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Biological age vs Chronological Age and Retirement Policy

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12 September 2019
Washington, L15

My Objective

Convince consumers to take longevity risk as seriously as we do and get them to take action, e.g. work longer, delay SS, annuitize, etc.

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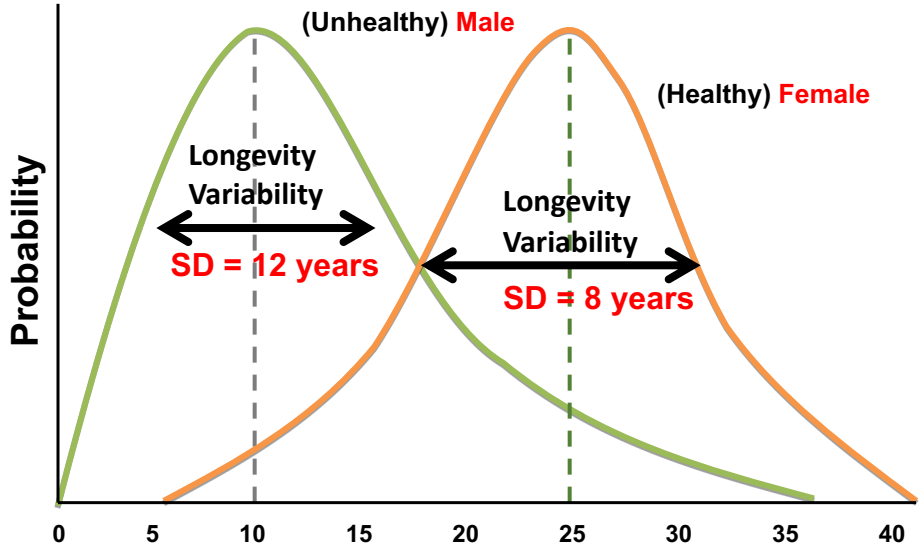
Longevity in the U.S. (2018)

Number of Americans…	
> Age 90	**2,000,000 +**
> Age 100	**78,000**

***Source**: NCHS Data Brief #233 (January 2016)*
U.S. Department of Health and Human Services
Total U.S. Population of 325 Million

An Insurance Economist view of Retirement



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Claim…

Real-world consumers have a tough time (thinking about) insuring an event that is 25 years away.

Largest Longevity Shock in the 20th century:

The Spanish Flu (H1N1) in 1918

- The first occurrence was observed in Kansas, in January 1918. Some say it began in Shanxi province in Dec/1917.
- Killed over 50m globally (compared to 17M in WWI), and possibly the worst catastrophe of the 20th century.
- Approximately 500m people were infected globally, with a mortality rate of 10% (vs. 0.1% for influenza)
- Approximately 675,000 Americans died from Spanish Flu.
- Spain's King Alfonso XIII was infected, well publicized, became quite ill and survived. Hence the name.
- U.S. insurance companies (survived and) paid 0.5% of GDP in death benefits which is $30B in today's dollars. (In comparison, in 2016 they paid $76B in death benefits)

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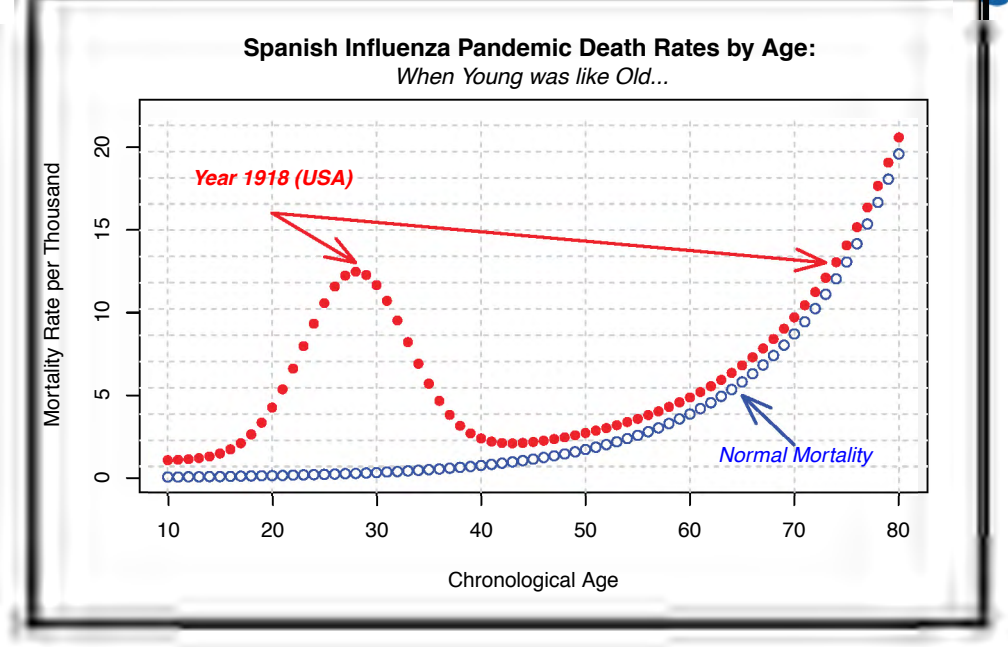
What was unexpected in 1918?

The elderly were more likely to survive,
vs. young healthy people in their 20s and 30s,
who experienced a much higher mortality rate.



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Implications for the Insurance Industry? National Underwriter (1922)

- The epidemic "*has brought forward the benefits of insurance to the people in a way that they had not appreciated…*"
- "*In every community where deaths have occurred in large numbers from influenza, the first question that arose was whether the deceased carried any life insurance.*"

Challenge…

How do we make longevity risk as salient as mortality risk?

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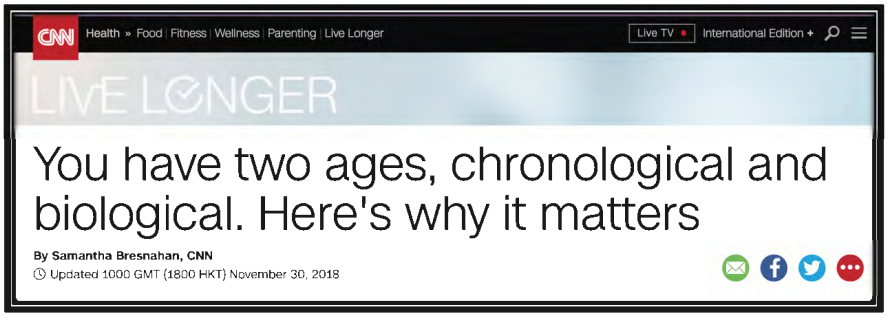
Here is a thought:

"You might become a centenarian…"

may not be the most effective way to get people to take action, today.



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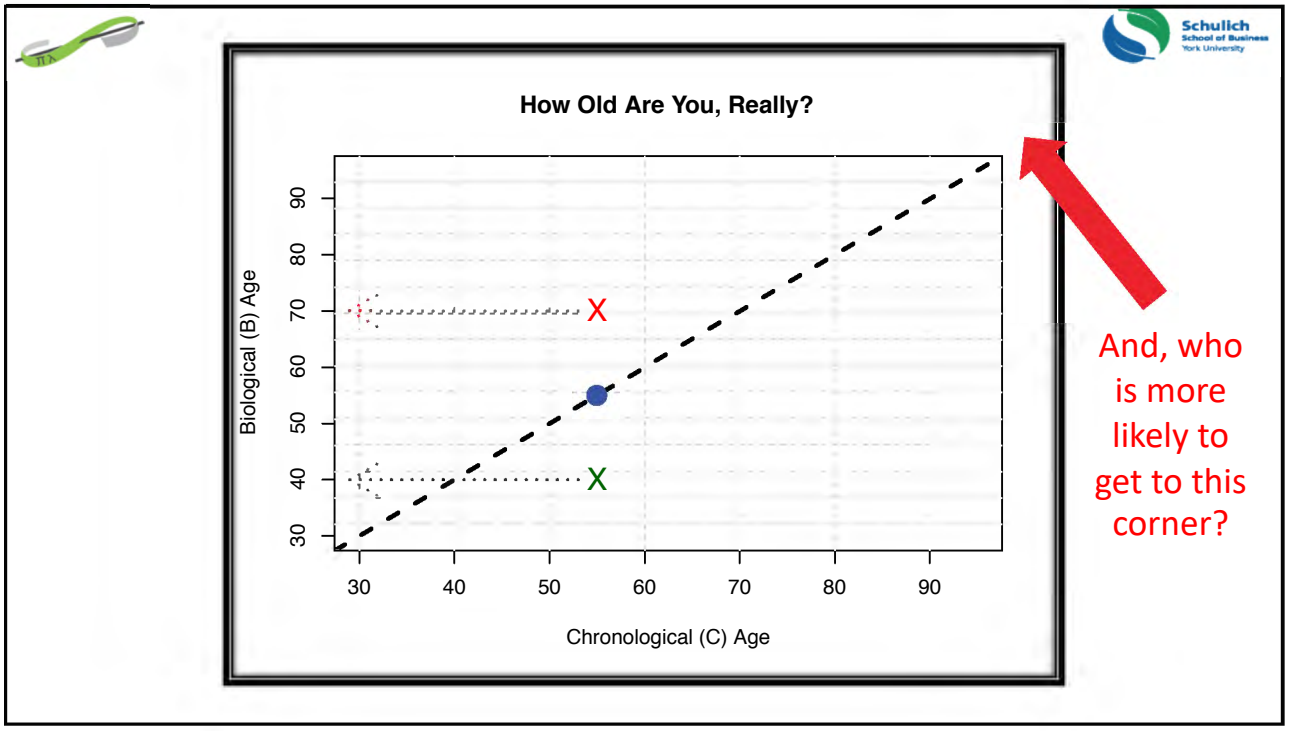

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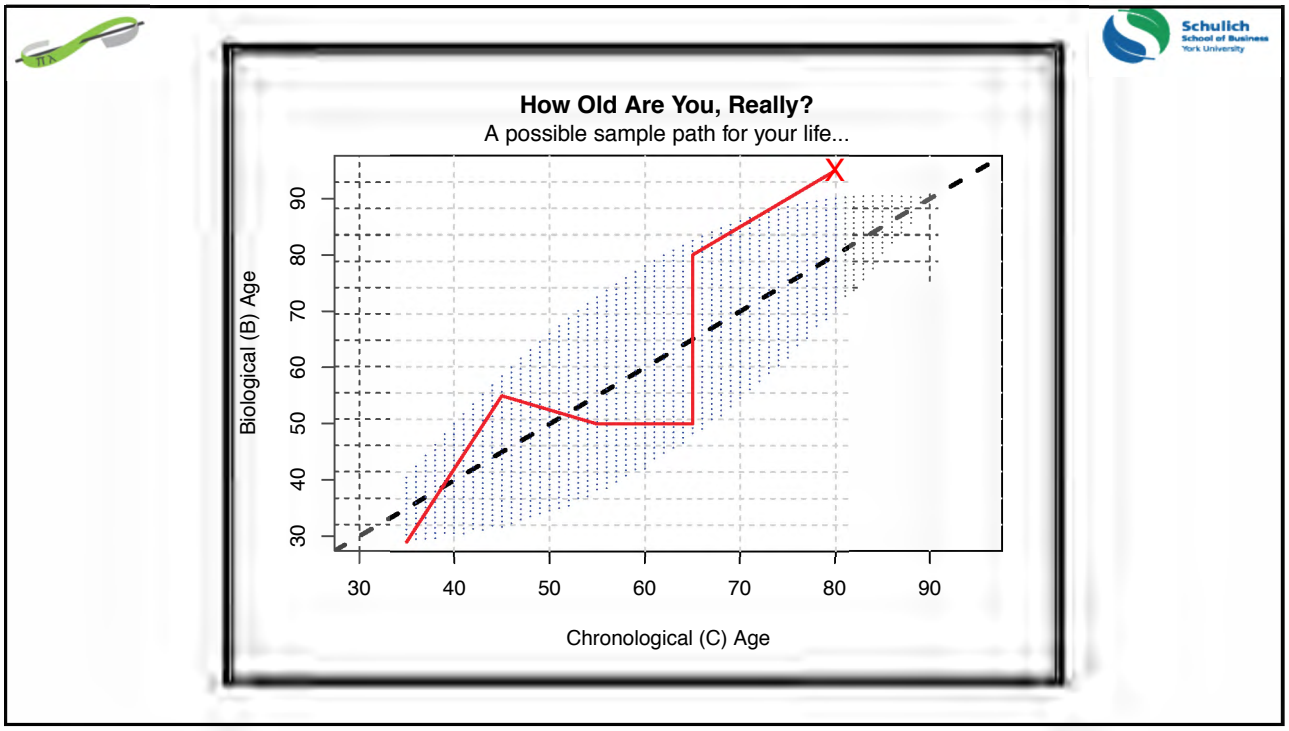

