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

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Abstract

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This paper develops a statistical and methodological framework for inverting the Gompertz-Makeham (GM) law of mortality for heterogenous populations in a manner consistent with a compensation law of mortality (CLaM), to formally define a global mortality-adjusted (biological) age. It implements and calibrates this framework using rates from the Human Mortality Database (HMD) to illustrate its salience and applicability. Among other things, this paper demonstrates that when properly benchmarked, the global mortality-adjusted (biological) age of a 55-year-old Swedish male is 48, whereas a 55-year-old Russian male is closer in age to 67. The motivation for this (new) framework for presenting age and relative aging is that this metric could be used for pension and retirement policy. In a world of growing mortality heterogeneity and the need for salient longevity metrics beyond simple life expectancy, "biological age" might help capture the public's attention and induce them to take action, for example to work longer and retire later. Perhaps a mortality-adjusted (biological) age could even be used to determine pension eligibility.

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1 Introduction and Motivation

The legacy of Benjamin Gompertz has withstood the test of time and Gompertz (1825) has been cited with increasing frequency in the last few decades[1]. Despite its age and noted empirical limitations, his eponymous law of mortality – which will soon be celebrating a 200th anniversary – is still widely used in the fields of demographics, biology, actuarial science and even financial engineering. To his credit Benjamin Gompertz appears regularly in the pages of *Insurance: Mathematics and Economics*. In particular, the ability to represent important actuarial expressions in closed form via the Gamma function has made the Gompertz representation especially convenient and popular in the annuity literature. The defining characteristic of the Gompertz law of mortality, when expressed in continuous time, is the linear relationship between the log of natural mortality rates and (chronological) age x. The natural law is written or expressed via the following parameterization:

$$\texttt{Natural Mortality Rate} \;=\; \mu_x - \lambda \;=\; he^{gx} \;=\; (1/b)e^{(x-m)/b}.$$

In this expression, $\lambda \geq 0$, which is subtracted from the total hazard rate $\mu_x >> \lambda$, is a non-age-dependent accidental death (a.k.a. Makeham constant) rate, and $(h > 0, g > 0)$ or alternatively $(m > 0, b > 0)$ determine the slope and intercept of $\ln[\mu_x - \lambda]$. Using the more common (m, b) formulation, the parameter m represents the modal value of the remaining lifetime random variable T_x, and b represents a dispersion coefficient. Either way, the implicit linearity assumption is empirically valid over adult ages across most countries around the world. However, it does not fit or work very well at young $(x < 30)$ ages, and the upper bound is subject to some debate in the bio-demographic and medical literature. See Gavrilov and Gavrilova (2014).

At the risk of jumping too far ahead, Table #1 [**placed here**] displays the best-fitting Gompertz (and Makeham) parameters for the 37 countries listed and available in the Human Mortality Database (HMD) at the time this analysis was conducted. Both sets of (h, g) and (m, b) are displayed, and are in line with values used by researchers in a variety of published papers in the actuarial literature, which will be discussed in the formal literature review.

[1] According to Google Scholar, no fewer 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).

It is obvious and quite evident (from Table #1) that although the Gompertz law itself fits or works in all the listed countries, the best-fitting parameters are country dependent. Some countries experience much higher mortality (for example $m = 71.7$ years for males in Belarus), while other countries experience much lower mortality (for example $m = 86.4$ for males in Australia.)

What is less known to scholars who may not specialize in this area is that upon closer examination of the country-by-country parameter estimates, there is a negative relationship between the estimated (log) initial natural mortality rate $\ln[h]$, and the estimated mortality growth rate g. Countries with a relatively low initial natural mortality rate tend to have a higher mortality growth rate and vice versa. The exact nature of this relationship – known as the compensation law of mortality – will be made precise later in the paper, but can be visualized in Figure #1 [**placed here.**] Countries with relatively low mortality rates are represented by the top lines, and countries with relatively higher mortality are represented by the bottom lines, all in log scale. One can then think of the (thick) middle line as representing a global average (log) mortality rate. In fact, the relative difference in natural mortality rates declines over (chronological) age, so that at some advanced age the differences between mortality in different populations is minimal, but at middle ages the difference in mortality rates – and perhaps true age – differs substantially across countries. This gets us to the notion of a *biological* age which is distinct from *chronological* age.

1.1 A Mortality-adjusted Age

Researchers in a variety of medical fields are working on uncovering bio-markers of aging which measure an individual's *true* physiological age, also known as *biological age*. This sort of information is used to generate more accurate forward-looking mortality rate projections as well as distributions of future lifetimes; both of which are obviously better than a simple period life expectancy. The age adjustment processes, which are similar to actuarial setbacks, are a refined form of the underwriting process employed by life insurance companies for centuries. What has received less attention in the insurance literature is how to map estimated mortality rates into a consistent Mortality-adjusted *biological age*. This paper develops a statistical and methodological framework for inverting the Gompertz-Makeham (GM) law of mortality for heterogenous populations in a manner consistent with a compensation law of mortality (CLaM) to formally define a global mortality-adjusted (biological) age.

The underlying two-stage process will soon be explained – but the end result is a simple equation which maps an individual's (chronological) age x, into a global mortality-adjusted (biological) age ξ. The value might be higher than x (indicating poor relative health) or lower than x (indicating better relative health).

So, why bother doing this? On a behavioral level, this information might represent an alternative way of explaining longevity metrics to a wider public who struggle with probabilistic concepts.[2] Indeed, informing a healthy 70-year-old that they face a 20% chance of living to age 90 – and should therefore plan for this *longevity risk* – might not be as impactful or salient as informing them they are really 55, biologically. This paper makes no claim about the efficacy of presenting longevity risk information in (what this author believes is) a more salient manner and leaves that task to other (future) researchers. Rather, this paper is focused and concerned with methodology. Namely: *Given a large set of local mortality rates, how does one compute a global mortality-adjusted (biological) age?* Now, to modern practicing actuaries the underlying Gompertz model might appear as an archaic remnant of an era prior to computers and spreadsheets. However, the Gompertz-Makeham law allows for analytic tractability that greatly benefits the inversion process which is at the heart of this paper.[3]

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 in the medical field, resulting in two different philosophies or views. Section (#3) provides an overview of the statistical and methodological framework proposed in this paper. Section (#4) gets into the details and the role of the *compensation law of mortality*, introduced by Gavrilov and Gavrilova (1991). Readers interested in the numerical values of global mortality-adjusted (biological) ages around the world can skip ahead 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 and pension policy.

2 Biological Age: Defined and Explained

Generally speaking, there are two (very) different approaches for how to compute and measure (mortality-adjusted) 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. Although researchers themselves don't use these terms, this papers labels the two approaches the "living" (i.e. medical) methodology and the "dying" (i.e. actuarial) methodology. Stated quite 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.

[2]See the work by Payne, et al. (2013), and their various references for more evidence on the difficulty consumers face with basic probabilistic information, especially as it relates to retirement income planning.

[3]The inversion process would be more difficult with general mortality laws – see Richards (2019) for an approach to modeling compensation laws with splines – but remains a possibility for future research.

2.1 The Living Approach

In the medical arena, 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, to extract various physiological and molecular (DNA, RNA, etc.) variables. These measurements, which could number in the hundreds, might range from red blood cell count, hemoglobin concentration, and total cholesterol, to items such as fasting blood sugar levels, urine specific gravity, triglycerides, or the average telomere length (or ATL), which for a while was the leading biomarker for aging, and is associated with the work of Blackburn with Epel (2017).

These physiological and molecular variables might then be augmented by physical variables (i.e., more easily measured, not requiring a laboratory) such as hand grip strength, visual perception, or even the number of missing teeth. Some researchers go so far as to augment their dataset with social variables, such as number of friends on Facebook, or a binary variable measuring whether they like to garden. The theory here is that anything remotely associated with the characteristics of older people can be added as a data point for measuring true age: See Ries and Pothig (1984), Dubina, Mints and Zhuk (1984), or more recently Jylhava, Pederson and Hagg (2017.) 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. The most important number, however, which one can visually imagine as being stored at the very beginning of this long vector, is the individual's chronological age. Here we 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.

The (usually medical) researcher would then generate 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., Facebook friends) and the multivariate regression is estimated again (and again) until the process converges on a small set of variables that relate (i.e., predict) the dependent variable, which is chronological age. The best-fitting regression equation becomes the formula for biological age, while the individual errors in the regression are the gaps between a person's chronological age and their biological age.

The statistically significant coefficients in this regression are declared as relevant biomarkers of aging – and the sign of the coefficients (a.k.a. factors), would determine whether scoring higher in those elements effectively makes one younger or older. Note this is **not** the approach taken in the current paper, but is what many (if not most) medical researchers mean by the term *biological age.* It's worth noting that the first attempt at measuring biological age was made by the mathematician Hugo Steinhaus in 1932, based on deteriorating eyesight.

2.2 Methodological Concerns

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

In some clinical studies, e.g. Jylhava et al. (2017), researchers track large groups of people over time to examine if the older ones 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 who are most similar 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 and data mining, as well as concerns about linearity assumptions, etc[4].

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. This process begins by collecting data on mortality rates as a function of chronological age plus other characteristics or elements. For example, these might include the number of cigarettes the now-deceased smoked before they died, or their body mass index before they died or their triglyceride level before they died. The "dying" approach for measuring biological age also invokes a regression process, but the dependent variable, denoted by the familiar q_x, is a mortality rate, not a chronological age.

[4]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.

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 mortality might be estimated to include a variable which is one minus the ratio of the average length of telomeres in the body (in units of nucleotides) to the number 10,000 (for example) in the year prior to death. If the average length of ones telomeres is 9,900 nucleotides, then the 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%. Needless to say, given the censored nature of the data (live people), there are natural biases that must be corrected, and so forth. Moreover, 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 the paper briefly touches upon this in 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 these factors are segmented into groups to determine whether they impact mortality rates conditional on age.

