How Much Can We Trust Life Tables?

Sensitivity of Mortality Measures to Right-Censoring Treatment

Trifon I. Missov[1,2], László Németh[1], and Maciej J. Dańko[1]

[1]Max Planck Institute for Demographic Research, Konrad-Zuse-Str. 1, 18057 Rostock, Germany

[2]Mathematical Demography, University of Rostock, Ulmenstr. 69, 18057 Rostock, Germany

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Abstract

International organizations, research institutions, insurance companies, pension funds, and health policy makers calculate human mortality measures from life tables. Life-table data, though, are usually right-censored and mortality measures are sensitive to the way censoring is addressed. In this article we propose fitting a parametric model that describes well human mortality patterns, the gamma-Gompertz-Makeham, accounting for censoring, and constructing model-based equivalents of five mortality measures: life expectancy, the modal age at death, life disparity, entropy, and the Gini coefficient. We show that, in comparison to life-table measures, model-based measures are less sensitive to the age at censoring and can be only slightly distorted even if the age at censoring is low. We also compare life-table and model-based mortality measures for a population with an underlying Gompertz mortality schedule in which a fixed proportion of the population is censored.

Keywords: right censoring; life tables; gamma-Gompertz-Makeham frailty model; model-based mortality measures; life expectancy; modal age at death; life disparity; entropy; Gini coefficient

1 Right-Censoring and Life-Table Mortality Measures

Human mortality data are aggregated in life tables that describe the distribution of deaths in a given period (or cohort) and directly provide mortality measures such as the age-specific death rates and (remaining) life expectancy at each age. Statistical offices, international organizations, research institutions, insurance companies, pension funds, and health policy makers calculate, compare, and project a number of mortality and longevity measures derived from the life table. However, statistical offices often provide age-specific death counts up to a given age x_C (e.g., $x_C = 80, 85, 90, 95, 100$) aggregating all subsequent death counts in a "x_C+" group, i.e., mortality data are right-censored.

Life expectancy, perhaps the most widely used longevity measure, is sensitive to the way we address right-censoring, i.e., what we assume about the exposure in the last (open-end) age group. The ${}_na_x$ column of the life table contains the average number of person-years lived between ages x and $x + n$ by an individual who died in this interval. In the absence of accurate individual data, ${}_na_x$ are usually taken either from the Coale-Demeny model life tables (Coale and Demeny, 1966) or by assuming $n/2$ exposure in all intervals $[x, x + n)$ but the first (capturing infant mortality) and the last (open-age) one (Wilmoth et al., 2007). Remaining life expectancy at any age x, calculated by life-table algebra, depends on the choice of the ${}_na_x$ column and especially on ${}_\infty a_x$, the average number of person years lived by an individual that dies in the last open-age interval. The standard assumption is that ${}_\infty a_x$ equals the reciprocal of the death rate in the last age category: ${}_\infty a_x = 1/m_x$, i.e., after the last available age in the life table individuals are exposed to a constant hazard [1] of death (Preston et al., 2001). This is a strong assumption that might not be empirically justified, especially when the open-end interval starts at an age with a high mortality rate: modifying ${}_\infty a_x$ leads to a completely new age vector of ${}_nL_x$ that affects remaining life expectancies at all ages.

Life tables impose a mortality model on the last open-end age group. When this model

[1] Throughout this article we will use the terms *hazard of death*, *hazard*, *hazard function*, *risk of dying*, and *force of mortality* interchangeably.

does not reflect the preceding age-pattern of mortality, the associated mortality measures will be distorted: the bigger the proportion of observations subjected to this type of censoring, the larger the distortion. Life tables for many (historical and contemporary) populations leave 10% or more of the population in the open-end interval (see Table 1) which questions the resulting mortality indicators, such as life expectancy, life disparity, entropy, or the Gini coefficient, that are widely used by governments, international organizations, and insurance companies. The credibility of reported mortality measures for many developing countries is questionable because the open-end age group contains a substantial proportion of the population. This problem is to be observed not only in historical populations, e.g. Bangladeshi life tables from 1974 to 1981 that end up with a 65+ or a 70+ open-end interval containing between 26% and 55% of the population (HLTD, 2015), but also in contemporary life tables like the ones for Brazil in 2007 – the last 80+ age group contains 34.16% of males and 50.89% of females[2].