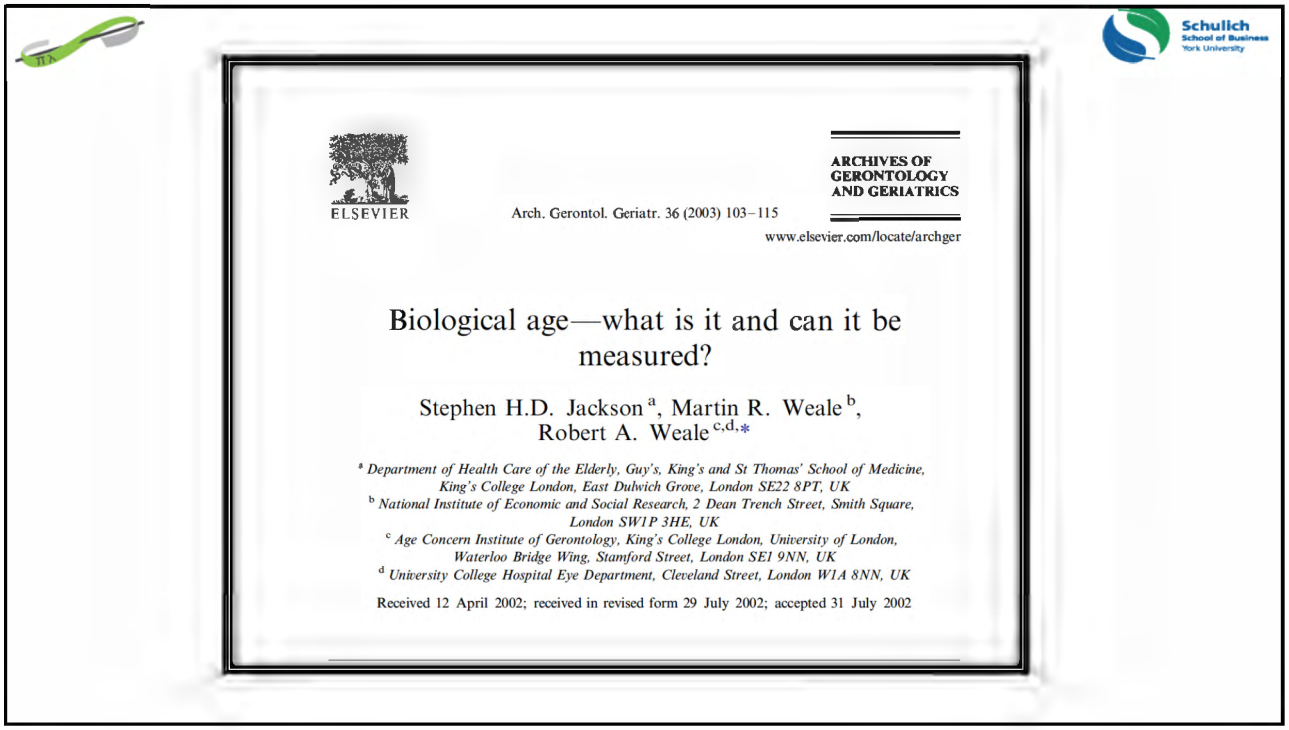

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Biological age—what is it and can it be measured?

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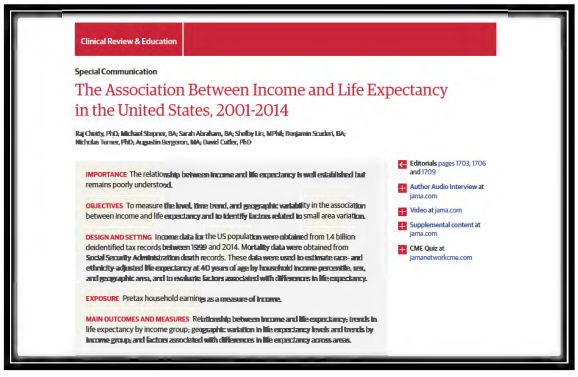
Your Biological Age (BA)

$$BA = CA + \left(\sum_{j=1}^{k} \beta_j U_j \right)$$

For example, if your **chronological** age (CA) is 55, and the sum in brackets is 10, then your **biological** age (BA) is 65. So, you want negative betas, assuming U>0.

Wealth, Income and Biological Age



Clinical Review & Education

Special Communication

The Association Between Income and Life Expectancy in the United States, 2001-2014

Raj Chetty, PhD; Michael Stepner, BA; Sarah Abraham, BA; Shelby Lin, MPhil; Benjamin Scuderi, BA; Nicholas Turner, PhD; Augustin Bergeron, MA; David Cutler, PhD

IMPORTANCE The relationship between income and life expectancy is well established but remains poorly understood.

OBJECTIVES To measure the level, time trend, and geographic variability in the association between income and life expectancy and to identify factors related to small area variation.

DESIGN AND SETTING Income data for the US population were obtained from 1.4 billion deidentified tax records between 1989 and 2014. Mortality data were obtained from Social Security Administration death records. These data were used to estimate race- and ethnicity-adjusted life expectancy at 40 years of age by household income percentile, sex, and geographic area, and to evaluate factors associated with differences in life expectancy.

EXPOSURE Pretax household earnings as a measure of income.

MAIN OUTCOMES AND MEASURES Relationship between income and life expectancy, trends in life expectancy by income group, geographic variation in life expectancy levels and trends by income group, and factors associated with differences in life expectancy across areas.

Editorials pages 1703, 1706 and 1709

Author Audio Interview at jama.com

Video at jama.com

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CME Quiz at jamanetworkcme.com

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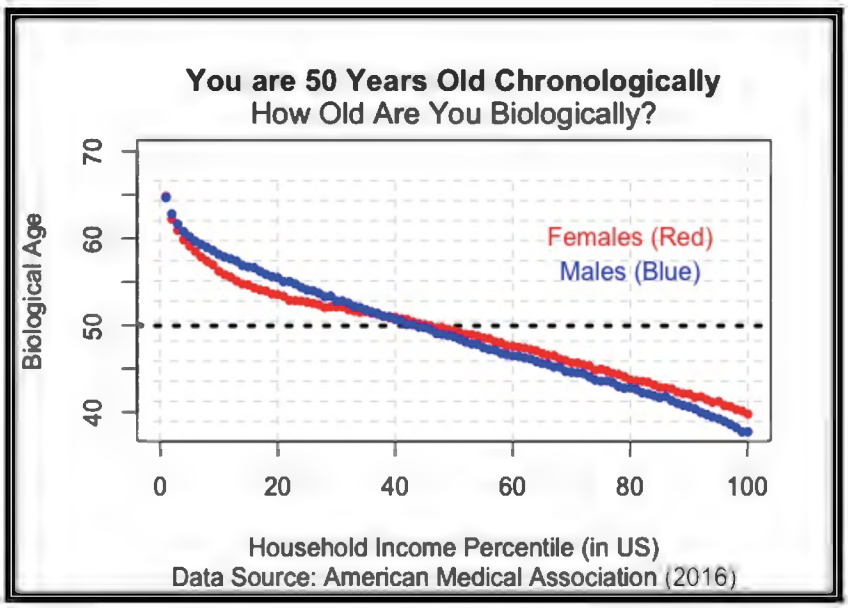

Claim…

Gap between Biological Age
and Chronological Age
can be as large as 20 years.

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Milestone Retirement Policy Ages (US): Chronological

- **50.0:** Catch-up contributions to IRA/401k.
- **55.0:** Penalty Free Withdrawals from 401k if separated from service.
- **59.5:** Penalty Free Withdrawal from 401k
- **60.0:** Minimum Social Security Survivor Benefits
- **62.0:** Minimum Social Security Retirement Benefits
- **65.0:** Medicare Begins. HSA penalty-free withdrawal.
- **67.0:** Full Retirement Age (if born after 1960)
- **70.0:** Maximum Social Security Benefit
- **70.5:** Required Minimum Distribution for Traditional IRA.
- **85.0:** Qualified Longevity Annuity Contract (QLAC) must begin.

Twitter Summary

Your current age is stochastic…

…but your life span is deterministic.

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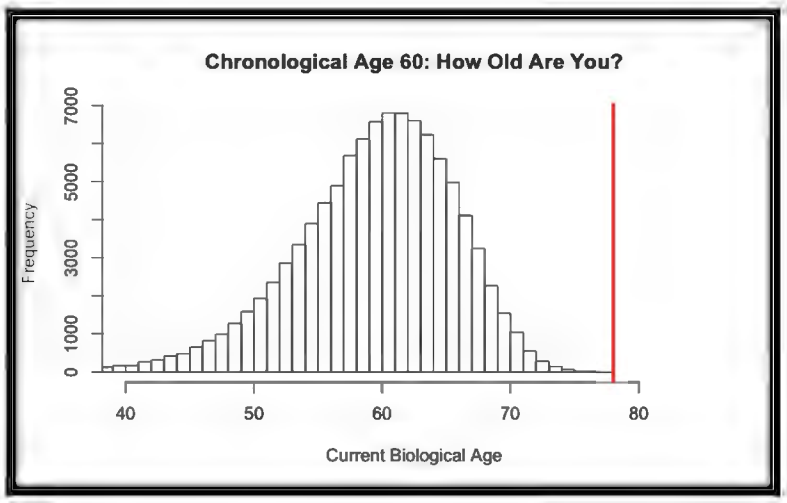

Final Takeaway

Make longevity risk salient by focusing attention on true age.

Are you insured against getting younger?

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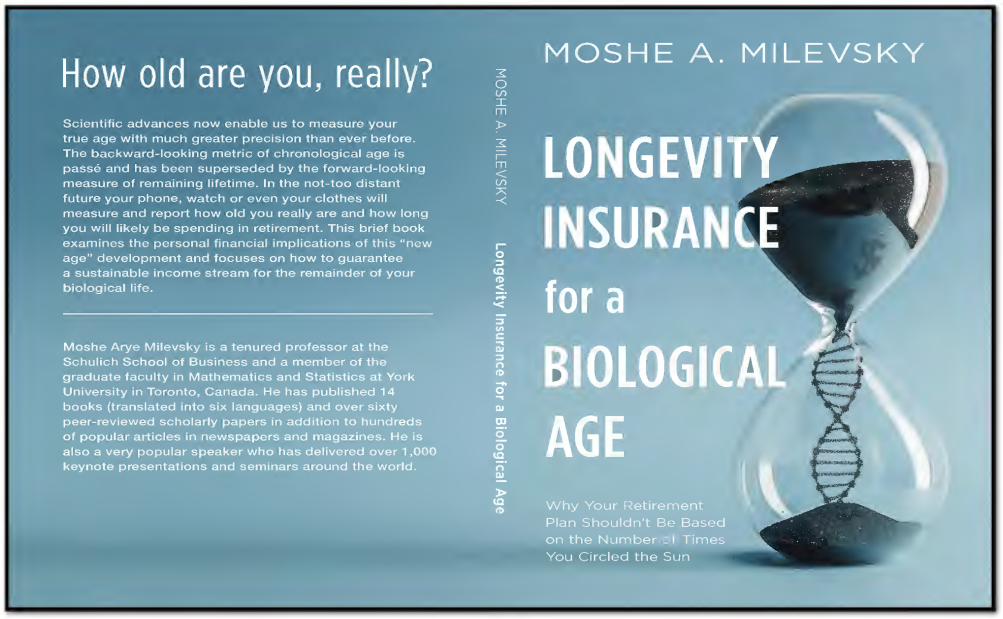

Calibrating Gompertz in Reverse: Mortality-adjusted Biological Ages around the World

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4 April 2019

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Abstract

Calibrating Gompertz in Reverse:
Mortality-adjusted Biological Ages around the World

The phrase *biological age* has become quite trendy in the media and popular press. And yet, while chronological age doesn't require much explanation or definition, the same can't be said for *biological age*. To provide an actuarial perspective on the matter, in this paper I present a statistical methodology for inverting the Gompertz-Makeham law of mortality for heterogenous populations assuming a compensation law of mortality (CLaM), to formally define a mortality-adjusted biological age (BA). Including the CLaM relationship within the estimation procedure simplifies the expression for BA and is arguably more accurate. I then provide some numerical estimates by applying this framework to a set of global mortality rates from the Human Mortality Database. Among other things, I demonstrate that the Biological Age of a 55 year-old Scandinavian male is 48, whereas a 55 year-old Russian male is closer in age to 67; and perhaps should be entitled to retire. The motivation for this (novel) method of presenting age and aging, is that this metric could potentially be used for pension and retirement policy in a world of growing mortality heterogeneity (awareness) and the need for salient (behaviorally motivated) longevity metrics – beyond simple life expectancy – that can capture the public's attention.