In this paper, the only differentiating factor considered is nationality, using data from the human mortality database (HMD). Just as importantly, the computation of mortality-adjusted (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, this paper derives mortality-adjusted (biological) ages by inverting the GM expression, taking as input both global and local mortality rates and then solving for the implied age ξ.

3 Conceptual Model: From Mortality Rates to an Age.

Every adult life in *country* i, $i = 1, ..., N$, obeys a GM law. The total hazard rate is:

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

where $x \geq 0$ denotes (chronological) age, $g[i] \geq 0$, is the mortality growth rate, $h[i] \geq 0$ is the initial natural mortality rate, and $\lambda[i] \geq 0$ is the accidental (non-age-related) hazard rate, a.k.a. Makeham constant. As stated earlier, this implies that the log of the total hazard rate minus the accidental date rate: $\ln[\mu_x[i] - \lambda[i]]$, is a linear function of (chronological) age x, with intercept $\ln[h[i]]$ and slope $g[i]$. Each country i is described by a set: $(\lambda[i], h[i], g[i])$, which is unique[5].

[5]Since both: $h[i]$ and $\lambda[i]$ tend to be very small numbers, that is, on the order of 10^{-5}, most of the tables and figures will display the natural log values.

Moving on, let ξ denote a mortality-adjusted (biological) age, which is also assumed (i.e., forced) to satisfy a GM-like relationship for the continuous total hazard rate. That relationship is captured by the thick middle curve in Figure #1. Every person in country i, at the chronological age of x, is assumed to have another "age" denoted by ξ, which may (or may not) equal their chronological age x. The arrows in Figure #1 go from chronological age to what this paper defines as the mortality-adjusted (biological) age. Formally, the ξ will satisfy:

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

where $\Lambda \geq 0$, $H > 0$, and $G \geq 0$ represent *global* GM parameters. The exact procedure by which Λ, H, G are estimated from specific country values will be described later on, but for now one can think of the global values as a *weighted average* of the vector of local values. Again, see the middle line in Figure #1 for intuition. The averaging process, however, is **not** linear **if** the procedure is to be consistent with the *compensation law of mortality*. 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 global mortality-adjusted (biological) age $\xi := \xi(x, i)$ in country i is obtained by equating hazard rates at (chronological) x and solving for the implied ξ. Formally:

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

Inverting the GM equation in terms of μ, will set and determine the global mortality-adjusted (biological) age ξ. Equating (1) and (2) and dispensing with the $[i]$ index on the local $\lambda[i], h[i], g[i]$ values, leads to:

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

For the above expression to make sense, one must further impose a restriction that $\lambda - \Lambda + he^{gx} > 0$, which as long as x is large enough (remember: adult ages) shouldn't pose a problem. In some sense, equation (4) is the main equation in the paper and the remainder is implementation. Equation (4) maps a (chronological) x, the country Gompertz-Makeham parameters (λ, h, g) and the global Gompertz-Makeham parameters (Λ, H, G) into a global mortality-adjusted (biological) age.

To obtain some intuition for equation (4), assume that in a particular country i, and within the Gompertzian age range, the Makeham constant $\lambda[i] = \Lambda$, which is the global Makeham parameter. (In general we will not make that assumption.) In that case, equation (4) can be expressed as:

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

Intuitively, one can see from equation (5) how the global mortality-adjusted (biological) age ξ, collapses to chronological age x when the country-specific initial natural mortality rate is equal to the global average value and the local mortality growth rate is equal to the global average value. Equation (5) also indicates that when $g = G$ and the mortality growth rates are equal, but $h \neq H$, the global mortality-adjusted (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 actuarial practice to model sub-standard or healthy lives, but is technically inconsistent with the *compensation law of mortality*.

Before moving on to estimation and implementation, here is a simple numerical value from equation (5) to further develop the intuition. Assume that in Mauritius the value of $h[i]$, which is the natural mortality rate at age zero, 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. Recall once again that high values of h are associated with lower values of g, and vice versa. So, in the same spirit, assume for this example that the global average values are: $H = 1 \times 10^{-5}$ and $G = 0.09$, both of which are completely fictitious at this point. Under these values, a chronological $x = 65$ year-old in Mauritius, according to equation (5), has a global mortality-adjusted (biological) age of: $\xi = 73.4$. One would thus conclude that they are older than their chronological age.

3.1 In sum: Two Regressions and an Inversion

1. The process starts with vectors of country-specific decrements $q_x[i]$, obtained from period mortality tables. They are converted to continuous total hazard rates $\mu_x[i]$ in a manner consistent with the Gompertz-Makeham law, since $\mu_x \neq q_x$.

2. Standard linear regression techniques are used to estimate the local Gompertz-Makeham parameters $(\lambda[i], h[i], g[i])$ for each one of the i countries.

3. With the N parameters, a second phase regression is implemented to estimate the global (i.e., average) values of: Λ, H, G, in a manner consistent with the compensation law.

4. Global mortality-adjusted (biological) ages are computed using equation (4) at various ages using the country-specific: $(\lambda[i], h[i], g[i])$, and particular values of Λ, H, G.

Before moving on to explain each one of the steps in detail, here is a brief recap of the mortality terminology. The model is formulated in continuous time, where the total hazard rate $\mu_x[i]$, at chronological age x, in country i, is the sum of the accidental (Makeham death) rate $\lambda[i]$ and the natural age-dependent mortality (Gompertz) rate $h[i]e^{g[i]x}$.

The country-specific parameter $h[i]$ is the initial (age zero) natural mortality rate, and $g[i]$ is the country-specific mortality growth rate. To be very clear, despite the term *initial*, $h[i]$ is not the infant mortality rate in country i. (The first 30 years of life don't obey the Gompertz law.) Rather, $h[i]$ is a *hypothetical* value that assumes the GM regime extends to age zero. In Figure #1 it is the point at which the various country-lines hit the y-axis, if the x-axis is extended leftwards to zero. On the other side, the natural (Gompertz) portion plateaus at chronological age x^*, which is a fixed global parameter, at a value of λ.

4 The Detailed Procedure

4.1 Step One: Estimating Country GM Parameters

Each country is identified and summarized by **3** local plus **2** global = **5** total parameters. They are: $\{h[i], \lambda[i], g[i], x^*, \lambda^*\}$, where $i = 1, ..., N$, is the number of countries. Again, $h[i]$, represents the initial natural mortality rate for country i, and the second parameter is the accidental (Makeham) death rate term $\lambda[i]$. Indeed, some countries are estimated to have (very) high accidental death rates (e.g., males in Russia, in Table #1a) and other countries have negligible (estimated) accidental death rate (e.g., males in Israel, in Table #1b). The third term which differentiates one country from another is $g[i]$, which is the corresponding mortality growth rate (MGR) during the *Gompertzian* range of life $x < x^*$. For those more familiar with the (m, b) representation of the GM law, it's the country-specific value of the inverse of the dispersion parameter $g = 1/b$.

The fourth parameter is global (i.e., not country-specific) and is denoted by x^*. This is the critical age at which the *Gompertzian* regime ends, and is also known as the species-specific lifespan, per Gavrilov and Gavrilova (1991). In Figure #1, it is pictured as the chronological age (approximately $x = 105$) at which the disparate (log) rates intersect and plateau to a constant. This critical parameter will also be estimated from the collection of country rates.

Finally, the (log) natural rates plateau at a fifth and final estimated parameter value: λ^*. If and when individuals (ever) reach that very advanced rate, they face a constant hazard rate (which is country dependent) and therefore an exponentially distributed remaining lifetime from that point onward. Technically speaking, it's very important to note that although $\ln[\mu_x[i] - \lambda[i]]$, which in Figure #1 is the y-axis, plateaus at a value of λ^*, the individual country-specific total hazard rates will equal: $\lambda[i] + \lambda^*$, after age x^*.

The total hazard rate μ_x, will obey the following relationship:

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

Empirically, the plateau mortality rate: $\lambda^* >> \lambda[i]$ and the corresponding age x^*, is a global constant, as per the strict version of the *compensation law of mortality.* Nevertheless, it's worth emphasizing that equation (6) is quite general, as it's conceivable $x^* \to \infty$, and there is no (finite) mortality plateau. Rearranging, the GM model can be expressed as:

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

which is the standard linear representation of (log) total hazard minus accidental death rates for all ages within the GM regime. A deliberate choice is made to use the capital Q_x, on top of the brace, and not the natural log of the one-year death rate $\ln[q_x]$, because q_x and μ_x are obviously not the same quantity. Although the two numbers are close for small values of q_x, in continuous time the total hazard rates can (obviously) exceed the value of one[6]. For greater accuracy, recall that q_x, at any given chronological age x, is related to the total hazard rate μ_x, via:

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

Now, when $\mu_x = \lambda$, is constant (i.e., $h = 0$), the survival rate to any time t is $e^{-\lambda t}$, and then $q_x = 1 - e^{-\lambda}$, for any one year. In this (simplistic, clearly non-Gompertz) case, the μ_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: $-\ln[1 - q_x] > \lambda \geq 0$, so one can subtract the accidental death (Makeham) rate λ from both sides of the above expression, take logs (again) and obtain a linear relationship between the (transformed value of the) one-year decrement q_x and chronological age x. The relationship can be written explicitly for each of the groups as:

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

[6]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), simply use $\ln[q_x]$ on the left-hand side of the above regression, and without subtracting a Makeham term.