In this article we address right-censoring in a typical survival-analysis setting. We fit a parametric model, the gamma-Gompertz-Makeham (ΓGM, throughout the paper) assuming that the death counts $D(x)$ at adult ages x are Poisson-distributed (Brillinger, 1986), i.e., $D(x) \sim \text{Poisson}(E(x)\mu(x))$, where $E(x)$ denotes age-specific exposure, and $\mu(x)$ is the ΓGM hazard of death at age x:

$$\mu(x) = \frac{ae^{bx}}{1 + \frac{a\gamma}{b}(e^{bx} - 1)} + c\,. \tag{1}$$

Parameter a denotes the level of senescent mortality at the starting age of analysis, b is the rate of individual aging, c is an age-constant external risk of death that is, in general, not related to the aging process, and γ equals the squared coefficient of variation of the distribution of unobserved heterogeneity (frailty). The gamma-Gompertz-Makeham frailty model is widely used in human mortality research as it captures well the S-shaped pattern of mortality at adult ages. For detailed discussion on the semantics and mathematics behind

[2]2007 mortality data for Brazil are freely available at the website of Instituto Brasileiro de Geografia e Estatística (IBGE): http://www.ibge.gov.br/home/estatistica/populacao/tabuadevida/2007/defaulttab.shtm

the ΓGM we redirect our readers to Vaupel et al. (1979), Missov and Finkelstein (2011), Vaupel and Missov (2014), and Missov and Vaupel (2015). We use maximum likelihood for fitting the ΓGM model, i.e., we maximize a Poisson log-likelihood

$$\ln L = \sum_x [D(x) \ln \mu(x) - E(x)\mu(x)] \,, \tag{2}$$

in which $E(x)$ contains all the information on censoring.

We compute model-based equivalents of five frequently used mortality measures (remaining life expectancy, the modal age at death, life disparity, entropy, and the Gini coefficient) that are based on the estimated ΓGM parameters. We illustrate the lower sensitivity of model-based mortality measures to the age at censoring in comparison to their life-table counterparts. As model-based mortality measures are only slightly distorted even when the age at censoring is low (or, what is equivalent, the proportion of censored individuals is high), we argue that international organizations, research institutions, insurance companies, pension funds, and even evolutionary biologists working with non-human life tables should use model-based mortality measures.

2 ΓGM Model-Based Mortality Measures

We will focus on the five perhaps most widely used mortality measures: $e(x)$, remaining life expectancy at age x; M, the modal age at death; $e^\dagger$, life disparity; H, entropy; and G, the Gini coefficient. For life-table calculation of these (and other) mortality measures we refer the reader to Shkolnikov and Andreev (2010), the only exception being the modal age at death. To avoid random fluctuation in the density of deaths that might affect M (see Figure 1), we apply LOESS smoothing ('loess' function in the 'stats' R-package) with a smoothing parameter equal to 0.25. The modal age at death is taken then with high precision directly from the values interpolated by LOESS.

1. Remaining life expectancy at x ($x \geq 0$) provides the average remaining lifespan of survivors to age x. It is calculated as