JEL: G22 (Insurance)
Keywords: Pensions, Insurance, Retirement, Longevity, Salience

1 Introduction and Motivation

Researchers in a variety of medical fields are uncovering bio-markers of aging that measure an individual's so-called *true* physiological age. his leads to more accurate forward-looking mortality rates and life expectancy estimates. The adjustment processes, which are similar to age setbacks, are a refined form of the underwriting employed by life insurance companies for centuries. What has received less attention in the insurance literature is how to map estimated mortality rates into an actuarially consistent *biological age*, a term which has become quite fashionable as of late. So, in this paper I present a statistical methodology that reverses the Gompertz-Makeham (GM) model for heterogenous populations assuming a compensation law of mortality, to then solve for a mortality-adjusted Biological Age (BA). A two-stage process which will be carefully explained over the next few pages. The end-result is a simple formula that maps an individuals chronological age x, into a chronological age ξ, resulting in a number which might be higher than x (indicating poor relative health and longevity prospects) or lower than x (indicating better relative health and longevity prospects). On a behavioral insurance and economics level, this might be an alternative way of presenting longevity metrics to a wider public who struggle with probabilistic concepts.

Now, to modern practicing actuaries the Gompertz model – which will soon celebrate its 200 year anniversary – might appear as an archaic remnant of an era prior to computers and spreadsheets. And yet, the simple analytic model remains quite popular and appears regularly in the pages of *Insurance: Mathematics and Economics*. In fact, as a testament to his legacy, Benjamin Gompertz is being cited with increasing frequency[1].

The remainder of this paper is organized as follows. The next section (#2) provides a brief overview of what is commonly meant by biological age and the two different philosophies one can employ to compute this number. Section (#3) provides an overview of the methodology I propose. Section (#4) gets into the details and the role of the *compensation law of mortality*, which was introduced by Gavrilov and Gavrilova (1991). Readers interested in the numerical values of biological ages around the world can skip to section (#5) and the associated tables. Finally, section (#6) concludes the paper and offers some suggestions for additional applications within the context of heterogenous mortality.

[1]According to google scholar, no less than 100 papers published in *IME* over the last decade or so, have assumed and/or cited a non-trivial Gompertz formulation in their analysis. In particular Gompertz remains quite popular in (i.) the valuation of annuities, (ii.) retirement income strategies and (iii.) stochastic mortality models. See for example: Angoshtari et al. (2016), Chen and Vigna (2017), Cohen and Young (2016), Dahl (2004), Delong and Chen (2016), Deelstra et al. (2016), Donnelly et al. (2014), Donnelly et a. (2013), Feng and Yi (2019), Fung et al. (2014), Gao et al. (2015), Haberman et al. (2011), Hainaut (2016), Jevtic et al. (2013), Luciano et al. (2012), Luciano and Regis (2014), Melnikov and Romaniuk (2006), Menoncin and Regis (2017), Meyricke and Sherris (2013), Moore (2009), Petrichev and Thorp (2008), Pitacco (2004), Shapiro (2013), Su and Sherris (2012), Valdez et al. (2014), Villegas and Haberman (2014), Wang (2009) and Willemse and Kaas (2007). So, despite his very old-age, Benjamin Gompertz continues to be a working horse for modeling longevity.

2 Biological Age: Defined and Explained

Generally speaking there are two (very) different approaches on how to compute and measure biological age. The difference between the two methodologies or viewpoints isn't just a matter of computational technique but is in fact motivated by one's background, discipline and field as well as the intended usage of the number. And, although researchers themselves don't exactly use these terms, I'll label the two approaches the "living" methodology versus the "dying" methodology, a.k.a. the mortality-adjusted approach I'll adopt.

Stated quite simply – and perhaps too simply – in the former (living) approach the benchmark for measuring true biological age is other people who are alive, and for the latter approach the benchmark is people who are dead. I'll start by explaining the former.

2.1 The Living Approach

Under this methodology a researcher would gather data on a very large group of people at a wide range of ages and collect samples of their saliva, blood and urine, and use those samples to extract various physiological and molecular (DNA, RNA, etc.) variables. These measurements, which could number in the hundreds, might include (item #5, for example) red blood cell count, (6) hemoglobin concentration, (7) total cholesterol, all the way to items such as (72) fasting blood sugar levels, (73) urine specific gravity, (74) triglycerides, or (94) the average telomere length (ATL), which for a while was the leading biomarker for aging, and is associated with the work of (Nobel laureate) Elizabeth Blackburn.

These physiological and molecular variables might then be augmented by physical variables (i.e. more easily measured, not requiring a laboratory) measuring things like (123) hand grip strength, (124) visual perception, or even (125) the number of missing teeth. Some social researchers go so-far as to augment their dataset with social variables, such as (179) number of friends on Facebook, or a binary variable measuring whether they like to garden (185). The theory here is that anything remotely associated with the behavior and characteristics of older people can be added as a data point for measuring age.

Once the data has been collected, each one of the elements is coded as a numerical score and every person in the sample is now associated with a vector of (for the sake of argument) 200 numbers, including their gender. But the most important number, which one can visually imagine as being stored at the very beginning of this long vector, is the individual's chronological age. Again, I'm describing the *living* approach. Denote the i'th person's chronological age by the symbol $y(i)$, and the vector of physiological, molecular, physical and social characteristics by $x(j, i)$, where the index letter j ranges from 1 to 200. For example, the variable $x(179, 98706) = 7$ would indicate that for individual number $i = 98706$, the sample's 179'th element (i.e. number of Facebook friends) was equal to a value of 7, (a number inspired by the author's count.)

The (usually medical) researcher would then run a multivariate (usually linear) regression of $y(i)$, as the dependent variable, on $x(j,i)$, the independent variables to obtain the best fitting function in the sense of least squares, etc. Variables that are not statistically significant are discarded (e.g. perhaps Facebook friends) and the multivariate regression is estimated again (and again) until the process converges on a collection of variables that relate (i.e. predict) the dependent variable, chronological age. So, the best fitting regression equation becomes the (magic) formula for biological age, while the individual errors in the regression are the gaps between a person's chronological age and biological age.

In the end, the statistically significant coefficients in this regression are declared as relevant biomarkers of aging. And, the sign of the coefficients (a.k.a. factors), whether positive or negative, would determine whether scoring higher in those elements effectively makes you younger or older. To repeat, this is not the approach taken in this paper, but is what many (if not most) researchers imply by the term *biological age*. Ergo, I am compelled to explain what I am not doing, and why.

2.2 Methodological Concerns

Here lies the problem or concern with the *living* approach. The implied biological age is based on how similar you are to other people, as opposed to directly estimating how long you are going to live or how soon you are likely to die – which would be of interest to the insurance economist or mathematician interested in describing human longevity. Yes, it's implicitly assumed that older people are more likely to die sooner, so the older your regression-measured biological age, the lower your life expectancy. But, for the most part, in this approach mortality is not involved directly, nor does this approach care about what is likely to kill you. The basic dataset is a cross section of live people at different ages.

Now, in some clinical studies, researchers track large groups of people over time to examine if the older ones (biologically) are more likely to die or if they did not live as long as their identically-aged neighbors, but it's an afterthought and obviously requires very long periods of time (decades, really) to establish. So, while the above approach doesn't ignore death, its focus is mainly on keeping people alive and in "young" health.

In sum, most equations for biological age come down to locating people you are most similar with and, more importantly, is primarily concerned with predicting functional impairments or the risk of chronic diseases. These researchers are interested in maximizing health span, not necessarily lifespan, which is why death and mortality rates aren't the focus of their attention. There are other concerns with this approach, mostly related to the statistical significance of regressions with multiple independent variables, data mining, as well as concerns about linearity assumptions, etc.[2]

[2]See Dubina et al. (1984), Jackson et al. (2003), Jylhava et al. (2017) and Ries and Pothig (1984), for more on this (conventional) approach to biological age estimation and their many references.

2.3 The Dying Approach

In contrast to the "living" methodology, the mortality-adjusted *biological age* approach is, as the name suggests, based on people who have died and is concerned with something that is much less complex than the multifaceted aspects of aging. Rather, it simply wants to better predict calendar time until death, or what might be designated T_x, where x is chronological age. The process begins by collecting data on mortality rates as a function of chronological age plus other characteristics or elements. For example, this might include the number of cigarettes the now-deceased smoked (from zero to a thousand) before they died, or their body mass index (in kilograms per meter squared) before they died or their triglyceride level (in milligrams per deciliter) before they died.

The "dying" approach for measuring biological age also involves a regression process, but the dependent variable, denoted by the familiar q_x, is now a mortality rate, as opposed to a chronological age. The statistical model is about finding elements that are associated with higher death rates, together with a formula that maps those elements into a mortality rate. In contrast to the living approach, chronological age is not the dependent variable, nor is anyone trying to predict it directly.

For example, the best fitting regression equation for your mortality rate might be estimated to include a variable which is one minus the ratio of the average length of your telomeres in your body (in units of nucleotides) to the number 10,000 (for example) in the year prior to death. So, if the average length of your telomeres is 9,900 nucleotides, then your forecast mortality rate is (1-99/100)=1% in that year, all else being equal. In contrast, if the average length of the telomeres in your body is 8,000 nucleotides, the one-year mortality rate would be 20%.

This is a toy example, but the point is to focus directly on mortality rates in the estimation process. In fact, within this approach, one might also include an individual's wealth and income as well, and I'll briefly touch upon that in concluding section (#6), but it's more common to focus on (obvious) factors that affect mortality such as alcohol consumption, smoking, body mass index, physical activity, quality of sleep, blood pressure, resting heart rate and perhaps even how much time you spend walking in a given day. All of these factors are segmented or clustered into groups and tested to examine whether they impact mortality rates conditional on chronological age.

Now, to the essence of this paper, the only factor (groups) I'll focus on is nationality, using freely available data from the human mortality database (HMD). Just as importantly, the computation of biological age involves mapping from mortality rates to an assumed age. This is precisely where the Gompertz-Makeham (GM) law of mortality is used. In particular, I obtain mortality-adjusted biological ages by inverting the GM formula, taking as input both global and local mortality rates and then solving for the implied age ξ. With the big picture in place, I now move on to technicalities.