The new constants (K_0, K_1) are defined for convenience and suggest the proper regression methodology for calibrating GM parameter values of λ, h, g from one-year decrement rates q_x. To be very clear though, $\ln[\ln[(1-q)^{-1}]] \approx \ln[q]$ for small values of q, so the approximation (that some researchers employ, in treating q_x and μ_x as the same) is justified. But, one does introduce errors when $\lambda[i] \neq 0$ and/or when $q_x[i] >> 0$. At the very least, if one insists on using equation (7) instead of equation (10) to estimate Gompertz-Makeham parameters, it's more accurate to use $\ln[q] + q/2$ as the dependent variable to match the first two terms of the Taylor series expansion[7]. For now, this leads to the GM regression equation:

$$z_{i,j} \;=\; K_0 \;+\; K_1 x_j \;+\; \epsilon_{i,j}, \tag{11}$$

where x_j is a vector of ages, for example $x_1 = 35, x_2 = 36, x_3 = 37$, etc., and the $z_{i,j}$ are computed from the one-year mortality decrements q_x in country i. Equation (11) embeds an implicit assumption that the error terms $\epsilon_{i,j}$, are independent across chronological age x and across countries i. See Figure #2 [**placed here**] for a graphic visualization of the full data used in this phase.

Note that to properly estimate the accidental (Makeham) death rate $\lambda[i]$, in each country i, an iterative procedure was used. Initially, $\lambda[i]$ is assumed to be zero and a basic canonical regression is estimated, per equation (10). The value of $\lambda[i]$ is gradually increased by units of 10^{-5}, and iterated (i.e., searched for) until the GM regression error is minimized. The upper bound (in the search for) $\lambda[i]$, is $\mu[i]$, since the accidental death rate can't be higher than the total death rate over the GM region.

Minutiae and details aside, once the unique and group-specific values for $\lambda[i]$ are located, the associated regression formulated in equation (11) leads to the best-fitting intercept and slope parameters $\tilde{K}_0$ and $\tilde{K}_1$. More importantly, based on equation (10), the unbiased estimates for the GM parameters for each one of the groups, is:

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

These are (i.) the natural mortality growth rate, (ii.) the log initial natural mortality rate, and (iii.) the natural initial mortality rate at age zero, for each i. This process is referred to as the first phase 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.

[7]Please refer to the recent work by Tai and Noymer (2018) for a full and proper discussion of the many and diverse methods that can be used to estimate Gompertz parameters from one-year decrements, and in particular the approach that minimizes root mean square (RMS) errors for life expectancy estimates.

As noted in the introduction to the paper, researchers in actuarial finance (and in particular the annuity literature) might be more accustomed to the probabilistic formulation of the Gompertz-Makeham law in terms of the modal value m and dispersion coefficient b of the remaining lifetime random variable. Using that formulation, the total hazard rate μ_x is expressed as: $\lambda + (1/b)e^{(x-m)/b}$. So, the conversion from estimates of (h, g) to estimates of (m, b) would be via $b = 1/g$ and $m = (\ln[g] - \ln[h])/g$. Either way, both the (h, g) and (m, b) parameter estimates are displayed in Tables #1a, #1b, for all available countries in the Human Mortality Database.

Now, in theory, one could stop (the estimation procedure) here and compute arithmetic or geometric (or some other harmonic) average global value for Λ, H, G and then use those with the individual values of $\lambda[i], h[i], g[i]$ to compute mortality-adjusted (biological) ages via equation (4). However, this would very critically ignore the fact that (i.) the sum of Gompertz variates isn't Gompertz, and more importantly would be inconsistent with (ii.) the *compensation law of mortality* which forces a strict relationship between h and g. The expression for global mortality-adjusted (biological) ages described in the next section will 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. 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: $g_{\texttt{min}} \leq g \leq g_{\texttt{max}}$. In some sense, while the classical Gompertz law allows for two degrees of freedom – the slope and intercept of the log hazard line – the CLaM stipulates that once the slope is known, the intercept is pre-determined. There is only one (real) degree of freedom in mortality, a fact which has recently been pointed out and leveraged by Richards (2019) in the context of fitting the Gompertz law using Hermite splines. To be clear though, 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 the natural 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:

$$L \;:=\; \ln(\lambda^*) \;=\; \ln \vec{h}(g) + gx^*. \tag{13}$$

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

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

In what follows, $\ln \vec{h}(g)$ is referred to as the CLaM line mapping a specific initial mortality growth rate g to a corresponding log natural mortality rate $\ln \vec{h}(g)$. Exponentiating equation (14), the initial natural mortality rate: $\vec{h}(g)$ can be expressed as: $\vec{h}(g) = e^{L - x^* g}$, which at $g = 0$ recovers the mortality plateau: $\lambda^* = \vec{h}(0)$. Either way, it follows that under the strong *compensation law of mortality*, one can rewrite the total hazard rate μ_x, from equation (6) as:

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

which could also (if needed) be expressed[8] in terms of (m, b). The new function $\vec{\mu}_x(g)[i]$, which at first might seem cumbersome and unnecessary is meant to remind readers of a number of implicit assumptions from this point onward. First, under a strict CLaM the initial natural mortality rate is driven and dictated (only) by the mortality growth rate: that is the one and only degree of mortality freedom per country, other than the accidental death (Makeham-constant) rate $\lambda[i]$. Second, although the natural mortality rate plateaus at a global value of λ^*, the country-specific *total* plateau must also account for the accidental rate $\lambda[i]$, which is why both are added together in the lower branch of equation (15). Procedurally, and from this point onward, only the N country-specific values of $\{\ln h[i], \lambda, g[i]\}$, are needed. These are used to estimate the (intercept) L, and (slope) x^* via a second phase regression. As per equation (14), the relationship is:

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

where the error terms ϵ_j, are distinct from the error terms in equation (10).

[8]Recall that under the (m, b) formulation of the Gompertz-Makeham law, $h = (1/b)e^{-m/b}$ and $g = 1/b$, when the total hazard rate is modeled as: $\mu_x = he^{gx}$. So, a linear relationship between $\ln[h] = L - x^* g$, per the compensation law of mortality, also forces a relationship between m and b, although obviously not linear. Fixing b, together with the global parameters (L, x^*), induces a value for m. Technically, after substitution and isolating: $m = x^* - b(L + \ln[b])$. So, if one assumes the easy to remember values: $x^* = 100$, and: $L = -1$, both of which are empirically reasonable per table (#2), then the modal value of the Gompertz distribution is forced to be: $m = 100 - b(\ln[b] - 1)$. Thus, for example, in a country (or population) where $b = 11$ then $m = 84.62$, but if $b = 9$ then $m = 89.2$. A greater b induces a lower value of m and vice versa. Finally, since the expected remaining lifetime at birth under a Gompertz model is: $E[T_0] = m - b\gamma$, where $\gamma \approx 0.577$, is Euler's constant, we obtain the rather intriguing expression $E[T_0] = x^* - b(L + \ln[b] + \gamma)$ under the compensation law. Estimate or pick the dispersion coefficient b (or the approximate standard deviation $\pi b/\sqrt{6}$ at birth) and the mean lifetime follows, assuming of course that (L, x^*) are globally determined.

The second phase regression simply can't be merged with the first phase regression used to estimate the original GM parameters in equation (10), because of the need to (i.) iteratively estimate and then (ii) subtract the country-specific accidental death rates $\lambda[i]$. Also, the first regression generates the: $\ln h[i], \lambda[i]$ and $g[i]$ values in Table #1a and #1b, which might be of independent use and interest to researchers who use this law for pricing annuities or modeling retirement strategies, as cited in footnote #1.

Moving on, Table #2 **[placed 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 of 85% (females) and 98% (males), providing support for a strong version CLaM for the $N = 37$ countries. Finally, the estimated $L = \ln(\lambda^*)$, reveals or locates the natural mortality rate once the plateau is reached. The slope $(-x^*)$ is the age at which it is achieved, a.k.a. the *species specific lifespan*. See Figure #3 **[placed here]** for a visual indication of the strong negative relationship between the two variables on a country-by-country basis.

4.3 Stage Three: Baseline Population Rates

We now have a set of diverse GM parameters as well as (one, global pair) (λ^*, x^*). Back to the computation of mortality-adjusted (biological) age: the next step is to compute population averages Λ, H, G values, to then invert the GM equation and map one-year decrements into biological ages, as per equation (4). 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 phase one. Once again, there is only one degree of freedom according to the CLaM. Rather, the suggested and proposed way to obtain the required global GM parameters is to locate the implied $h[i]$ in a manner consistent with CLaM for that country's particular natural mortality growth rate $g[i]$. Accordingly, the next step is to average the mortality growth rates $g[i]$, to arrive at global value of G and then use the CLaM line to obtain an implied H.