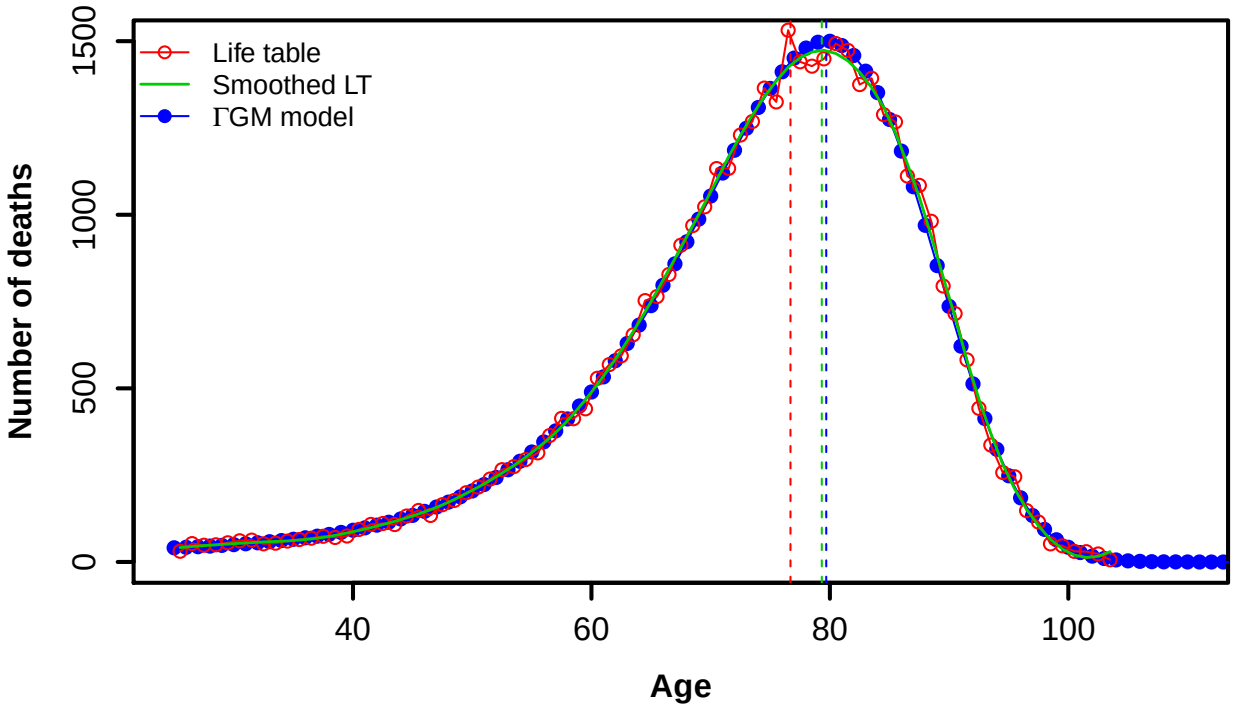

Figure 1: The effect of random fluctuation on the identification of the modal age at death (identified by a dashed line). The red curve corresponds to the d_x column for the Swedish male population in 1970 (Source: HMD, 2015). Without smoothing, the modal age at death is misspecified by about 3 years due to random fluctuation. The ΓGM fit (blue curve) is almost identical to the non-parametrically smoothed (by LOESS) d_x (green curve).

$$e(x) = \frac{1}{s(x)} \int_x^\infty s(t)\, dt\,, \tag{3}$$

where $s(x)$ denotes the survival function of the distribution of deaths. A ΓGM life expectancy can be calculated by either substituting the ΓGM survival function

$$s(x) = e^{-cx} \left(1 + \frac{a\gamma}{b}(e^{bx} - 1)\right)^{-1/\gamma}\,, \tag{4}$$

in (3) and taking the resulting integral numerically or taking advantage of a closed-form expression containing hypergeometric series (see Missov and Lenart, 2013: section 2.3,

p.30–31).

2. The modal age at death M, i.e., the age of highest concentration of deaths in a population, is an important indicator for policy makers as this is the age around which public health spends most of its resources. The modal age at death in a ΓGM is determined by maximizing $\mu(x) \cdot s(x)$ (see eq. 1 and 4).

3. Life disparity $e^\dagger$ measures, on the one hand, how much lifespans differ among individuals and, on the other hand, how many life years are lost due to death (Keyfitz, 1977). It is defined as the average remaining life expectancy at ages when deaths occur:

$$e^\dagger = \int_0^\infty e(x)\mu(x)s(x)\,dx = -\int_0^\infty s(x)\ln s(x)\,dx\,. \tag{5}$$

In a ΓGM setting, $e^\dagger$ is calculated by numerical integration of (5) taking $s(x)$ from (4).