3 Conceptual Model: From C-Age to B-Age.

I assume that every life in the *local* group denoted and indexed by: $i = 1..N$, obeys a Gompertz-Makeham (GM) mortality law. (In the estimation procedure I'll use $N = 37$ countries.) For every group i, the continuous mortality hazard rate is modeled as:

$$\lambda_x[i] \;=\; \lambda[i] + h[i]\, e^{g[i]\, x}, \tag{1}$$

where x denotes chronological age, $g[i]$ is the mortality growth rate (MGR), $h[i]$ is the (hypothetical) age-zero mortality hazard rate, a.k.a. initial mortality rate (IMR), and $\lambda[i]$ is the accidental (non-age related) hazard rate, a.k.a. Makeham constant. This formulation implies that the log-hazard rate minus the accidental rate: $\ln[\lambda_x[i] - \lambda[i]]$, is a linear function of chronological age x, with intercept $\ln[h[i]]$ and slope $g[i]$.

Recall that the range over which the GM relationship is valid (and log hazard rates are linear) is somewhere between $x \approx 35$ and $x \approx 95$ (which is what I later use for estimation), and is an empirical matter. To be clear, each one of the i countries will have their own set of: $(\lambda[i], h[i],\, g[i])$, values. Note that both: $h[i]$ and $\lambda[i]$ tend to be very small numbers, on the order of 10^{-5}, so I will mostly work with and display the natural log values.

Moving on, I assume the existence of a biological age denoted by ξ, which satisfies its own GM-like relationship for the continuous mortality hazard rate:

$$\Lambda_\xi \;=\; \Lambda + H e^{G\,\xi}, \tag{2}$$

where these three constants: Λ, H, G, represent *global* GM parameters, or perhaps a *world index* for mortality, relative to which local ages are measured. The exact procedure will be described, but for now one can think of the global values as a sort of *nonlinear average* of the vector of local values. I should warn though that the averaging process is **not** linear, **if** the procedure is to be consistent with the *compensation law of mortality*, which I'll return to in a moment. Note also that the average (or sum) of Gompertz random variables isn't Gompertz, which is another reason not to average $h[i]$ values and set them equal to H.

To our main objective, the biological age $\xi := \xi(x, \lambda[i], h[i], g)$, in locality or group i, is defined by equating the mortality hazard rate at x, and solving for the implied ξ. Formally:

$$\Lambda_\xi \;= \lambda_x[i]. \tag{3}$$

Inverting the GM formula determines the mortality-adjusted biological age ξ. Equating equation (1) and (2), and dispensed with the $[i]$ index on the local λ, h, g, values, we have:

$$\xi \;=\; \frac{\ln[\lambda - \Lambda + h e^{gx}] - \ln[H]}{G}. \tag{4}$$

In some sense, equation (4) is the main equation in the paper, and the remainder is implementation. It maps age x, local (country) Gompertz parameters (λ, h, g) and global Gompertz parameters (Λ, H, G) into the mortality-adjusted biological age. Now, if by chance or assumption the local value of Makeham's $\lambda \approx \Lambda$, then equation (4) is simply:

$$\xi \;=\; \left(\frac{\ln[h/H]}{G}\right) + \left(\frac{g}{G}\right) x. \tag{5}$$

Equation (5) is a special case of equation (4) which is (only) valid when we the age-independent accidental death rate λ, is identical across all *local* regions, at least within the Gompertzian age range. Intuitively, we can easily see from equation (5) how biological age ξ, collapses to chronological age x when the local initial mortality rate is equal to the global value: $h = H$, and the local mortality growth rate is equal to the global value: $g = G$.

Equation (5) also demonstrates that when $g = G$, and the mortality growth rates are equal, but $h \neq H$, the biological age ξ is a linear shift (a.k.a. the popular age set-back) of the chronological age x, by: $\ln[h/H]/G$ years. The age set-back approach is common in practice, but is technically inconsistent with the *compensation law of mortality*, which I'll get to in the next section. Stated differently, if $h \neq H$, then it follows $g \neq G$ as well.

Before I move-on to implementation, here is a simple application of equation (5) to set the intuition. Assume that in Mauritius the value of $h[i]$ is estimated to be: 15×10^{-5}, and the corresponding value of $g[i]$ is estimated at: 0.06, that is a 6% mortality growth rate. Now, generally speaking, high values of h are associated with lower values of g, and vice versa. This is the essence of the *compensation law of mortality*. So, to be consistent, assume the global average values are: $H = 1 \times 10^{-5}$ and $G = 0.09$, both of which are completely fictitious (for the example) at this point. Well then, an $x = 65$ year-old in Mauritius, according to equation (5), has a mortality-adjusted biological age of: $\xi = 73.4$.

3.1 In sum: The Four Steps from C-Age to B-Age

1. Start with a vector of country mortality rates: q_x. They are converted to continuous mortality hazard rates λ_x, in a manner consistent with the GM law, (since $\lambda_x \neq q_x$.)

2. Use (iterative) regression techniques to estimate the local GM parameters $(\lambda[i], h[i], g[i])$, in each one of the i groups.

3. Using the N sets of three parameters, estimates a second regression to solve for the global values of Λ, H, G, in a manner consistent with the CLaM.

4. Finally, select local: $(\lambda[i], h[i], g[i])$, and reference ages, such as $x = 55$ and solve for mortality-adjusted biological age using a modified version of equation (4).

4 The Detailed Procedure

4.1 Step One: Estimating Sub-Group Parameters

Each group member is identified by five unique parameters: $\{h[i], \lambda[i], g[i], x^*[i], \lambda^*[i]\}$, where $i = 1..N$, is the number of groups. A group (in this paper) is just a country, but it could represent the level of nicotine in blood, or any other observable characteristic for which mortality rates are available. The first parameter $h[i]$, represents the initial mortality rate (IMR) for group i. Note that the IMR isn't the infant mortality rate, which is much higher than $h[i]$. Indeed, age $x = 0$ is well outside the GM regime. The second parameter is the accidental (Makeham) term $\lambda[i]$. The third term $g[i]$ is the corresponding mortality growth rate (MGR) during the *Gompertzian* range of life $x < x^*$. The fourth parameter x^* denotes the age at which the *Gompertzian* regime ends – also known as the species specific lifespan, per Gavrilov and Gavrilova (1991). Then, mortality rates plateau to a (fifth and final) parameter value $\lambda^*[i]$. At this point, I assume no other restrictions other than: $h[i] \geq 0$ and $g[i] \geq 0$. See Figure #1 for a visualization. **[Figure #1 goes here.]**

Written formally and more precisely than equation (1), I assume the continuous mortality hazard rate will obey the following relationship:

$$\lambda_x[i] \;=\; \begin{cases} \lambda[i] + h[i]e^{g[i]x} & x < x^*[i] \\ \lambda^*[i] & x \geq x^*[i] \end{cases} \tag{6}$$

Note that the plateau mortality hazard rate: $\lambda^*[i] >> \lambda[i]$ and the corresponding age x^*, is group (i.e. country) specific. Later on I'll assume one global λ^* and x^* which are shared by all groups, as per the strict version of the *compensation law of mortality*. Nevertheless, it's worth emphasizing again that equation (6) is quite general. First, the plateau could depend on i, that is $\lambda^*[i] \neq \lambda^*[j]$, for $i \neq j$. Furthermore, for some i, it's conceivable $x^*[i] \to \infty$, and there is no (finite) mortality plateau. In other words, other than an assumption of a *Gompertz-Makeham* regime, there is no mention (yet) of any analytic relationship between the groups (i.e. countries). Rearranging, the GM model can be expressed as:

$$\overbrace{\ln(\lambda_x[i] \;-\lambda[i])}^{Q_x} \;=\; \overbrace{\ln h[i]}^{C_0} + \overbrace{g[i]}^{C_1} x, \quad \forall\, x < x^*[i], \tag{7}$$

which is the standard linear representation of (log) mortality rates for all ages within the GM regime. I deliberately use Q_x, on top of the brace and not the natural log of the one-year death rate $\ln[q_x]$, since q_x and λ_x are (obviously) not the same thing. Although they are close for small values of q_x, mortality hazard rates can (obviously) exceed the value of one[3].

[3]Most published estimates of C_0 and C_1 in the economic literature on mortality heterogeneity, such as Chetty et al. (2016), or Milligan and Schirle (2018) use $\ln[q_x]$ on the left-hand side of the above regression.

Recall that q_x, at any given chronological age x, is related to the continuous mortality rate λ_x, via:

$$1 - q_x \;=\; e^{-\int_x^{x+1} \lambda_y dy}. \tag{8}$$

Now, when $\lambda_x = \lambda$, is constant (i.e. $h = 0$), the survival rate to any time t is $s(t) = e^{-\lambda t}$, and then $q_x = 1 - e^{-\lambda}$, for any one year. In this (simplistic, clearly non-Gompertz) case, the parameter λ_x is synonymous with a *continuously* compounded mortality rate and q_x is the *effective* annual (one year) death rate. In the full Gompertz-Makeham ($h > 0$) case, equation (8) leads to the following relationship between q_x, and the model parameters (λ, h, g):

$$-\ln[1 - q_x] \;=\; \lambda + he^{gx}\left(e^g - 1\right)/g \tag{9}$$

Note that by definition, the mortality hazard rate $-\ln[1 - q_x] > \lambda \geq 0$, so we can subtract λ from both sides, take logs (again) and obtain a linear relationship between the one-year death rate q_x and age x. I write this explicitly for each of the group parameters:

$$\overbrace{\ln\left(\ln\left(\frac{1}{1 - q_x[i]}\right) - \lambda\right)}^{z} \;=\; \overbrace{\ln[h[i]] \;+\; \ln[(e^{g[i]} - 1)/g[i]]}^{K_0} + \overbrace{g[i]}^{K_1}\; x, \tag{10}$$

where the new constants (K_0, K_1) are defined for convenience and suggests the proper regression methodology for calibrating GM values of λ, h, g from one-year mortality rates q_x. To be clear, $\ln[\ln[(1-q)^{-1}]] \approx \ln[q]$ for small values of q, but one does introduce errors when $\lambda \neq 0$ and/or when $q >> 0$. At the very least, if one insists on using equation (7), it would be proper to use $\ln[q] + q/2$ as the dependent variable, to match the first two terms of the Taylor series expansion[4]. For now, this leads to the (basic) Gompertz regression equation:

$$z_j \;=\; K_0 \;+\; K_1 x_j + \epsilon_j, \tag{11}$$

where x_j is a vector of ages, for example $x_1 = 35, x_2 = 36, x_3 = 37$, etc., and the z_i are computed from the one-year death rates q_{x_i}. See Figure #2 for a visualization of the data. **[Figure #2 Goes Here]** Note that to properly handle and estimate Makeham's $\lambda[i]$, which should not be assumed to be zero, I implement an iterative procedure in which $\lambda[i]$ starts at zero and is increased in units of 10^{-5} until the GM regression error is minimized. Caution is warranted here since the Makeham term obviously can't exceed the IMR, which is why I capped the iteration at 20. Minutia and details aside, once the unique and group-specific value for $\lambda[i]$ is located, the associated regression formulated in equation (11) leads to the best-fitting intercept and slope parameters $\tilde{K}_0$ and $\tilde{K}_1$.

[4] I refer interested readers to the recent work by Tai and Noymer (2018) for a full and proper discussion of the many ways in which to estimate Gompertz parameters from one-year mortality rates, and in particular the approach that minimizes root mean square (RMS) error for life expectancy estimates.

More importantly, based on equation (10), the unbiased estimates for the GM parameters, for each one of the groups, is:

$$g[i] = \tilde{K}_1, \quad \ln[h[i]] = \tilde{K}_0 - \ln[(e^{\tilde{K}_1} - 1)/\tilde{K}_1], \quad h[i] = \tilde{K}_1 e^{\tilde{K}_0} \left(e^{\tilde{K}_1} - 1\right)^{-1} \tag{12}$$

To recall, these are the mortality growth rate (MGR), the log mortality hazard rate and the initial mortality hazard rate (IMR) at age zero, for each of the i groups. I refer to this entire process as the first-stage regression, although in reality each group-set of $h[i], g[i]$ values requires multiple regressions until the error-minimizing value of $\lambda[i]$ is located.