4.4 Stage Four: Computing Biological Age

The global mortality-adjusted (biological) age: $\xi(x, i)$, of someone whose (chronological) age is x in country i, is constructed by equating mortality hazard rates per equation (3). With the CLaM in place, we proceed by assuming that the total hazard rate is classified entirely by the country-specific mortality growth rate g and the country-specific accidental death rate λ, so there is no need to explicitly include the initial natural mortality rate h. In particular, the definition of the global mortality-adjusted (biological) age from equation (3), can now be written as:

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

To be clear, the only parameters required at this point are the mortality growth rate $g[i]$, the accidental death (Makeham-constant) rate $\lambda[i]$, and the respective global averages G, and Λ, as well as the global plateau age x^* and natural plateau mortality rate λ^*. As to the global averages, they are defined arithmetically:

$$\begin{aligned} G &= \frac{1}{N}\sum_{i=1}^{N} g[i] \\ \Lambda &= \frac{1}{N}\sum_{i=1}^{N} \lambda[i], \end{aligned} \tag{18}$$

which will be discussed in section (5.1). For now, referring back to the formulation expressed in equation (15), the next step is to eliminate some redundant terms. Recall that the equation for ξ is designed to equate total hazard rates, so:

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

The objective now is to isolate the biological age ξ as a function of the (estimated) country-specific and global parameters. After dividing both sides of the above equation by $\lambda^* > 0$, the relevant equality can be re-written as:

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

In words, if the *global* total hazard rate at (biological) age ξ is equal to the *local* total hazard rate at age x, then the relationship between ξ and x, per equation (20), must satisfy:

$$e^{g[i]x + (G - g[i])x^*} - \left(\frac{\Lambda - \lambda[i]}{\lambda^*}\right) e^{Gx^*} = e^{G\xi}.$$

This expression is valid as long as the left-hand side is positive which (it is, and) will be justified in a moment. This then leads to the following expression for ξ as a function of x, and the key parameters:

$$\xi = x^* + \frac{1}{G} \ln\left[e^{g[i](x-x^*)} - \left(\frac{\Lambda - \lambda[i]}{\lambda^*}\right)\right]. \tag{21}$$

This expression only makes sense, or is properly defined, if the quantity: $(\Lambda - \lambda[i])/(\lambda^*)$ is smaller than $e^{g[i](x-x^*)}$. If not, the argument within the logarithm becomes negative. But, considering that Λ and $\lambda[i]$ are on the order of 10^{-5}, and $e^{g[i](x-x^*)}$ is many multiples greater, in practice this is a non-issue, especially over the adult-age ($x \geq 35$) range. In fact, $(\Lambda - \lambda[i])/(\lambda^*)$ itself is very close to zero.

Effectively, we are done. Equation (21) is the proper formula for ξ. It maps a *local* chronological age x, in country i, into a *global* mortality-adjusted (biological) age ξ. There are no approximations or assumptions made at this point, other than the (i.) Gompertz-Makeham law, and (ii.) the compensation law of mortality.

However, we can push this further by exploiting the relatively small (or near zero) value of the constant within the logarithm: $(\Lambda - \lambda[i])/(\lambda^*)$, in equation (21). It's possible to create a restricted and more compact version of equation (21), similar to the one presented at the very beginning of this section. Namely, ξ can be approximated by:

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

where the newly defined constant: $\kappa[i] = (g[i]/G) - 1$, is the *relative* aging rate. Although this is an approximation, the loss or error from ignoring $(\Lambda - \lambda[i])/(\lambda^*)$ is on the order of a few months. Under the approximation, the global mortality-adjusted (biological) age is expressed (only) as a function of (1.) the relative aging rate κ, and (2.) the species-specific lifespan x^*. And, if the mortality growth rate $g[i] = G$, which is the global average rate, then the global mortality-adjusted (biological) age is equal to chronological age. All of these are (expected and) consistent with intuition.

Here is a numerical example using the approximate expression for ξ, in (22). Assume the mortality plateau occurs at age: $x^* = 110$, chronological age is: $x = 50$, the natural mortality growth rate in country i is: $g[i] = 10\%$, the global average mortality growth rate is: $G = 9\%$, in which case the relative aging rate is: $\kappa_i = 11.11\% = 1/9$. For these parameters, the estimated global mortality-adjusted (biological) age is: $\xi = 50 - (110 - 50)/9 = 43\frac{1}{3}$. This individual mortality-adjusted (biological) age is 6.66 years less than their chronological age. Again, there is no need or mention of the initial natural (or terminal) mortality rate, which is embedded inside x^*.

Now, it's an open empirical question as to what happens after the species-specific lifespan x^*, and whether the (log) curve is constant in Figure #1 after age x^*. But, as modeled, it's assumed that from that age onward the *the total hazard rate is constant*, which once again implies that conditional lifetimes are exponentially distributed after they reach a chronological (and biological) age of x^*. The framework assumes no crossover in log-mortality rates.[9]

Focusing once again on the approximate (and intuitive) expression in equation (22), all else being equal, a larger value of x^* lowers the global mortality-adjusted (biological) age ξ, and the same is true for larger values of $\kappa[i]$.

[9]To this point, it is noted 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..."

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 natural 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 deemed to be younger. All else being equal, an individual with a lower global mortality-adjusted (biological) age, which means they have a lower-than-global-average total hazard rate, ages faster.

A final convenient item to remind readers is that (when using the approximate expression) the link between chronological age x and global mortality-adjusted (biological) age ξ doesn't require explicit knowledge of the current total hazard rate $\mu_x[i]$, or the country-specific decrement $q_x[i]$. That information is embedded (implicitly) in the parameters: x^*, $\kappa[i]$. Of course, the exact expression for ξ in equation (21) would require knowing the value of the accidental death rates $\lambda[i]$ relative to the plateau value λ^*.

To summarize, assuming a compensation law within the estimation procedure is not only realistic and consistent with current theories of aging, per Gavrilov and Gavrilova (1991), but also reduces the number of individual parameters required to estimate mortality-adjusted (biological) age.

5 Estimates: Biological Ages Around the World

Table #3 [**placed 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 $N = 37$ countries from the human mortality database (HMD) in the year 2011, the most recent year for which the largest and most complete country data is available. Each of the 37 decrement vectors $q_x[i]$ were used to iteratively estimate the best fitting $(h[i], \lambda[i], g[i])$ values for males and females separately. Those results were displayed and discussed in Table #1a and Table #1b, noted earlier. Note that the R-squares values (and all other summary statistics for goodness of fit) are not reported within Table #1, mainly because those values were all above 95%. The (very) good fit of the Gompertz-Makeham law of mortality on a country-by-country basis over the (chronological) age range $[35, 95]$ is well known in the demographic or actuarial literature, as per Tai and Noimer (2018). Log mortality rates are nearly linear in the range $x = 35$ to $x = 95$.

With the first stage regression numbers in hand in Table #1, the next step is to estimate 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.

The estimated (second phase) regression line is displayed in Figure #3, and the parameter estimates are presented in Table #2. In particular, the estimated male plateauing age was $x^* \approx 98$ and the female plateauing age was $x^* \approx 96$. The standard error for those point estimates was approximately 2 years for males and 6 years for females. Likewise, although the R-squared values were quite high (98.5%) for males, they were much lower (86%) for females. Practically speaking, it's entirely plausible the mortality plateau occurs much later in life, perhaps even at the age of 110 (or perhaps there is no plateau at all.) Indeed, this is still an open question in the bio-demographic and actuarial literature and arguably outside the scope of this article. See Barbi et al. (2018) for (much) more on this. Either way, the (+2 standard errors) upper and (-2 standard errors) lower bounds for x^*, which are required for equation (22) and equation (21), are used in Figure #4 [**placed here**], to provide a graphical range for the global mortality-adjusted (biological) ages.

For example, an $x = 55$-year-old Russian male has a biological age $\xi = 64.5$, whereas a $x = 55$-year-old Swedish male has a biological age of $\xi = 47.9$, which is a gap of almost 18 years between the youngest and oldest in Table #3. Using the same equation for females with a unique $g[i]$ and higher x^*, the largest gap at age $x = 55$, is between the Greek whose biological age is $\xi = 52.2$ and the Ukrainian whose global mortality-adjusted (biological) age is $\xi = 62.3$. The gap for females is (only) ten years. This (lower dispersion in age) is due to the fact that the range of mortality growth rates $g[i]$ themselves, 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 shrinks to zero across the different countries. This, effectively, is the compensation law of mortality in action, as the one-year mortality decrements $q_x[i]$ are converging as well.

Figure #4 (mentioned earlier) goes beyond point estimates and displays a range of global mortality-adjusted (biological) ages for each country, based on the (above-noted) upper and lower bounds 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 $\xi_{\texttt{low}}$ represents a global mortality-adjusted (biological) age 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.

5.1 Further Discussion of Assumptions

This paper, and the advocated framework, made a number of simplifying (and perhaps even *ad hoc*) assumptions which at this juncture are worth clarifying and defending. First and foremost, much of what motivates many of the the assumptions made was *convenience* plus a healthy respect and admiration for the history of the Gompertz law.

Now certainly, the Gompertz law of mortality might be one of the oldest, simplest and best-known laws of mortality, but it certainly isn't the only one. One could imagine implementing the same inversion with Perks (1932), Beard (1959), Weibull or logistic-based Vaupel-Kannisto models, perhaps as a follow-up. And yet, it's worth noting that Gavrilov and Gavrilova (2014) claim: "It was found that for all studied HMD birth cohorts, the Gompertz model demonstrated better fit of mortality data than the Kannisto model in the studied age interval." With all of that in mind, Table #4 **[goes here]** and Figure #5 **[goes here]** offer a historical view of the Gompertz-Makeham model to get a sense of how the estimated parameters have evolved over the last 50 years.

Indeed, aggregate mortality has been improving and the global Λ, G parameters do change over time and are not universal constants. Along the same lines, the Λ, G values were computed via a simple arithmetic average, although they could certainly have been weighted by population size, population wealth or even geographic country size. Another alternative would have been to match life expectancy $E[T_x] = E[T_\xi]$ instead of hazard rates, or perhaps use (inverted) phase-type laws, similar to Lin and Liu (2017) or Govuron et al. (2018). All these alternatives are equally valid, but equally questionable.

On a related note this paper is silent on the issue of forward-looking stochastic mortality, and how mortality-adjusted (biological) ages evolve over time, as per Lee-Carter for example. Rather, the current framework focuses on measuring *biological* age at a single point in time, as opposed to forecasting how it evolves over time and whether or not it's stochastic. As such, this paper is consistent with – and in fact can be used to calibrate parameters – in the work by Huang, et al. (2017), where mortality is assumed to evolve according to a Brownian Bridge. In particular, the estimates provided in this paper for (ages) x^* and (rates) λ^*, can be used to pin down the ends-points of the bridge.