4. Entropy H measures heterogeneity in the age at death or, alternatively, the elasticity of life expectancy with respect to proportional changes in age-specific mortality (Keyfitz, 1977). It is defined as

$$H = \frac{-\int_0^\infty s(x)\ln s(x)\,dx}{\int_0^\infty s(x)\,dx} = \frac{e^\dagger}{e(0)}\,. \tag{6}$$

In a ΓGM setting, we take $s(x)$ from (4) and calculate $e(0)$ and $e^\dagger$ from (3) and (5), respectively.

5. For a given population, the Gini coefficient G measures inter-individual inequality in the length of life (Shkolnikov et al., 2003). It is defined as

$$G = 1 - \frac{\int_0^\infty s^2(x)\,dx}{\int_0^\infty s(x)\,dx} = 1 - \frac{\int_0^\infty s^2(x)\,dx}{e(0)}\,. \tag{7}$$

G can be also represented as the mean of the absolute differences in individual ages of death relative to life expectancy (Kendall and Stuart, 1966). The range of the Gini coefficient is $[0, 1]$ with 1 representing a population in which all deaths occur at the same time. In most developed countries the Gini coefficient increases with time as many early deaths are postponed to later ages. Demographers address this phenomenon as "life-table rectangulatization" referring to the shape of the corresponding survival curve (Shkolnikov et al., 2003). In a ΓGM setting, we calculate the Gini coefficient by substituting (4) in (7) and integrating the corresponding expressions numerically.

3 Life-Table vs Model-Based Mortality Measures

Life-table mortality measures depend on the way the life table is "closed", i.e., on the assumption about exposure in the last (open-age) interval. Most often life-table calculations are based on the assumption that the hazard after the age at censoring x_C is constant (Preston et al., 2001), and the lower x_C, the more unrealistic this assumption (the force of mortality at adult ages has an S-shaped strictly increasing pattern). In this section we illustrate by how much life-table mortality measures can be distorted if the age at censoring is low, i.e., when a high proportion of individuals is censored. While the latter is rather rarely seen in human mortality data, perhaps except for some developing countries, it is common practice in experiments with non-human species – researchers wait until a certain (not necessarily high) percentage of the organisms die. We show for both human and non-human data that model-based mortality measures must be used because they accurately account for censoring. As a result they can only be slightly distorted even if the age at censoring is low, i.e., when the proportion of censored individuals is high. To illustrate that this effect is not restricted to the ΓGM model, i.e., to the model that describes best adult human mortality (Missov and Vaupel, 2015), we consider an additional example with experimental non-human data (rats) where the mortality pattern is well captured by a Gompertz model.

3.1 Sensitivity to Censoring in Human Mortality

We simulate individual lifespans from a ΓGM model: the generating parameters correspond to the estimated ΓGM parameters ($a = 3.28 \cdot 10^{-4}$, $b = 0.105$, $c = 6.52 \cdot 10^{-4}$, $\gamma = 0.094$) for Swedish males in 1970, ages 25-110. Our choice fell on this population because of its clear S-shaped mortality pattern. We aggregate the death counts and exposures age-wise to construct a life table. We compare then five life-table mortality measures - remaining life expectancy, the modal age at death, life disparity, entropy, and the Gini coefficient - to their ΓGM model-based counterparts, knowing the true value of each measure.