For those researchers who are (more) used to the probabilistic formulation of the Gompertz law in terms of modal value m and dispersion coefficient b, the mortality hazard rate is expressed as: $\lambda + e^{(x-m)/b}/b$. So, the final conversion from (h, g) to (m, b) would be $b = 1/g$ and $m = (\ln[g] - \ln[h])/g$. Both the (h, g) and (m, b) parameter estimates are displayed in Tables #1a, #1b. **[Table #1a, #1b Goes Here]**

Now in theory we could stop (the regressions) here and compute average (global) values Λ, H, G and use those together with the individual values of $\lambda[i], h[i], g[i]$ to compute mortality-adjusted biological age via equation (4). However, this would ignore the theoretical fact that (i.) the sum of Gompertz variates isn't Gompertz, and (ii.) the *compensation law of mortality* forces a strict relationship between h and g. The expression for mortality-adjusted biological age I propose will (conveniently) account for both.

4.2 Step Two: Including CLaM

The weak-form of the *compensation law of mortality* states that groups with relatively higher initial mortality hazard rates: $h[i] > h[j]$, experience relatively lower mortality growth rates $g[i] < g[j]$, and vice versa. See Gavrilov & Gavrilova (1991, 2001) for more about this important but often neglected natural phenomenon. In other words, the CLaM posits a formal analytic relationship between $h[i]$ and $g[i]$, denoted by $\vec{h}(g)$, within a range of: $g_{\texttt{min}} \leq g \leq g_{\texttt{max}}$. To be clear, the weak form CLaM (only) stipulates that: $\partial\vec{h}(g)/\partial g < 0$, if one thinks of h as a function of g.

In contrast to the weak form, a strong-form CLaM begins at the very end of the lifecycle by postulating that: $x^*[i] = x^*, \forall i$, and the mortality plateau is identical for all sub-groups. This actually places much tighter restrictions on the function $\vec{h}(g)$, and by equation (6) implies:

$$L \;:=\; \ln(\lambda^* \; - \lambda[i]) \;=\; \ln h(g) + g x^*, \tag{13}$$

where L is a new and convenient constant. Rearranging equation (13) leads to a linear representation for the function: $\ln \vec{h}(g)$, and can be expressed as:

$$\ln \vec{h}(g) \;=\; L \; - x^*\, g, \tag{14}$$

I will refer to $\ln \vec{h}(g)$, as the CLaM line relating a specific mortality growth rate g to corresponding log initial mortality rate $\ln \vec{h}(g)$. Exponentiating equation (14), the initial mortality hazard rate: $\vec{h}(g)$ can be expressed as: $\vec{h}(g) = e^{L - x^* g}$, which at $g = 0$ recovers the mortality plateau: $\lambda^* = \vec{h}(0) + \lambda$. Therefore, under the strong *compensation law of mortality*, I can rewrite equation (6) as:

$$\vec{\lambda}_x(g) \;=\; \begin{cases} \lambda + (\lambda^* - \lambda) e^{g(x - x^*)} & x < x^* \\ \lambda^* & x \geq x^* \end{cases} \tag{15}$$

The function $\vec{\lambda}_x(g)$ is meant to remind readers that under a strict CLaM the mortality hazard rate is driven and dictated (only) by the mortality growth rate. That is the only degree of freedom. Either way, I now have N values of: $\{\ln h[i], \lambda, g[i]\}$, and *assuming* they are consistent with the strong form of the CLaM, I can estimate the (intercept) L, and (slope) x^* via a second phase regression involving all countries.

In particular, as per equation (14), the relationship is:

$$\overbrace{\ln h[i]}^{w_j} \;=\; \overbrace{L}^{C_0} \;+\; \overbrace{(-x^*)}^{C_1} \, g[i] \;+ \epsilon_j. \tag{16}$$

Note that this second phase regression can't be merged with the first phase regression procedure that is used to estimate the original Gompertz parameters in equation (10), given the need for country-specific values of Makeham's $\lambda[i]$. Also, the first regression leads to the: $\ln h[i], \lambda[i]$ and $g[i]$ values in Table #1a and #1b, which might be of independent use and interest to the many researchers who work and use the Gompertz-Makeham law for pricing annuities or modeling decumulation strategies, as cited in footnote #1.

Moving on, Table #2 [**Table #2 Goes Here**] displays the estimated values using the individual GM parameters, which effectively test for the presence of a strong CLaM in the data. Indeed, the relationship between: $\ln h[i]$ and $g[i]$ is linear with R^2 values close to 98%, providing support for a strong version CLaM for the $N = 37$ countries examined. Finally, the estimated $L = \ln(\lambda^* - \lambda)$, reveals or locates the mortality hazard rate once the plateau is reached. And, the slope $(-x^*)$ is the age at which it's achieved, a.k.a. the *species specific lifespan*. See Figure #3 [**Figure #3 Goes Here**] for a visual indication of the strong negative relationship between the two variables.

4.3 Stage Three: Baseline Population Rates

We now have a set of diverse Gompertz Makeham parameters as well as λ^*, x^* estimated for each of the countries. Back to the computation of mortality-adjusted biological age, we now require population average Λ, H, G values so that we can invert the GM equation and map one-year death rates into biological ages, as per equation (4).

But by this point in the narrative it should be clear that one can't average *both* the individual $h[i]$ and the individual $g[i]$ values estimated in stage one. There is only one degree of freedom according to the CLaM. Rather, I suggest the proper way to obtain global GM parameters is to locate the implied $h[i]$ in a manner that is consistent with CLaM for that country's particular mortality growth rate $g[i]$. In other words average the mortality growth rates to arrive at global value of G and then use the CLaM line to obtain an implied H, which in fact will drop-out in the process. The details will be carefully explained in the next section.

4.4 Stage Four: Computing Biological Age

The mortality-adjusted biological age: $\xi(x)$, of someone whose chronological age is currently x, is defined and constructed by equating mortality hazard rates, per equation (3). With the CLaM in place, we proceed by assuming that $\vec{\lambda}_x(g)$ is classified entirely by the mortality growth rate g, so there is no need to explictely use the initial mortality rate h. In particular, the definition of mortality-adjusted biological age from equation (3) can now be written as:

$$\vec{\lambda}_\xi(G) \;=\; \vec{\lambda}_x(g[i]). \tag{17}$$

To be clear, all that is needed at this point is the parameter $g[i]$ (the country or group specific mortality growth rate), and the G (global average mortality growth rate). The average is defined:

$$G \;=\; \frac{1}{N}\sum_{i=1}^{N} g[i]. \tag{18}$$

Referring to the formulation expressed in equation (15), our next objective is to eliminate some redundant terms. We start by writing the defining equality as:

$$\Lambda + (\lambda^* - \Lambda)e^{G(\xi - x^*)} \;=\; \lambda[i] + (\lambda^* - \lambda[i])e^{g[i](x-x^*)}. \tag{19}$$

The objective now is to solve and isolate the biological age ξ as a function of the (estimated) regression parameters. Equation (19) can be expressed as:

$$\Lambda\left(1 - e^{G(\xi - x^*)}\right) \;-\; \lambda[i]\left(1 - e^{g[i](x-x^*)}\right) \;=\; \lambda^*\left(e^{g[i](x-x^*)} \;-\; e^{G(\xi-x^*)}\right) \tag{20}$$

At this point let's focus attention on the global (average) Makeham constant, denoted by Λ and the local (country specific or regional) Makeham constant, denoted by $\lambda[i]$. Recall that the latter quantity was estimated in the first stage regression. To be very clear, the country specific $\ln h[i]$ (intercept) and $g[i]$ (slope) values from the regression were estimated jointly with the $\lambda[i]$ values. Every one of the N mortality vectors listed in Table #1a and #b, includes a unique error-minimizing $\lambda[i]$.

But alas, at this point I will proceed by assuming that the left hand side of the above equation (20) is very close to, and without much loss can be approximated to be, zero. Why? First, recall Λ and $\lambda[i]$ are quite small to begin with, on the order of 10^{-5}, and both of them are multiplied by a number (very) close to zero and then subtracted from each other. Hence, I proceed by assuming that although $\Lambda \neq \lambda[i]$, the left-hand side of equation (20) is effectively zero. And, after dividing both sides of the equation by λ^*, what is left is to isolate ξ from the equation:

$$e^{G(\xi - x^*)} \;=\; e^{g[i](x - x^*)} \tag{21}$$

Voila, this leads to the mortality-adjusted biological age. It is a restricted (but more compact) version of the more general equation (4), because equation (21) is built on the backbone of the *compensation law of mortality*. It can be additionally expressed as:

$$\xi \;=\; \left(\frac{g[i]}{G}\right) x + \left(1 - \frac{g[i]}{G}\right) x^* \;=\; x - \kappa[i](x^* - x), \tag{22}$$

where the new constant: $\kappa[i] = (g[i]/G) - 1$, is defined as the *relative* aging rate. Mortality-adjusted biological age is expressed (only) as a function of (i.) the relative aging rate κ, and (ii.) the series specific lifespan x^*, a.k.a. the plateauing age. And, if the mortality growth rate $g[i] = G$, which is the global average rate, well then the mortality adjusted biological age is equal to chronological age.

Here is a numerical example. Assume the mortality plateau is estimated to occur at age: $x^* = 110$, current chronological age is: $x = 50$, the mortality growth rate is: $g[i] = 10\%$, the population (global) mortality growth rate is: $G = 9\%$, then the relative aging rate is: $\kappa_i = 11.11\% = 1/9$. For these parameters the estimated biological age is: $\xi = 50 - (110 - 50)/9 = 43\frac{1}{3}$. So, this individual is 6.66 years younger than their chronological age. There is no mention of the initial mortality rate (IMR) which is buried inside x^*.

Note once again that equation (22) is similar to – although not exactly – equation (5). The difference between the two expressions is that in the above equation (22) there is no mention of the initial mortality rate $h[i]$, or the global average H.

Before I move-on to the numerical estimates of mortality-adjusted biological ages around the world, here are some things to note. First, at the chronological age $x = x^*$, the mortality-adjusted biological age ξ converges to x, as per equation (22). I have assumed mortality hazard rates are constant (from there). It's an open empirical question as to what happens after the species specific lifespan. But, without any loss of generality for the definition of mortality-adjusted biological age, from that point onward I assume lifetimes are exponentially distributed. So, for the sake of completeness $\xi = x$ after x^*. In other words, there is no crossover in log-mortality rates.[5]

[5]To this point I should note that Cairns et al. (2019) write in relation to the *compensation law of mortality* that "...we have not found any evidence that groups cross over, even at very high ages..."

Note also that all else being equal, a larger value of x^*, lowers the mortality-adjusted biological age ξ. The same is true for larger values of $\kappa[i]$. Indeed, it's worth emphasizing that when the value of $\kappa[i] > 0$, aging for that group (i.e. country) is faster than average ($g[i] > G$), and yet mortality-adjusted biological ages, ξ are lower than chronological age, x. This might seem odd at first, but is driven by the *compensation law of mortality* which underlies equation (22). Note that a value of $g[i] > G$, is associated (under CLaM) with an initial mortality rate: $\vec{h}(g[i]) < \vec{h}(G)$, where $\vec{h}(.)$ is expressed as a function of the group's mortality growth rate. Stated graphically, this person is on a lower curve within Figure #1, so they are biologically younger. If you are (biologically) younger than someone who shares your birthday, you must be aging faster than they are.

A third and convenient thing to note (again) is that the link between chronological age x and biological age ξ doesn't require explicit knowledge of the current hazard rate $\lambda_x[i]$ either, because (again) they are embedded (implicitly) in the parameters: x^* and $\kappa[i]$. So, assuming the CLaM is not only realistic with an interesting theory of aging, but also reduces the number of individual parameters required to estimate mortality-adjusted biological age. And so, with the formal modeling out of the way I'll move on to report results.