6 Conclusion

This paper leverages the *compensation law of mortality* and the Gompertz-Makeham model to develop an expression for global mortality-adjusted (biological) ages. That is the main equation (21) in the paper. In its approximate form, equation (22) is a function of only: (1.) the local-relative-to-global mortality growth rate and (2.) the global plateauing age, which is the series-specific lifespan. This number is the (hypothesized) age at which all of natural mortality curves converge to a constant. The equation for global-mortality adjusted (biological) age was then calibrated to (adult) one-year mortality decrements in 37 countries from the Human Mortality Database (circa 2011) using a two-phase regression methodology. Using this approach, the data indicate that at chronological age 55, the so-called gap between high-mortality and low-mortality countries is as high as 18 years.

6.1 Behavioral Policy Implications and Uses

It's important to note – with an eye towards future research – that the identical methodology could be applied to heterogenous groups within a country or population, for example mortality rates based on income, wealth, race or education. Recall that all that is really needed to properly use the equation (22) is a relative mortality growth rate κ and a series-specific lifespan x^*. The rest is algebra[10].

Now, one could just as easily use the same mortality rates to compare period life expectancy values between high-mortality and low-mortality countries and arrive at similarly large gaps between the two extremes. In fact, the Organization for Economic Co-operation and Development (OECD) regularly publishes these reports comparing (more generally) quality of life across different countries and regions. However, one could argue that there are behavioral (a.k.a. psychological) reasons and benefits to *anchoring* on global mortality-adjusted (biological) ages versus life expectancy, since (simply put) these numbers are more memorable. In fact, by expressing the relevant integrals in terms of the Incomplete Gamma function, one can prove that the gap in global mortality-adjusted (biological) ages, 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 countries, or even 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 global mortality-adjusted (biological) age of the 65 year-old in group P is 72, versus 58 for group R, 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 informing said person that their life expectancy is actually 22 years, versus the population average of 19, and they should therefore wait to draw their pension. Will it be as effective as informing them they are much younger than their (chronological) age?

[10]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), results in a gap of almost 20 years. See Milevsky (2019) for a discussion of mortality heterogeneity and CLaM in the context of longevity risk pooling.

This definition of mortality-adjusted (biological) age 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,[11] 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 of the work of Benjamin Gompertz, this paper offers yet another application of his timeless model. He was the first to teach the world how to map (chronological) age into hazard rates, but this paper argues the same idea can be used in the other direction, to convert hazard rates into (biological) age.

[11] 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, and the work of Shoven and Goda (2008).

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Table # 1a

Gompertz-Makeham Parameters Around the World: MALE

Country	$\ln h[i]$	$\mu_0 = h + \lambda$	*Makeham:* λ	$g[i]$	$\mu_{55}[i]$	m	b
1. Australia	-11.854	66.8×10^{-5}	66.0×10^{-5}	11.19%	0.440%	86.37	8.94
2. Austria	-10.769	3.3×10^{-5}	1.0×10^{-5}	10.08%	0.596%	84.08	9.92
3. Belarus	-7.746	50.4×10^{-5}	4.0×10^{-5}	7.12%	2.335%	71.68	14.04
4. Belgium	-10.900	29.0×10^{-5}	27.0×10^{-5}	10.28%	0.610%	83.92	9.73
5. Canada	-11.292	37.4×10^{-5}	36.0×10^{-5}	10.53%	0.490%	85.85	9.50
6. Croatia	-10.156	5.3×10^{-5}	1.0×10^{-5}	9.75%	0.914%	80.29	10.26
7. Czechia	-10.258	4.9×10^{-5}	1.0×10^{-5}	9.81%	0.855%	80.88	10.19
8. Denmark	-10.835	16.2×10^{-5}	14.0×10^{-5}	10.25%	0.626%	83.50	9.76
9. Estonia	-9.045	13.8×10^{-5}	1.0×10^{-5}	8.33%	1.256%	78.72	12.00
10. Finland	-10.594	36.8×10^{-5}	34.0×10^{-5}	9.91%	0.679%	83.57	10.09
11. France	-10.378	35.4×10^{-5}	32.0×10^{-5}	9.44%	0.647%	84.92	10.59
12. Germany	-10.731	3.4×10^{-5}	1.0×10^{-5}	10.10%	0.625%	83.57	9.90
13. Greece	-10.485	17.1×10^{-5}	14.0×10^{-5}	9.67%	0.643%	84.26	10.34
14. Hungary	-9.421	9.9×10^{-5}	1.0×10^{-5}	8.98%	1.239%	78.07	11.13
15. Ireland	-11.291	39.4×10^{-5}	38.0×10^{-5}	10.67%	0.529%	84.87	9.37
16. Israel	-11.038	2.8×10^{-5}	1.0×10^{-5}	10.19%	0.486%	85.88	9.81
17. Italy	-11.647	23.0×10^{-5}	22.0×10^{-5}	11.06%	0.450%	85.39	9.04
18. Japan	-11.282	39.4×10^{-5}	38.0×10^{-5}	10.55%	0.502%	85.60	9.48
19. Korea	-10.803	37.3×10^{-5}	35.0×10^{-5}	10.19%	0.647%	83.59	9.81
20. Latvia	-8.517	22.6×10^{-5}	1.0×10^{-5}	7.86%	1.635%	75.98	12.72
21. Lithuania	-8.143	32.3×10^{-5}	1.0×10^{-5}	7.34%	1.772%	75.37	13.63
22. Luxembourg	-11.334	2.3×10^{-5}	1.0×10^{-5}	10.83%	0.517%	84.11	9.23
23. Netherlands	-11.628	18.0×10^{-5}	17.0×10^{-5}	11.12%	0.468%	84.83	8.99
24. New Zealand	-11.542	49.1×10^{-5}	48.0×10^{-5}	10.88%	0.478%	85.68	9.19
25. Norway	-11.566	33.1×10^{-5}	32.0×10^{-5}	11.01%	0.484%	85.00	9.08
26. Poland	-9.198	12.0×10^{-5}	1.0×10^{-5}	8.48%	1.172%	79.34	11.79
27. Portugal	-10.377	42.4×10^{-5}	39.0×10^{-5}	9.62%	0.721%	83.50	10.39
28. Russia	-8.246	315.3×10^{-5}	287.0×10^{-5}	7.62%	2.158%	74.43	13.12
29. Slovakia	-9.593	8.5×10^{-5}	1.0×10^{-5}	9.10%	1.117%	79.06	10.99
30. Slovenia	-10.559	3.9×10^{-5}	1.0×10^{-5}	10.02%	0.712%	82.40	9.98
31. Spain	-10.827	3.2×10^{-5}	1.0×10^{-5}	10.04%	0.551%	84.92	9.96
32. Sweden	-11.898	25.8×10^{-5}	25.0×10^{-5}	11.40%	0.427%	85.34	8.77
33. Switzerland	-11.721	25.9×10^{-5}	25.0×10^{-5}	11.07%	0.424%	86.02	9.04
34. Taiwan	-9.849	97.8×10^{-5}	92.0×10^{-5}	8.89%	0.859%	83.56	11.25
35. U.K.	-11.386	64.3×10^{-5}	63.0×10^{-5}	10.73%	0.525%	85.30	9.32
36. U.S.A.	-10.275	65.8×10^{-5}	62.0×10^{-5}	9.42%	0.735%	84.01	10.62
37. Ukraine	-8.616	223.6×10^{-5}	204.0×10^{-5}	8.12%	1.914%	75.18	12.31
Average:	$\mathbf{-10.427}$	$\mathbf{41.0 \times 10^{-5}}$	$\mathbf{34.3 \times 10^{-5}}$	**9.77%**	**0.844%**	**82.41**	**10.39**