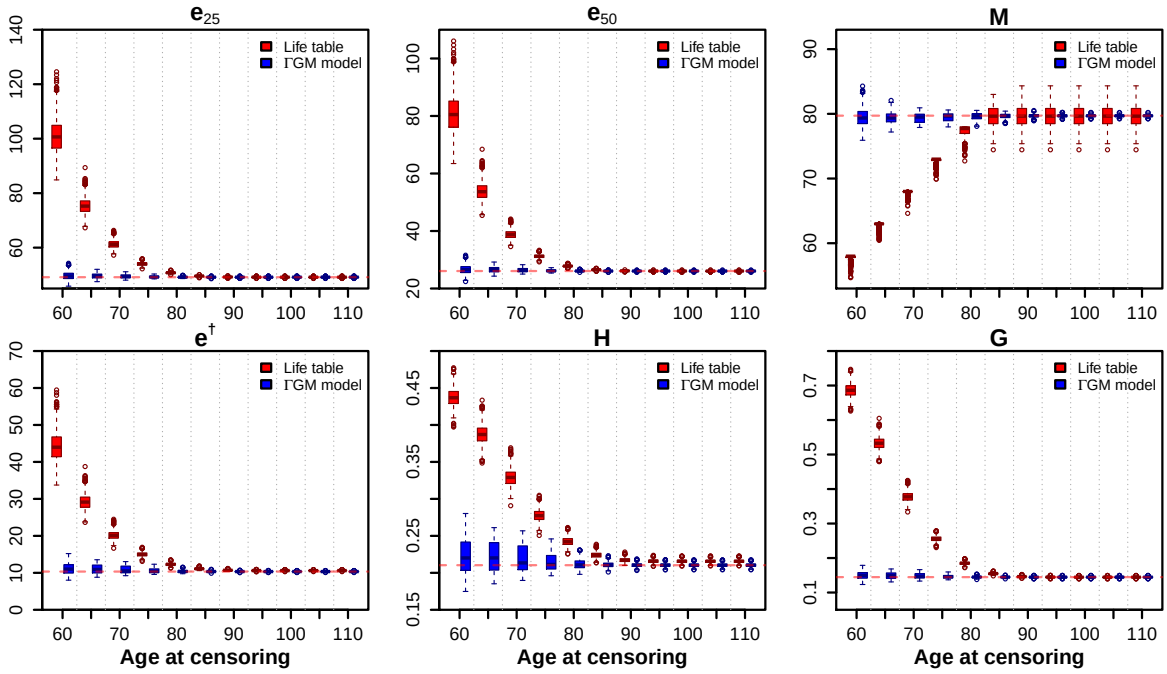

Figure 2: Life-table (red) vs ΓGM model-based (blue) mortality measures for a population of size 10000. Individual lifetimes have been simulated from a ΓGM (1000 repetitions): the resulting death counts and exposures have been aggregated age-wise. The red dashed line in each graph denotes the true value of the measure.

Figures 2 and 3 show the life-table vs model-based versions of the five mortality measures (we considered remaining life expectancy at two ages: 25 and 50) for populations of size 10000