5 Estimates: Biological Ages Around the World

Table #3 [**Table #3 Goes Here**] displays numerical results from the methodology described in the prior section, using period mortality rates (between chronological ages $x = 35$ to $x = 95$) for 37 countries from the human mortality database (HMD) in the year 2011. That is the most recent year for which the largest and most complete country data is available. Each of the 37 mortality rate vectors $q_x[i]$ were used to iteratively estimate the best fitting $(h[i], \lambda[i], g[i])$ values for males and females separately. This is what I call the *first* stage regression, although there are 37×2 regression in total. Those results are displayed in Table #1a and Table #1b, alluded to earlier.

With those (first stage regression) numbers in hand I then estimated the second phase regression – separately for males and females – to obtain the best fitting *compensating law of mortality* line combining the mortality rates of these 37 countries, as per the graphic display in Figure #3. The parameter estimates for that second phase regression are presented in Table #2. In particular, the estimated male plateauing age was $x^* = 95.7$ and the female plateauing age was $x^* = 99.9$. The standard error for those point estimates was approximately 2 years for males and 5 years for females. In other words, its entirely plausible that the mortality plateau occurs much later in life, perhaps even at the age of 110, which is (also) an open question. These (+2 standard errors) upper and (-1 standard errors) lower bounds are used in Figure #4 [**Figure #4 Goes Here**], to provide a graphical range for the mortality-adjusted biological age.

As for point estimates, an $x = 55$ year-old Russian male has a biological age $\xi = 66.7$, whereas an $x = 55$ year-old Swedish male has a biological age of $\xi = 47.93$, which is a gap of almost 20 years between the youngest and oldest in Table #3. Using the same formula for females with a unique $g[i]$ and higher x^*, the largest gap at age $x = 55$, is between the Italian whose biological age is $\xi = 51.7$ and the (again) Russian whose biological age is $\xi = 61.7$. The gap for females is (only) ten years. Indeed, the range of mortality growth rates $g[i]$ isn't as wide. Notice also that as one increases the chronological age from $x = 55$ to $x = 70$ and then $x = 85$, the gap between the highest and lowest mortality-adjusted biological age (country) shrinks. This is effectively the compensation law of mortality in action, and the mortality death rates $q_x[i]$ are converging as well.

Finally, Figure #3 goes beyond point estimates and displays the range of mortality-adjusted biological ages for each country, based on the upper and lower bound for x^*. It should be clear from the figure that as we move away from the mean age of $x = 55$, and the associated mortality growth rate $g[i]$, the spread increases. More precisely, the variable $\xi_{\texttt{low}}$ is computed using equation (22) in which two standard errors are added to the point estimate of the plateau age x^*, and $\xi_{\texttt{high}}$ is defined similarly but with two standard deviations removed.

6 Conclusion and Caveats

This exploratory and conceptual paper leverages the *compensation law of mortality* and the (inverse of) the Gompertz-Makeham model to develop a simple expression for mortality-adjusted biological ages, as a function of (i.) the relative mortality growth rate and (ii.) the plateauing age, a.k.a. the series specific lifespan, which is the (hypothesized) age at which all of mortality curves converge to a constant.

The expression derived in equation (22) was then calibrated to (adult) mortality rates in 37 countries from the Human Mortality Database (circa 2011) using a two-phase regression methodology. The first phase estimated the (country) mortality growth rates $g[i]$, and the second phase regression estimated the (global) series specific lifespan x^*.

Using this approach for measuring mortality-adjusted biological age, I demonstrate that at chronological age 55, the biological age gap between high-mortality and low-mortality countries can be as high as 20 years. The same exact methodology could be applied to heterogenous groups within a country or population, for example mortality rates based on income, wealth, race or education. All that is required from the key equation (22) is a relative mortality growth rate κ and a series specific lifespan x^*. The rest is algebra[6].

[6]Employing the main equation for mortality-adjusted biological age with Gompertz coefficients by income percentile in the US, as reported by Chetty et. al. (2016), result in a gap equivalent to the one between Sweden vs. Russia at age 55. See Milevsky (2019) for a discussion of mortality heterogeneity and CLaM in the context of longevity risk pooling.

Of course, one could just as easily use the same mortality rates to compare period life expectancy values between high-mortality and low-mortality groups and arrive at similarly large gaps between the two extremes. However, I would argue that there are behavioral (a.k.a. psychological) benefits to focusing on mortality-adjusted biological age versus life expectancy as these numbers are more memorable. In fact, on a technical note, by expressing the relevant integrals in terms of the Incomplete Gamma function, one can prove that the gap in mortality-adjusted biological age – for two individuals who share the same chronological age but are on opposite sides of G – will actually be larger than the gap in life expectancy.

For example, imagine we have (only) two groups, with mortality growth rates $g_P = 8\%$ and $g_R = 12\%$ respectively. Perhaps the former group is of low income (and poor health) while the latter has higher income (and better health.) Assuming a species specific lifespan of $x^* = 100$, an average mortality growth rate of 10% and a mortality plateau $\lambda^* = e^{-1}$, we can say the following: Integrating the survival probability from age 65 to age 100, the life expectancy at age $x = 65$ for all members of group P is: $E[T_{65}(0.08)] = 15.8$ years. For members of group R the equivalent number is: $E[T_{65}(0.12)] = 22.3$ years, and the population life expectancy would be $E[T_{65}(0.10)] = 19.3$ years. So, the life expectancy gap between the two sub-groups is approximately $22.3 - 15.8 = 6.5$ years at age 65. To compare, the mortality-adjusted biological age of the 65 year-old in group P is 72, versus 58 for group R. This according to equation (22). That is a gap of 14 years and is much more *salient.*

Arguably, notifying a 65-year-old that their (true) biological age is 58 is more impactful and might help them take action, such as delaying retirement. Compare this – again, with a behavioral framework in mind – to telling them their life expectancy is only 22 years, versus the population average of 19, and they should wait to draw their pension. Will it be as effective as informing them they are much younger than their chronological age?

This then opens the door to discussions around retirement policy that are geared towards demographic parameters. On a policy level perhaps retirement age should be based on biological age – which is a proxy for life expectancy – versus chronological age. This would obviously be controversial,[7] goes well beyond the technical scope of this article and might just be too far ahead of its time. Needless to say, the formal (scientific) definition of biological age has yet to be settled and some might argue that it never will be resolved. At the very least then, as we get close to celebrating the 200 year anniversary of the publication (initially 1820, then 1825) of the work of Benjamin Gompertz, this paper offers yet another application of his timeless model.

[7]See the paper by Stevens (2017) for a survey and discussion of the various ways to adjust retirement ages for increases in longevity and life expectancy. See also the writing of John Shoven at Stanford University.

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Table # 1a

Gompertz-Makeham Parameters Around the World: MALE

Country	$\ln h[i]$	$\lambda_0[i]$	*Makeham:* λ	$g[i]$	$\lambda_{55}[i]$	m	b
1. Australia	-10.922	21.8×10^{-5}	20×10^{-5}	10.09%	.485%	85.50	9.91
2. Austria	-10.585	3.5×10^{-5}	1.0×10^{-5}	9.91%	.590%	83.49	10.09
3. Belgium	-10.573	22.6×10^{-5}	20.0×10^{-5}	9.91%	.616%	83.38	10.09
4. Belarus	-7.563	52.9×10^{-5}	1.0×10^{-5}	6.89%	2.294%	70.97	14.52
5. Canada	-10.912	21.8×10^{-5}	20.0×10^{-5}	10.11%	.494%	85.26	9.89
6. Croatia	-9.963	5.7×10^{-5}	1.0×10^{-5}	9.56%	.905%	79.68	10.46
7. Switzerland	-11.428	20.1×10^{-5}	19.0×10^{-5}	10.77%	.426%	85.40	9.28
8. Czech	-10.041	5.4×10^{-5}	1.0×10^{-5}	9.58%	.846%	80.35	10.44
9. Germany	-10.526	3.7×10^{-5}	1.0×10^{-5}	9.89%	.620%	83.02	10.11
10. Denmark	-10.535	3.7×10^{-5}	1.0×10^{-5}	9.93%	.627%	82.84	10.07
11. Spain	-10.635	3.4×10^{-5}	1.0×10^{-5}	9.86%	.547%	84.35	10.14
12. Estonia	-8.915	14.4×10^{-5}	1.0×10^{-5}	8.23%	1.245%	77.95	12.15
13. Finland	-10.270	21.5×10^{-5}	18.0×10^{-5}	9.56%	.683%	82.89	10.46
14. France	-10.075	24.2×10^{-5}	20.0×10^{-5}	9.12%	.654%	84.25	10.97
15. Greece	-10.195	5.7×10^{-5}	2.0×10^{-5}	9.36%	.646%	83.59	10.68
16. Hungary	-9.141	11.7×10^{-5}	1.0×10^{-5}	8.64%	1.241%	77.48	11.58
17. Ireland	-10.902	21.8×10^{-5}	20.0×10^{-5}	10.26%	.539%	84.09	9.75
18. Iceland	-11.454	2.1×10^{-5}	1.0×10^{-5}	10.78%	.400%	85.57	9.27
19. Italy	-11.348	18.2×10^{-5}	17.0×10^{-5}	10.75%	.454%	84.79	9.30
20. Japan	-10.831	22.0×10^{-5}	20.0×10^{-5}	10.05%	.519%	84.88	9.95
21. Korea	-10.446	22.9×10^{-5}	20.0×10^{-5}	9.80%	.658%	82.88	10.20
22. Lithuania	-7.957	36.0×10^{-5}	1.0×10^{-5}	7.11%	1.753%	74.70	14.06
23. Luxemburg	-11.201	2.4×10^{-5}	1.0×10^{-5}	10.74%	.503%	83.53	9.31
24. Latvia	-8.333	25.0×10^{-5}	1.0×10^{-5}	7.63%	1.603%	75.45	13.10
25. Netherlands	-11.414	20.1×10^{-5}	19.0×10^{-5}	10.91%	.465%	84.31	9.16
26. Norway	-11.232	21.3×10^{-5}	20.0×10^{-5}	10.66%	.486%	84.36	9.38
27. New Zealand	-10.954	21.7×10^{-5}	20.0×10^{-5}	10.21%	.501%	84.92	9.79
28. Poland	-8.996	13.4×10^{-5}	1.0×10^{-5}	8.25%	1.160%	78.78	12.12
29. Portugal	-10.003	24.5×10^{-5}	20.0×10^{-5}	9.21%	.735%	82.75	10.86
30. Russia	-7.497	75.5×10^{-5}	20.0×10^{-5}	6.70%	2.236%	71.51	14.92
31. Slovakia	-9.413	9.2×10^{-5}	1.0×10^{-5}	8.92%	1.104%	78.43	11.21
32. Slovenia	-10.299	4.4×10^{-5}	1.0×10^{-5}	9.71%	.705%	82.03	10.30
33. Sweden	-11.565	18.9×10^{-5}	18.0×10^{-5}	11.05%	.431%	84.74	9.05
34. Taiwan	-9.230	29.8×10^{-5}	20.0×10^{-5}	8.18%	.902%	82.23	12.22
35. Ukraine	-7.939	55.6×10^{-5}	20.0×10^{-5}	7.29%	1.980%	73.02	13.73
36. UK	-10.623	22.4×10^{-5}	20.0×10^{-5}	9.84%	.567%	84.37	10.16
37. USA	-9.746	25.9×10^{-5}	20.0×10^{-5}	8.81%	.763%	83.07	11.35
Average:	**-10.099**	**19.9**$\times 10^{-5}$	**11.1** $\times 10^{-5}$	**9.41%**	**.848%**	**81.64**	**10.81**