Source: Human Mortality Database, Period 2011

Table # 1b

Gompertz-Makeham Parameters Around the World: FEMALE

Country	$\ln h[i]$	$\mu_0 = h + \lambda$	*Makeham:* λ	$g[i]$	$\mu_{55}[i]$	m	b
1. Australia	-12.349	31.5×10^{-5}	31.0×10^{-5}	11.29%	0.273%	90.04	8.85
2. Austria	-11.952	4.7×10^{-5}	4.0×10^{-5}	10.99%	0.308%	88.65	9.10
3. Belarus	-10.797	76.3×10^{-5}	74.0×10^{-5}	10.21%	0.695%	83.44	9.80
4. Belgium	-11.620	15.0×10^{-5}	14.0×10^{-5}	10.60%	0.355%	88.43	9.43
5. Canada	-11.773	18.9×10^{-5}	18.0×10^{-5}	10.64%	0.317%	89.56	9.40
6. Croatia	-11.784	1.9×10^{-5}	1.0×10^{-5}	11.25%	0.416%	85.34	8.89
7. Czechia	-12.009	19.7×10^{-5}	19.0×10^{-5}	11.44%	0.388%	86.01	8.74
8. Denmark	-11.608	2.0×10^{-5}	1.0×10^{-5}	10.75%	0.376%	87.21	9.30
9. Estonia	-11.132	18.6×10^{-5}	17.0×10^{-5}	10.20%	0.460%	86.75	9.80
10. Finland	-11.860	9.8×10^{-5}	9.0×10^{-5}	10.84%	0.315%	88.90	9.22
11. France	-11.614	22.0×10^{-5}	21.0×10^{-5}	10.25%	0.302%	91.08	9.75
12. Germany	-11.898	11.8×10^{-5}	11.0×10^{-5}	11.00%	0.332%	88.12	9.09
13. Greece	-12.594	24.4×10^{-5}	24.0×10^{-5}	11.78%	0.272%	88.77	8.49
14. Hungary	-10.812	3.2×10^{-5}	1.0×10^{-5}	10.14%	0.590%	84.06	9.86
15. Ireland	-11.725	8.9×10^{-5}	8.0×10^{-5}	10.75%	0.342%	88.29	9.30
16. Israel	-12.494	23.4×10^{-5}	23.0×10^{-5}	11.64%	0.277%	88.84	8.59
17. Italy	-12.478	15.4×10^{-5}	15.0×10^{-5}	11.49%	0.252%	89.80	8.71
18. Japan	-12.344	38.5×10^{-5}	38.0×10^{-5}	11.00%	0.245%	92.12	9.09
19. Korea	-13.266	47.2×10^{-5}	47.0×10^{-5}	12.46%	0.233%	89.77	8.03
20. Latvia	-10.646	20.6×10^{-5}	18.0×10^{-5}	9.86%	0.612%	84.51	10.15
21. Lithuania	-10.649	43.6×10^{-5}	41.0×10^{-5}	9.74%	0.597%	85.39	10.26
22. Luxembourg	-11.877	1.8×10^{-5}	1.0×10^{-5}	10.92%	0.316%	88.47	9.16
23. Netherlands	-11.700	12.9×10^{-5}	12.0×10^{-5}	10.73%	0.350%	88.22	9.32
24. New Zealand	-12.079	33.6×10^{-5}	33.0×10^{-5}	11.17%	0.329%	88.48	8.95
25. Norway	-12.006	5.7×10^{-5}	5.0×10^{-5}	11.05%	0.303%	88.68	9.05
26. Poland	-11.280	10.4×10^{-5}	9.0×10^{-5}	10.45%	0.449%	86.31	9.57
27. Portugal	-12.249	27.5×10^{-5}	27.0×10^{-5}	11.31%	0.297%	89.00	8.84
28. Russia	-11.156	160.6×10^{-5}	159.0×10^{-5}	10.65%	0.714%	83.74	9.39
29. Slovakia	-11.560	10.1×10^{-5}	9.0×10^{-5}	11.00%	0.460%	85.04	9.09
30. Slovenia	-11.683	1.9×10^{-5}	1.0×10^{-5}	10.73%	0.345%	88.07	9.32
31. Spain	-12.362	17.5×10^{-5}	17.0×10^{-5}	11.24%	0.248%	90.57	8.90
32. Sweden	-12.377	11.5×10^{-5}	11.0×10^{-5}	11.51%	0.276%	88.79	8.69
33. Switzerland	-12.233	6.5×10^{-5}	6.0×10^{-5}	11.15%	0.256%	90.05	8.97
34. Taiwan	-12.051	40.7×10^{-5}	40.0×10^{-5}	11.15%	0.341%	88.40	8.97
35. U.K.	-11.861	29.8×10^{-5}	29.0×10^{-5}	10.90%	0.345%	88.48	9.17
36. U.S.A.	-11.063	41.7×10^{-5}	40.0×10^{-5}	9.97%	0.457%	87.83	10.03
37. Ukraine	-11.460	129.2×10^{-5}	128.0×10^{-5}	11.12%	0.662%	83.29	8.99
Average:	$\mathbf{-11.795}$	$\mathbf{27.0 \times 10^{-5}}$	$\mathbf{26.0 \times 10^{-5}}$	**10.90%**	**0.381%**	**87.80**	**9.20**

Source: Human Mortality Database, Period 2011

Table #2:						
Compensation Law Regression Line Around the World						
Variable	**MALE**			**FEMALE**		
	Coeff.	Std.Er	t-val.	Coeff.	Std.Er	t-val.
Intercept (L)	-0.851	0.193	-4.413	-1.289	0.705	-1.828
Slope: ($-x^*$)	**-97.964**	1.960	-49.987	**-96.360**	6.459	-14.919
Adj. R^2	98.58%			86.02%		
Range: $g[i]$	$(7.12\%, 11.40\%)$			$(9.74\%, 12.46\%)$		
Average: $g[i]$	G = 9.77%%			G = 10.90%%		
Plateau (+/-): λ^*	$(0.321, 0.547)$			$(0.176, 0.477)$		
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. 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.

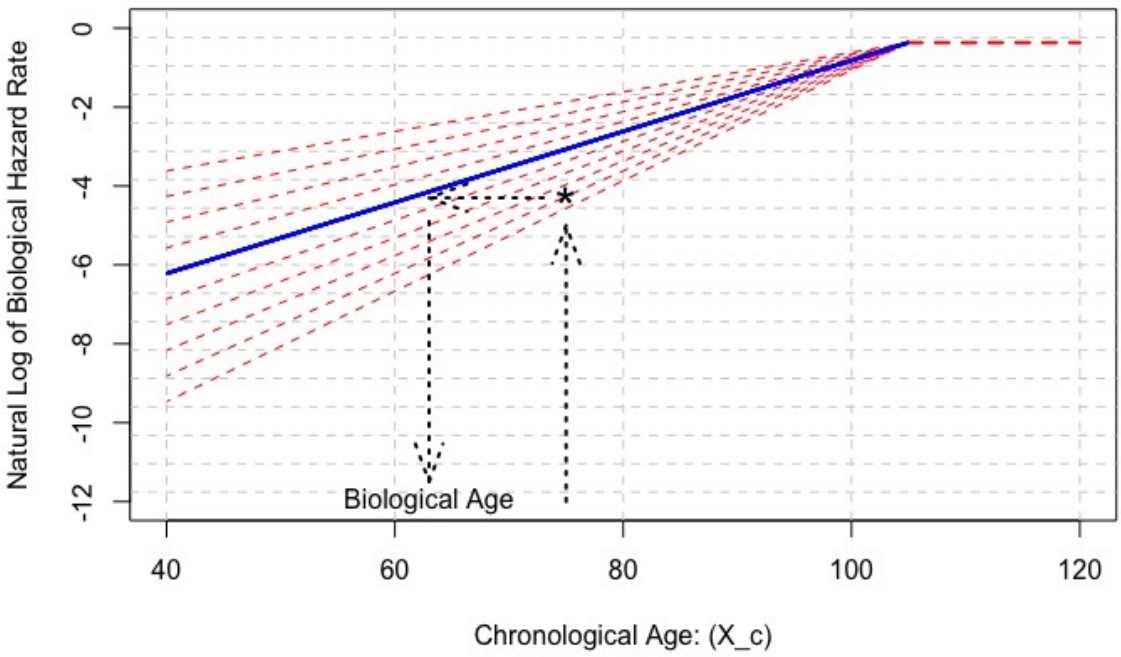
Table # 3						
Mortality-Adjusted Biological Ages (ξ) Around the World						
	MALE			**FEMALE**		
Country	$x = 55$	$x = 70$	$x = 85$	$x = 55$	$x = 70$	$x = 85$
1. Australia	48.79	65.96	83.12	53.52	69.06	84.59
2. Austria	53.66	69.13	84.60	54.66	69.79	84.91
3. Belarus	66.67	77.59	88.52	57.64	71.69	85.73
4. Belgium	52.80	68.57	84.33	56.14	70.72	85.31
5. Canada	51.67	67.84	84.00	55.98	70.63	85.27
6. Croatia	55.11	70.07	85.03	53.69	69.16	84.64
7. Czechia	54.83	69.89	84.95	52.96	68.70	84.44
8. Denmark	52.93	68.65	84.37	55.56	70.36	85.15
9. Estonia	61.34	74.12	86.91	57.66	71.70	85.73
10. Finland	54.40	69.61	84.82	55.23	70.15	85.06
11. France	56.46	70.95	85.44	57.47	71.57	85.68
12. Germany	53.58	69.08	84.57	54.64	69.77	84.90
13. Greece	55.46	70.30	85.14	51.68	67.88	84.09
14. Hungary	58.49	72.27	86.05	57.89	71.84	85.79
15. Ireland	51.08	67.45	83.82	55.57	70.36	85.16
16. Israel	53.16	68.80	84.44	52.19	68.21	84.23
17. Italy	49.35	66.32	83.30	52.79	68.59	84.39
18. Japan	51.58	67.78	83.97	54.62	69.75	84.89
19. Korea	53.17	68.81	84.45	49.10	66.24	83.38
20. Latvia	63.40	75.47	87.54	58.97	72.53	86.09
21. Lithuania	65.71	76.97	88.23	59.40	72.80	86.21
22. Luxembourg	50.36	66.98	83.60	54.93	69.95	84.98
23. Netherlands	49.10	66.16	83.22	55.64	70.41	85.18
24. New Zealand	50.13	66.83	83.53	53.97	69.34	84.72
25. Norway	49.56	66.46	83.36	54.42	69.63	84.84
26. Poland	60.67	73.69	86.71	56.71	71.09	85.47
27. Portugal	55.66	70.43	85.20	53.44	69.00	84.57

28. Russia	64.47	76.16	87.86	55.97	70.62	85.27
29. Slovakia	57.96	71.92	85.89	54.64	69.77	84.90
30. Slovenia	53.91	69.29	84.67	55.65	70.41	85.18
31. Spain	53.82	69.23	84.64	53.74	69.19	84.65
32. Sweden	47.87	65.36	82.85	52.71	68.54	84.37
33. Switzerland	49.32	66.30	83.29	54.06	69.40	84.74
34. Taiwan	58.89	72.53	86.17	54.06	69.40	84.74
35. U.K.	50.79	67.26	83.73	55.01	70.01	85.00
36. U.S.A.	56.57	71.02	85.47	58.53	72.25	85.97
37. Ukraine	62.27	74.73	87.19	54.17	69.47	84.77
Average:	**55.00**	**70.00**	**85.00**	**55.00**	**70.00**	**85.00**
Source: Human Mortality Database, Period 2011						

