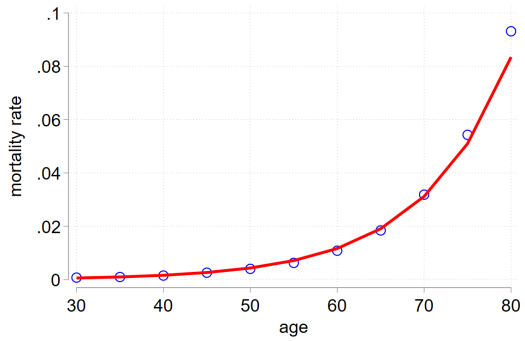
The frailty index is based on prevalence rates for the following diseases (32 in total):

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.

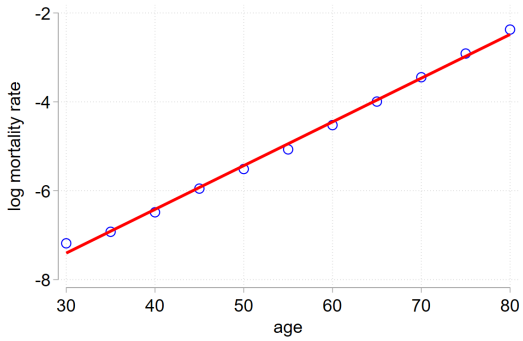
Appendix B. Additional Figures and Results

Figure B.1. **Gompertz Law**

(A) mortality rate



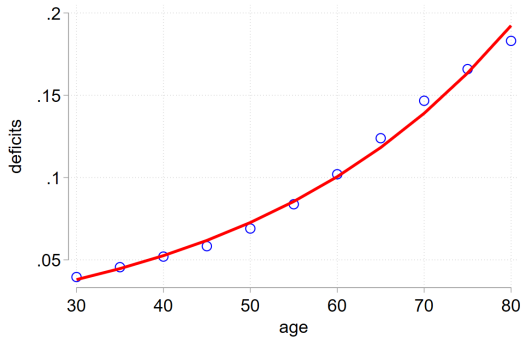
(B) log mortality rate



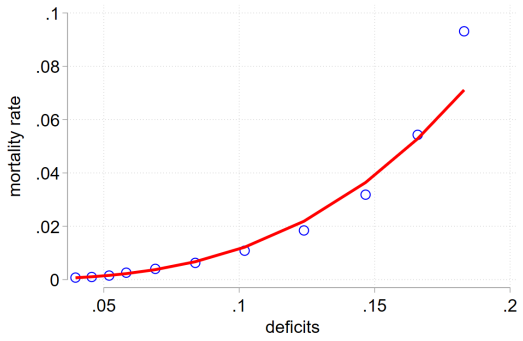
Notes: Swedish men, year 2000; circles: data from HMD (2024). Regression line: $\log(m) = -10.35 + 0.98x$ ($R^2 = 0.99$) with mortality rate m and age x.