Source: Human Mortality Database, Period 2011

Table # 1b

Gompertz-Makeham Parameters Around the World: FEMALE

Country	$\ln h[i]$	$\lambda_0[i]$	*Makeham:* λ	$g[i]$	$\lambda_{55}[i]$	m	b
1. Australia	-11.855	20.7×10^{-5}	20.0×10^{-5}	10.76%	.284%	89.44	9.29
2. Austria	-11.727	3.8×10^{-5}	3.0×10^{-5}	10.79%	.308%	88.05	9.27
3. Belgium	-11.366	12.2×10^{-5}	11.0×10^{-5}	10.36%	.356%	87.84	9.65
4. Belarus	-10.131	24.0×10^{-5}	20.0×10^{-5}	9.44%	.735%	82.34	10.60
5. Canada	-11.580	17.9×10^{-5}	17.0×10^{-5}	10.49%	.316%	88.94	9.54
6. Croatia	-11.608	1.9×10^{-5}	1.0×10^{-5}	11.10%	.409%	84.74	9.01
7. Switzerland	-12.272	15.5×10^{-5}	15.0×10^{-5}	11.29%	.248%	89.39	8.86
8. Czech	-11.726	16.8×10^{-5}	16.0×10^{-5}	11.16%	.390%	85.43	8.96
9. Germany	-11.771	15.8×10^{-5}	15.0×10^{-5}	10.92%	.328%	87.52	9.16
10. Denmark	-11.474	2.0×10^{-5}	1.0×10^{-5}	10.67%	.369%	86.56	9.37
11. Spain	-12.222	20.5×10^{-5}	20.0×10^{-5}	11.15%	.247%	89.95	8.97
12. Estonia	-10.867	10.9×10^{-5}	9.0×10^{-5}	9.94%	.460%	86.12	10.06
13. Finland	-11.644	8.9×10^{-5}	8.0×10^{-5}	10.65%	.316%	88.28	9.39
14. France	-11.402	21.1×10^{-5}	20.0×10^{-5}	10.07%	.304%	90.44	9.93
15. Greece	-12.252	20.5×10^{-5}	20.0×10^{-5}	11.44%	.278%	88.15	8.74
16. Hungary	-10.593	3.5×10^{-5}	1.0×10^{-5}	9.92%	.589%	83.49	10.08
17. Ireland	-11.561	11.0×10^{-5}	10.0×10^{-5}	10.63%	.340%	87.66	9.41
18. Iceland	-11.984	1.6×10^{-5}	1.0×10^{-5}	11.07%	.277%	88.35	9.03
19. Italy	-12.253	14.5×10^{-5}	14.0×10^{-5}	11.30%	.252%	89.15	8.85
20. Japan	-11.630	20.9×10^{-5}	20.0×10^{-5}	10.21%	.264%	91.58	9.80
21. Korea	-12.145	20.5×10^{-5}	20.0×10^{-5}	11.16%	.266%	89.20	8.96
22. Lithuania	-10.360	23.2×10^{-5}	20.0×10^{-5}	9.46%	.597%	84.55	10.57
23. Luxemburg	-11.645	1.9×10^{-5}	1.0×10^{-5}	10.71%	.317%	87.90	9.34
24. Latvia	-10.405	23.0×10^{-5}	20.0×10^{-5}	9.59%	.612%	84.03	10.42
25. Netherlands	-11.544	16.0×10^{-5}	15.0×10^{-5}	10.61%	.347%	87.63	9.42
26. Norway	-11.762	2.8×10^{-5}	2.0×10^{-5}	10.83%	.304%	88.05	9.23
27. New Zealand	-11.585	20.9×10^{-5}	20.0×10^{-5}	10.64%	.343%	87.83	9.40
28. Poland	-11.098	12.5×10^{-5}	11.0×10^{-5}	10.29%	.446%	85.73	9.72
29. Portugal	-11.903	20.7×10^{-5}	20.0×10^{-5}	10.97%	.303%	88.34	9.11
30. Russia	-9.749	25.8×10^{-5}	20.0×10^{-5}	8.96%	.824%	81.91	11.17
31. Slovakia	-11.271	4.3×10^{-5}	3.0×10^{-5}	10.70%	.462%	84.43	9.34
32. Slovenia	-11.530	2.0×10^{-5}	1.0×10^{-5}	10.64%	.343%	87.32	9.40
33. Sweden	-12.116	8.5×10^{-5}	8.0×10^{-5}	11.27%	.277%	88.16	8.88
34. Taiwan	-11.416	21.1×10^{-5}	20.0×10^{-5}	10.44%	.364%	87.69	9.58
35. Ukraine	-10.163	23.9×10^{-5}	20.0×10^{-5}	9.56%	.760%	81.77	10.46
36. UK	-11.476	21.0×10^{-5}	20.0×10^{-5}	10.50%	.354%	87.83	9.52
37. USA	-10.593	22.5×10^{-5}	20.0×10^{-5}	9.45%	.475%	87.09	10.58
Average:	**-11.424**	**14.5**$\times 10^{-5}$	**13.1** $\times 10^{-5}$	**10.52%**	**.391%**	**87.10**	**9.54**

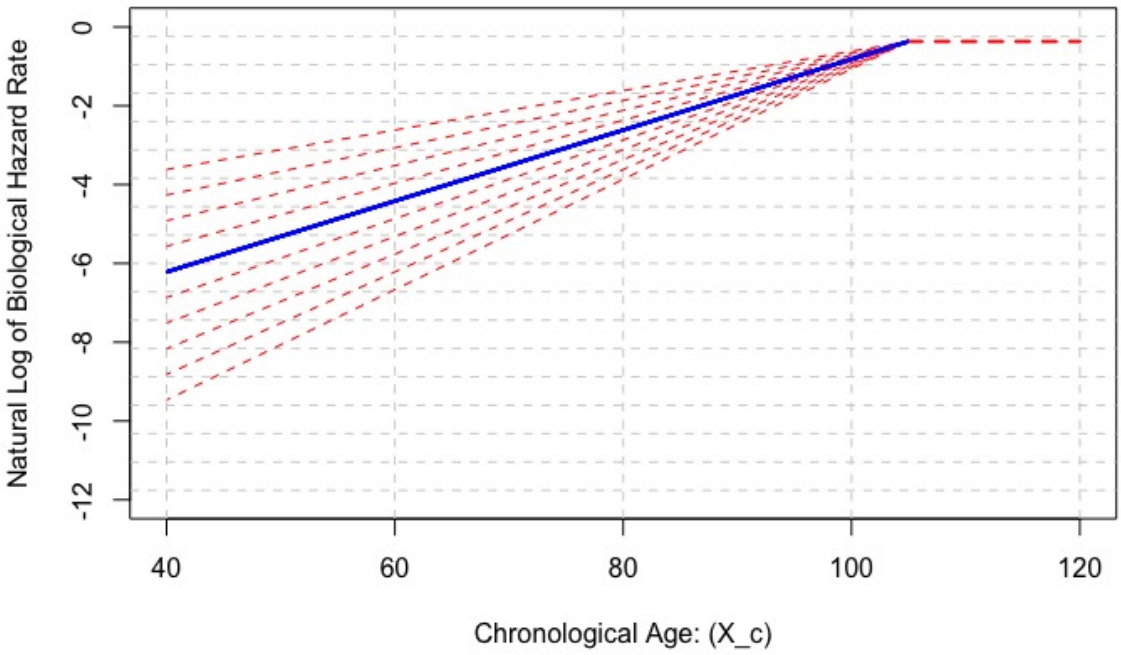
Source: Human Mortality Database, Period 2011

Table #2:						
CLaM Regression Line Around the World						
Variable	**MALE**			**FEMALE**		
	Coeff.	Std.Er	t-val.	Coeff.	Std.Er	t-val.
Intercept (L)	-1.09	0.185	-5.895	-0.9151	0.5416	-1.69
Slope: ($-x^*$)	**-95.70**	1.95	-49.07	**-99.9**	5.1407	-19.44
Adj. R^2	98.53%			91.28%		
Range: $g[i]$	(6.70%, 11.05%)			(8.96%, 11.44%)		
Average: $g[i]$	G = 9.41%			G = 10.52%		
Plateau (+/-): λ^*	(0.2322, 0.4867)			(0.1355, 1.1830)		
Countries	$N = 37$			$N = 37$		

Note: These are the results from regressing the (male and female) Gompertz-Makeham (log) mortality intercepts $\ln h[i]$ on the mortality growth rates $g[i]$, from the Human Mortality Database (HMD) for *period* mortality in 2011. This is the second phase regression.

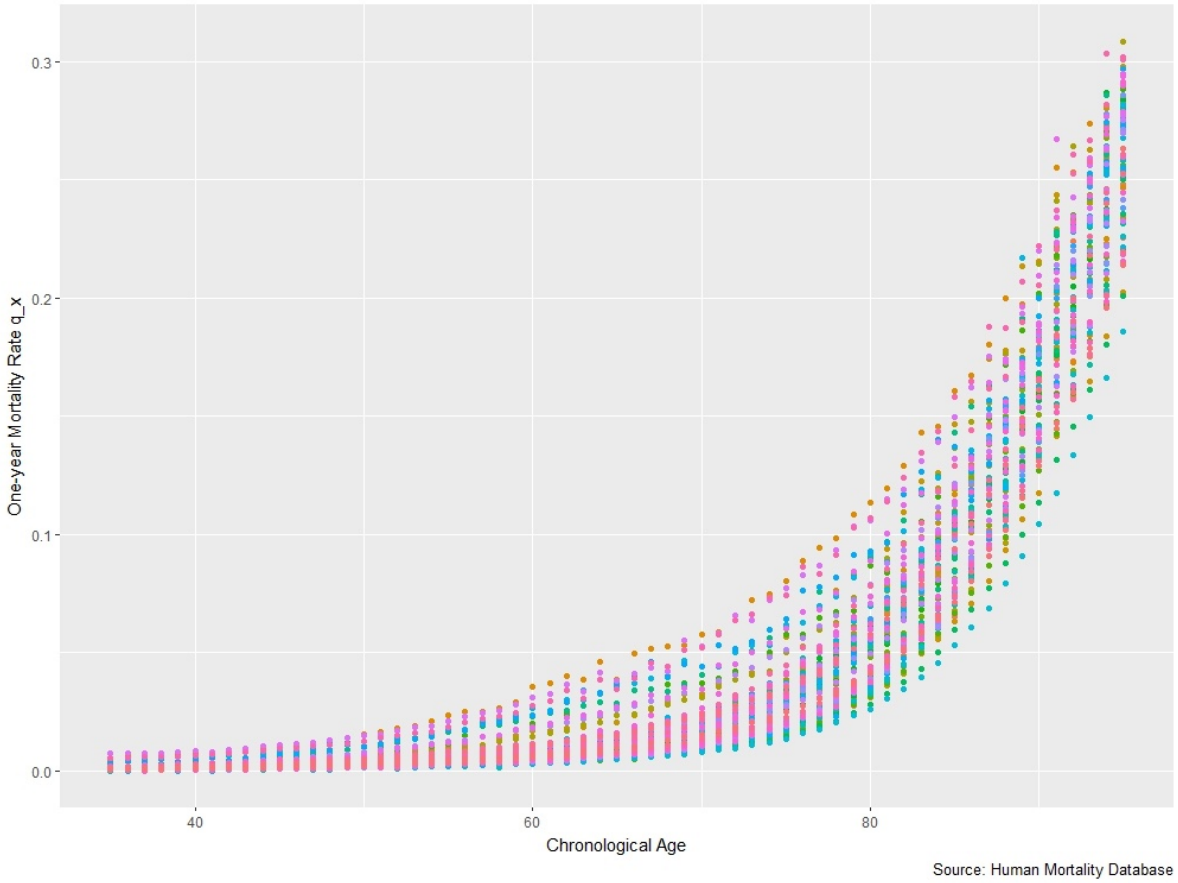
Table # 3						
Mortality-Adjusted Biological Ages (ξ) Around the World						
	MALE			**FEMALE**		
Country	$x = 55$	$x = 70$	$x = 85$	$x = 55$	$x = 70$	$x = 85$
1. Australia	52.07	68.15	84.23	53.95	69.30	84.65
2. Austria	52.85	68.64	84.44	53.84	69.23	84.61
3. Belgium	52.86	68.65	84.44	55.68	70.45	85.23
4. Belarus	65.93	76.90	87.87	59.61	73.07	86.53
5. Canada	51.98	68.10	84.21	55.14	70.09	85.05
6. Croatia	54.38	69.61	84.84	52.49	68.33	84.17
7. Switzerland	49.12	66.29	83.45	51.71	67.81	83.91
8. Czech	54.29	69.55	84.81	52.26	68.17	84.09
9. Germany	52.93	68.69	84.46	53.28	68.86	84.43
10. Denmark	52.77	68.59	84.41	54.34	69.56	84.78
11. Spain	53.06	68.78	84.49	52.30	68.20	84.10
12. Estonia	60.10	73.22	86.34	57.48	71.65	85.82
13. Finland	54.37	69.60	84.83	54.42	69.61	84.81
14. France	56.29	70.81	85.34	56.91	71.27	85.63
15. Greece	55.22	70.14	85.06	51.06	67.38	83.69
16. Hungary	58.35	72.12	85.88	57.55	71.70	85.85
17. Ireland	51.35	67.70	84.04	54.51	69.68	84.84
18. Iceland	49.08	66.26	83.44	52.63	68.42	84.21
19. Italy	49.21	66.34	83.48	51.67	67.78	83.89
20. Japan	52.23	68.25	84.27	56.32	70.88	85.44
21. Korea	53.32	68.94	84.56	52.27	68.18	84.09
22. Lithuania	64.94	76.28	87.61	59.50	72.99	86.49
23. Luxemburg	49.27	66.38	83.49	54.20	69.46	84.73
24. Latvia	62.69	74.86	87.02	58.94	72.63	86.31
25. Netherlands	48.52	65.91	83.30	54.59	69.72	84.86
26. Norway	49.61	66.59	83.58	53.65	69.10	84.55
27. New Zealand	51.54	67.82	84.09	54.48	69.66	84.83
28. Poland	60.02	73.17	86.32	55.96	70.64	85.32
29. Portugal	55.90	70.57	85.24	53.06	68.71	84.36
30. Russia	66.71	77.40	88.08	61.67	74.44	87.21
31. Slovakia	57.13	71.35	85.56	54.21	69.47	84.74
32. Slovenia	53.70	69.18	84.66	54.49	69.66	84.83
33. Sweden	47.93	65.54	83.14	51.80	67.87	83.94
34. Taiwan	60.33	73.37	86.40	55.32	70.21	85.11
35. Ukraine	64.20	75.81	87.42	59.10	72.73	86.36
36. UK	53.14	68.82	84.51	55.08	70.05	85.03
37. USA	57.62	71.65	85.69	59.54	73.02	86.51
Average:	**55.0**	**70.0**	**85.0**	**55.0**	**70.0**	**85.0**
Source: Human Mortality Database, Period 2011						