Table # 4				
The Canonical Gompertz Regression: Historical Coefficients				
	Modal Value m		**Dispersion** b	
Year	MALE	FEMALE	MALE	FEMALE
1945	**76.60** ±1.98	**79.89** ±1.08	**10.45** ±1.05	**9.77** ±0.87
1948	**76.97** ±2.04	**80.76** ±0.86	**10.95** ±0.78	**9.78** ±0.57
1951	**76.58** ±2.17	**80.38** ±0.90	**10.54** ±0.83	**9.44** ±0.53
1954	**77.33** ±1.67	**81.18** ±0.73	**10.49** ±0.64	**9.47** ±0.51
1957	**77.03** ±1.69	**81.29** ±0.76	**10.48** ±0.60	**9.33** ±0.49
1960	**77.38** ±1.54	**81.74** ±0.73	**10.37** ±0.67	**9.18** ±0.49
1963	**77.08** ±1.48	**81.76** ±0.75	**10.37** ±0.62	**9.19** ±0.47
1966	**77.30** ±1.53	**82.26** ±0.87	**10.53** ±0.59	**9.24** ±0.48
1969	**77.20** ±1.55	**82.34** ±0.94	**10.60** ±0.57	**9.41** ±0.50
1972	**77.71** ±1.32	**83.03** ±0.80	**10.61** ±0.54	**9.34** ±0.54
1975	**77.93** ±1.23	**83.51** ±0.86	**10.55** ±0.55	**9.39** ±0.58
1978	**78.43** ±1.06	**84.18** ±0.87	**10.55** ±0.51	**9.31** ±0.66
1981	**78.92** ±0.97	**84.60** ±0.87	**10.42** ±0.49	**9.32** ±0.62
1984	**79.50** ±0.96	**85.16** ±0.88	**10.35** ±0.47	**9.34** ±0.56
1987	**80.03** ±0.86	**85.55** ±0.88	**10.27** ±0.41	**9.30** ±0.56
1990	**80.57** ±0.92	**85.85** ±0.99	**9.99** ±0.39	**9.15** ±0.60
1993	**81.08** ±1.06	**86.13** ±1.17	**9.76** ±0.35	**9.06** ±0.51
1996	**81.70** ±1.05	**86.65** ±1.10	**9.67** ±0.46	**9.08** ±0.52
1999	**82.12** ±0.99	**86.92** ±1.12	**9.56** ±0.50	**9.06** ±0.42
2002	**82.77** ±0.99	**87.32** ±1.17	**9.54** ±0.52	**9.06** ±0.40
2005	**83.47** ±1.02	**88.04** ±1.10	**9.55** ±0.59	**9.11** ±0.40
2008	**84.08** ±0.99	**88.49** ±1.07	**9.59** ±0.64	**9.13** ±0.40
2011	**84.83** ±0.94	**89.11** ±1.06	**9.59** ±0.63	**9.18** ±0.38
Source: Human Mortality Database, Period 1945-2011, 17 Countries				

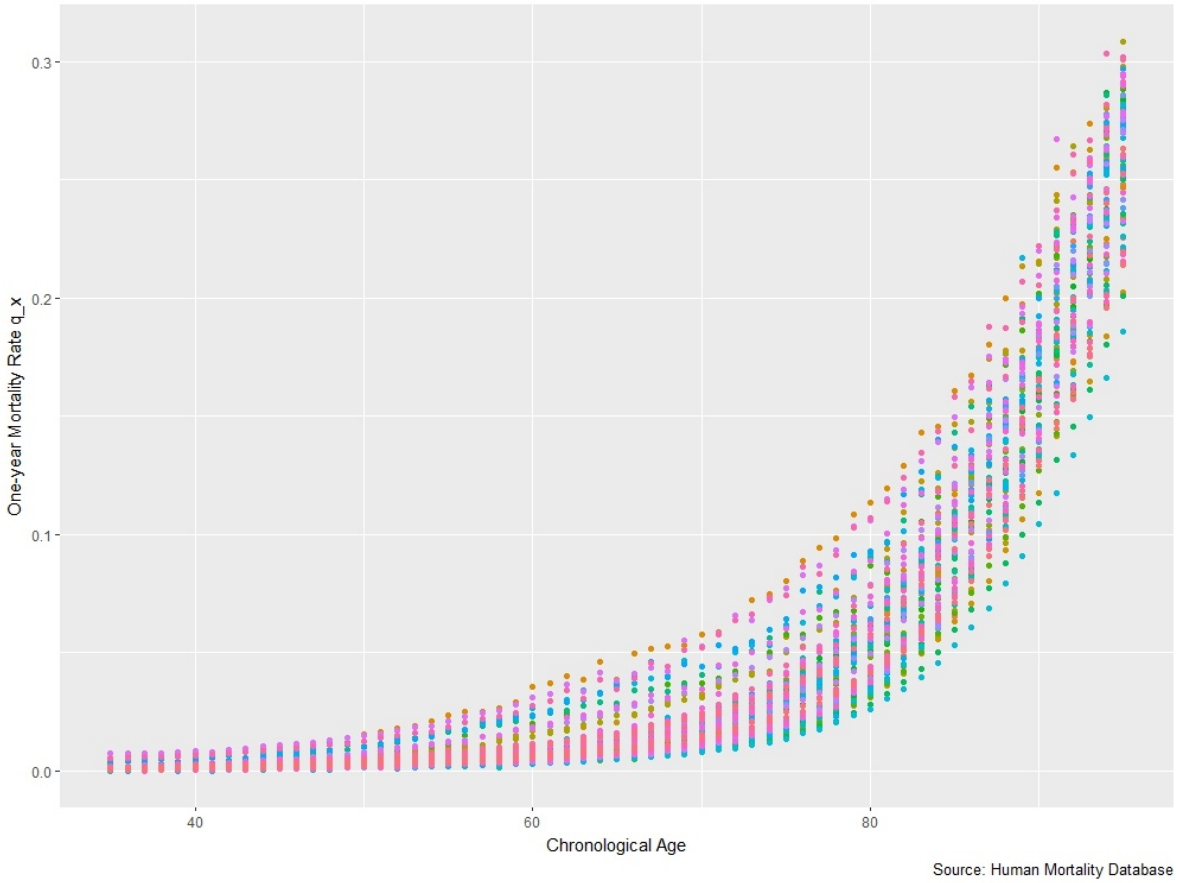
Note: The phase one procedure was applied to historical period mortality decrement data from the HMD, resulting in the following estimates for global (m, b) parameters. Notice that the mortality dispersion b is consistently larger for males versus females, and vice versa for the mortality growth rate $g = 1/b$. Over the years, m has increased as b has declined.

Figure 1: Visualizing Gompertz and the Compensation Law of Mortality



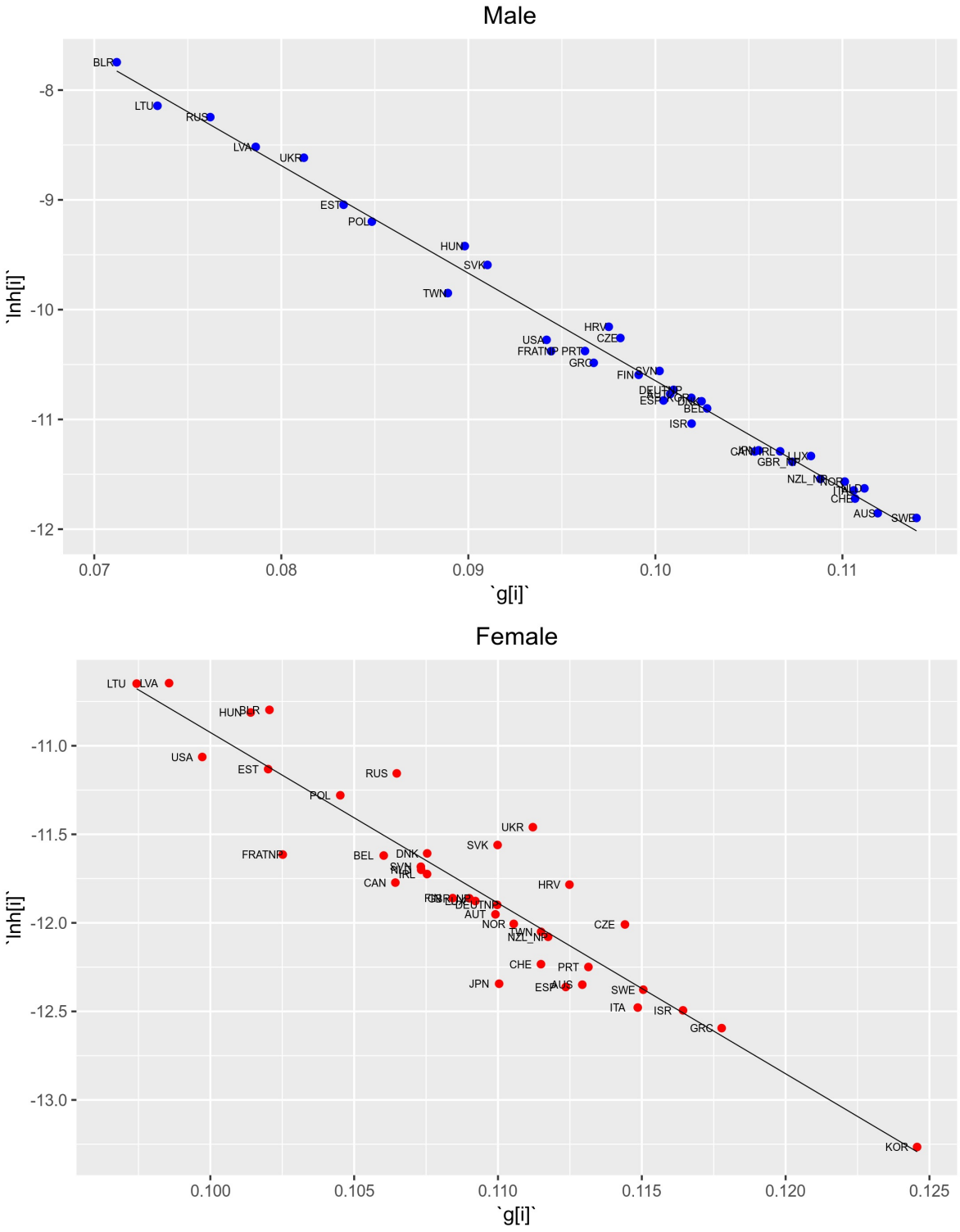
Note: The *compensation law of mortality* in its strong form implies that (log) mortality rates increase linearly and then converge to a constant mortality plateau. This leads to a linear and negative relationship between the (initial natural mortality) intercept: $\ln h[i]$, and the (mortality growth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