Figure B.2. **Health Deficits and Mortality**

(A) deficits and age

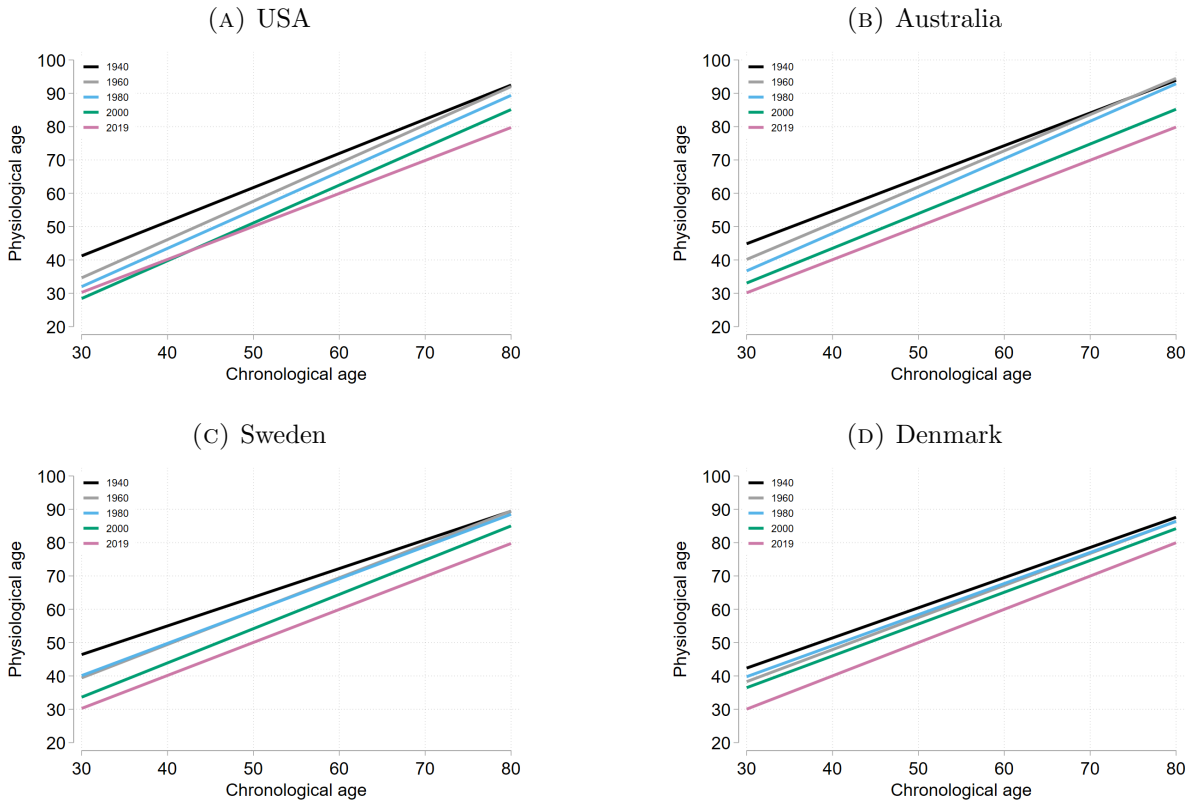


(B) mortality and deficits



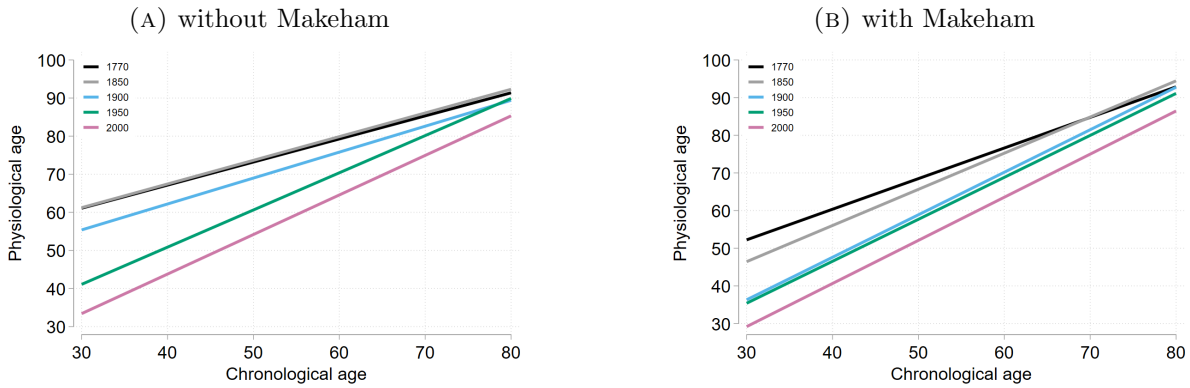
Notes: Swedish men, year 2000. The curve to left-hand side is an exponential function with parameter from the log-level regression: $\log(D) = -4.24 + 0.032x$ ($R^2 = 0.99$) with frailty index D and age x. The curve to the right-hand is the power-law with parameters based on the log-log regression: $\log(m) = 2.48 + 3.01 \log(D)$ ($R^2 = 0.99$). See below for construction of frailty index; mortality data from HMD (2024).

FIGURE B.3. **Robustness: Biological age 1940 – 2019**



This figure follows Figure 5. However, the historical deficit predictions are now based on estimating the coefficients of an inverted version of equation 7, which we apply to the observed historical mortality rates to predict historical deficits. We still make use of equation 14 to transform deficits into biological age.

FIGURE B.4. **Biological Age: Sweden 1770, 1850, 1900, 1950, and 2000**



Notes: Panel A follow the same procedure as in Figure 5 for Sweden, but using mortality data going back to 1770. Panel B subtracts the Makeham constant to the observed age-specific mortality rates, before calculating predicted deficits and biological age. We estimate the Makeham constant for each year in question, using mortality data for Sweden and non-linear regressions.