Figure 1: Visualizing Gompertz and the Compensation Law of Mortality



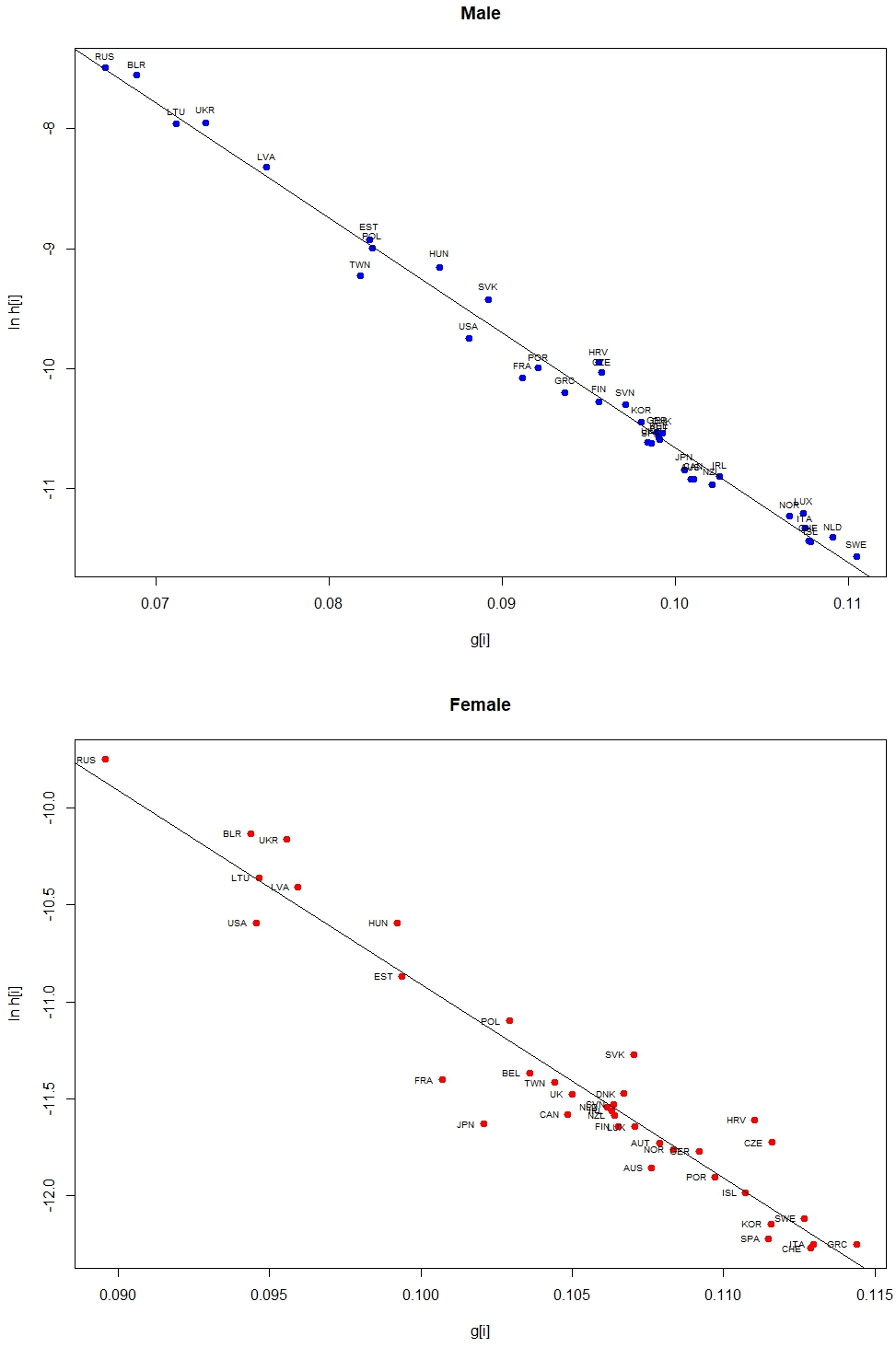
Note: As per Gavilov and Gavrilova (1991), the *compensation law of mortality* in its strong form implies that (log) mortality rates increase linearly and then converge to a constant mortality plateau. This leads to a linear and negative relationship between the intercept: $\ln h[i]$, and the slope: $g[i]$, in a Gompertz regression of log mortality rate on chronological age. The thick (blue) line in the center is based on the average $g[i]$, and used to convert chronological age into mortality-adjusted biological age.

Figure 2: Raw Mortality Rates Around the World



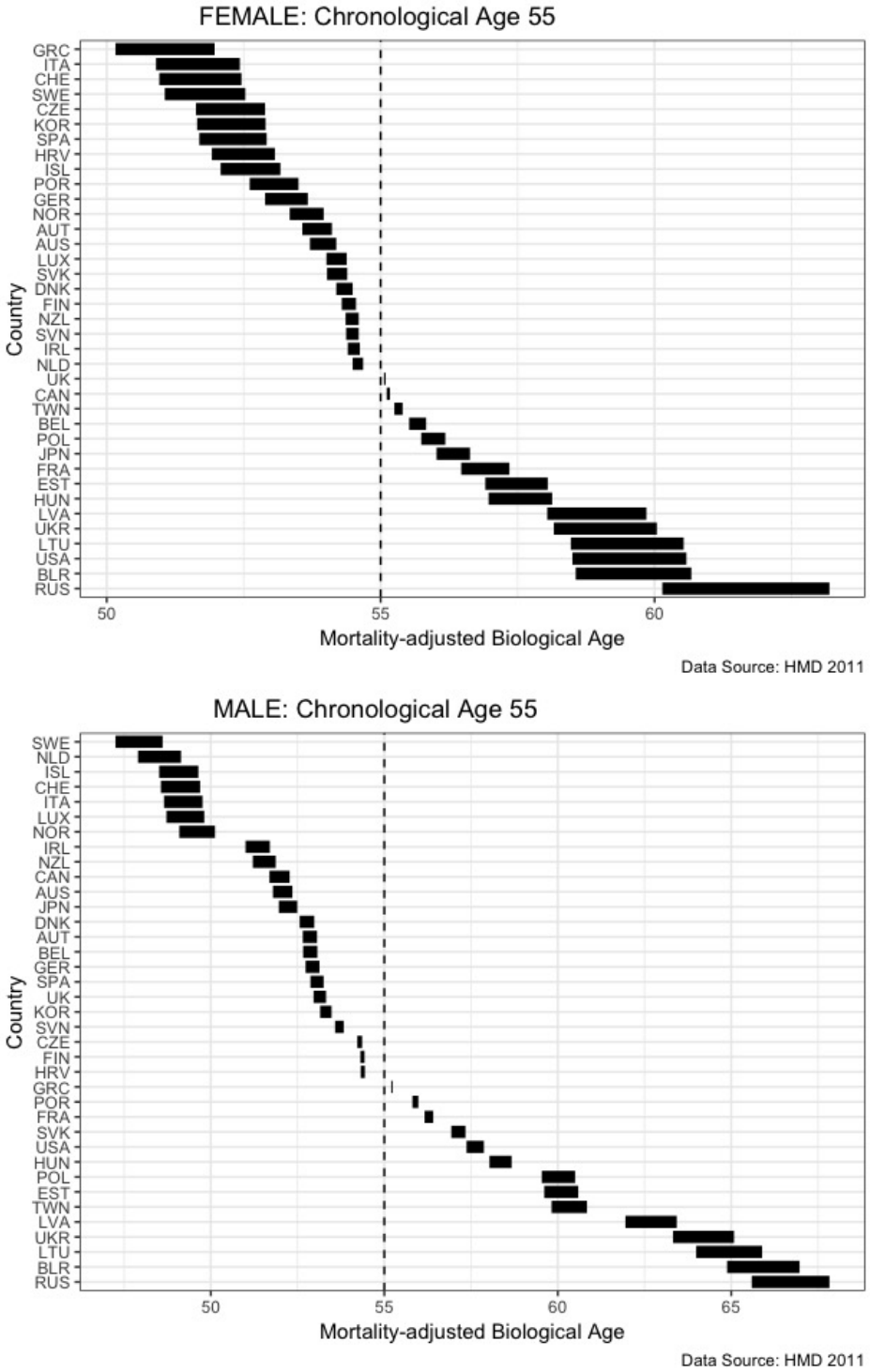
Note: Raw q_x data used in the first regression. Mortality rates in 37 countries (male and female) from the Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de (data downloaded on 15 December 2018).

Figure 3: Evidence for the Compensation Law of Mortality Around the World



Note: There is a clear and evident (negative) relationship between the mortality growth rate $g[i]$ in a given country, and the (log) of initial mortality rate $\ln h[i]$. The resulting slope and intercept are displayed in Table #2. See also the paper by Tarkov et al. (2017) for a detailed and recent discussion of the analytic relationship between g and $\ln h$, and in particular its connection to the so-called Strehler-Mildvan correlation.

Figure 4: Range of mortality-adjusted biological ages around the world at age $x = 55$.



Note: The lower value for mortality-adjusted biological age $\xi_{\texttt{low}}$ and upper value $\xi_{\texttt{high}}$ are based on equation (22) in which the point estimate of $x^* = 95.7$ for males and $x^* = 99$ for females, are replaced with plus or minus two standard errors, which are 1.95 years (male) and 5.14 years (female) respectively, as estimated in the second phase regression (Table #2.)