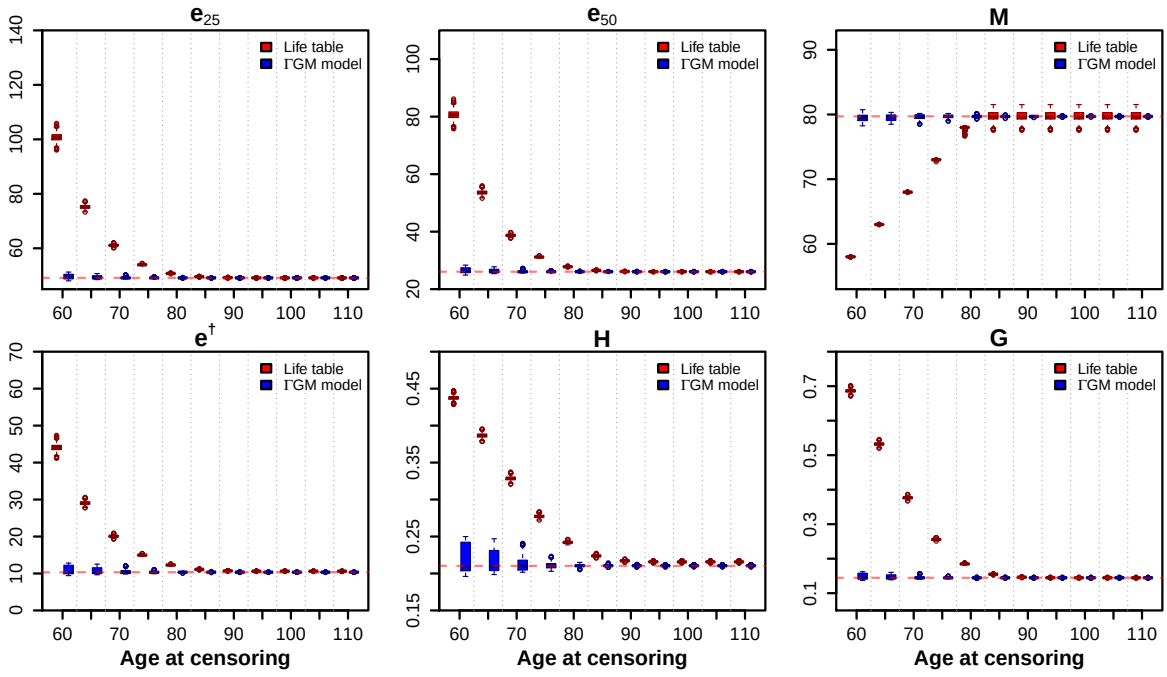

Figure 3: Life-table (red) vs ΓGM model-based (blue) mortality measures for a population of size 200000. Individual lifetimes have been simulated from a ΓGM (1000 repetitions): the resulting death counts and exposures have been aggregated age-wise. The red dashed line in each graph denotes the true value of the measure.

and 200000, respectively. If the age at censoring is not lower than 85, life-table measures deviate slightly from their true values (see Table **??**). It is not surprising that discrepancy increases as the age at censoring x_C gets lower. However, even for x_C between 75 and 85, all five life-table measures are already distorted by 10-20%. While the statistical offices in many countries "close" their life tables at least at age 85, there is a number of countries in which the last open-age group starts at lower ages (Wilmoth et al., 2007). As a result, their mortality indicators can potentially be distorted if calculated by conventional life-table algebra. Note that the proportion of censored individuals is much more important than the age at censoring. In the simulation example an 85+ open-end interval corresponds to 20% censoring, while the 85+ age group in the Brazilian life tables for 2007 contains 34.16% of

male and 50.89% of female deaths (HLTD, 2015).

3.2 Sensitivity to Censoring in Non-Human Mortality

Experimental mortality data for non-human species are often characterized by heavy censoring, leaving sometimes only a small proportion of fully observed individuals. Human mortality data are typically subjected to type-I censoring (with a fixed age at censoring), while experimental data often exhibit random or deterministic censoring. Depending on the experimental setup, we can observe type-I, type-II (experiment ends when a fixed proportion of the organisms die, e.g., Dawidowicz et al., 2010; Pietrzak et al., 2015), or, more rarely, hybrid censoring (experiment ends when a fixed proportion of the organisms die or a given age is reached, see Balakrishnan and Kundu, 2013). Here we focus on the effect of type-II censoring on the mortality measures for a population of rats (Anisimov et al., 1989). The mortality pattern in this dataset, unlike the human one, is not captured by the ΓGM model: Lenart and Missov (2014) apply a goodness-of-fit test for the Gompertz distribution to verify the exponential increase in the hazard of death. The hazard of the Gompertz model is given by (1) for $c = \gamma = 0$. We calculate model-based mortality measures by performing parametric bootstrapping (1000 repetitions). Note that parameter estimation is carried out by maximizing a Gompertz likelihood as we deal with individual data (for aggregated data, as it is in the case of human mortality, we maximize a Poisson likelihood, see section 1 and eq. 2).

Figure 4 illustrates the distortions in rat mortality measures when type-II censoring is addressed in a life-table style. Depending on the proportion of censored individuals (from 0% to 70%), life expectancy and the modal age at death can be mismatched on average by up to 100 days, life disparity and entropy can be calculated as much as twice as low, while the Gini coefficient can be off by 10%.