Figure 2: One-year Mortality Decrement Rates Around the World



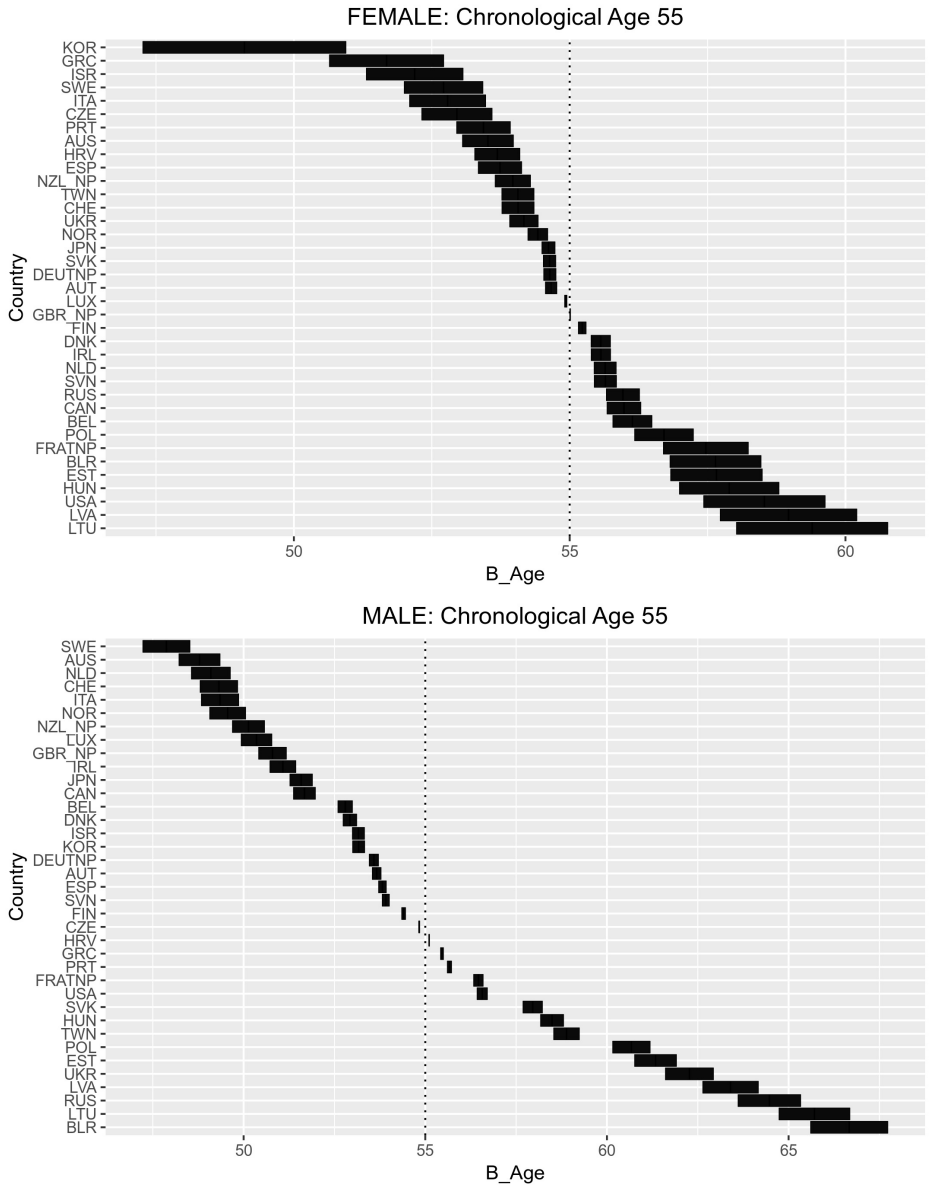
Note: Raw one-year decrement q_x data used in the first phase regression, from 37 countries (male and female) in the Human Mortality Database. University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany). Available at www.mortality.org or www.humanmortality.de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

Figure 3: Growth Rates g, vs. (log) Initial Natural Mortality Rates $\ln[h]$, around the World



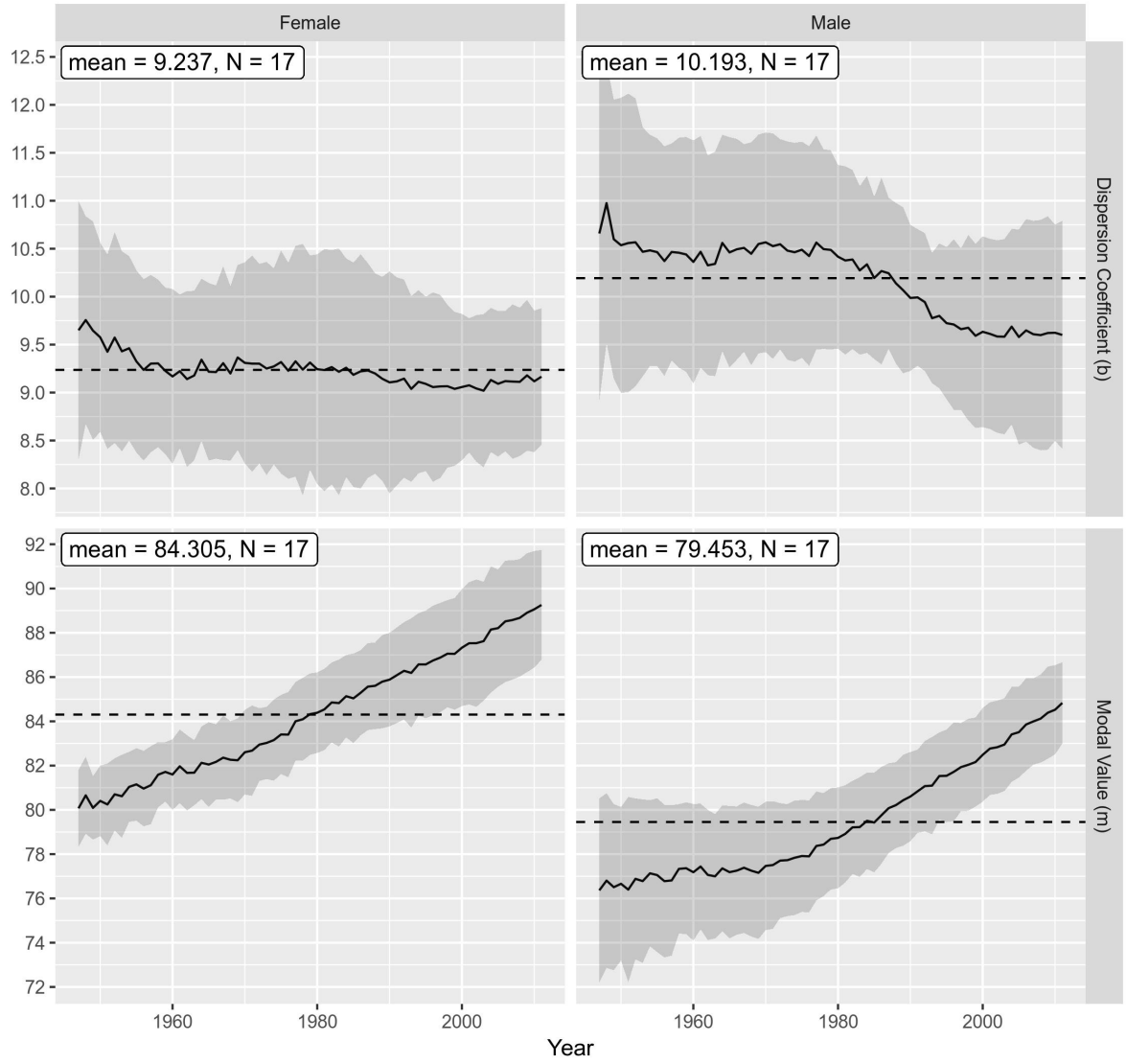
Note: There is a negative relationship between the mortality growth rate $g[i]$ in a given country, and the (log) of initial natural mortality rate $\ln[h[i]]$. This is consistent with the *compensation law of mortality*, central to the procedure used to compute mortality-adjusted (biological) ages. The estimated slope and intercept values are displayed in Table #2.

Figure 4: Range of Global Mortality-adjusted (biological) Ages at (chronological) Age 55.



Note: The lower values for mortality-adjusted (biological) age $\xi_{\texttt{low}}$ and upper value $\xi_{\texttt{high}}$ are based on the (exact) equation (21). However, the point estimates of $x^* = 97.96$ for males and $x^* = 96.36$ for females, are replaced with plus or minus two standard errors, which are 1.96 years (male) and 6.46 years (female) respectively, as estimated in the second phase regression (and displayed in Table #2.)

Figure 5: Estimated Gompertz Parameters and their Range over Time



Note: This figure displays the historical Gompertz parameters using the (m, b) formulation, based on the regression methodology described in the paper as phase #1, across 17 countries from which historical data is available (to 1945) in the *Human Mortality Database*. The shaded regions capture (+/-) two standards errors, listed in Table #4.