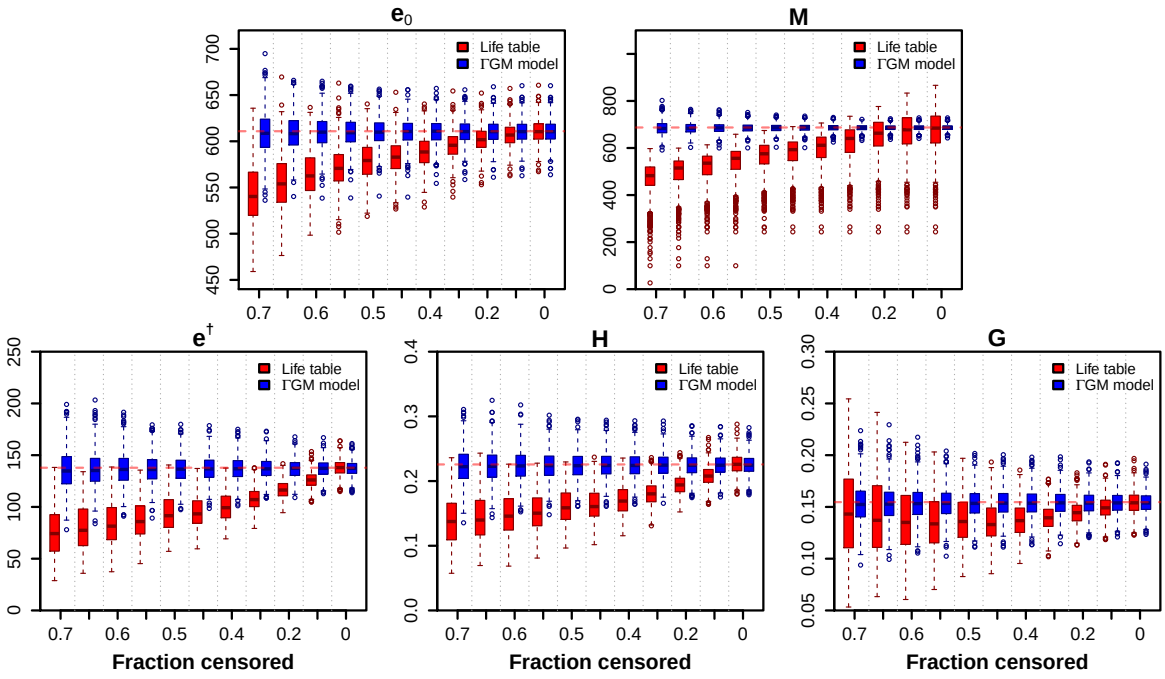

Figure 4: Life-table (red) vs ΓGM model-based (blue) mortality measures for a rat population of size 200 (1000 repetitions, simulation based on the estimates by Lenart and Missov (2014) for the dataset in Anisimov et al. (1989)). The red dashed line in each graph denotes the true value of the measure.

4 Discussion

Mortality measures calculated from conventional life tables, i.e., constructed on the basis of raw death counts, might be misleading because of the way right censoring is addressed: in the last open-end age group, life tables assume a constant hazard equal to the death rate in the beginning of the interval. The latter, constructed as the ratio of raw death counts over exposure, can be higher or lower than the "true" hazard. If it is lower, remaining life expectancy at any preceding age will be overestimated. If, on the contrary, the death rate at the starting age of the last interval exceeds the "true" force of mortality, then remaining life expectancy will be overestimated (underestimated) if area A is smaller (bigger) than area B

(see Figure 5)[3].

The Human Mortality Database (HMD, 2015) smooths mortality rates at the oldest ages. If statistical offices provide censored (at age x_C) data, age-specific mortality reconstruction from x_C onwards is performed by fitting a Kannisto model to the last 20 ages with available age-specific death counts and extrapolating the estimated model to subsequent ages (Wilmoth et al., 2007). If exact death counts are available for every single age, the HMD smooths the death counts after the first age x_T, at which the number of deaths is lower than 100, by fitting a Kannisto model from age x_T to age 110 (Wilmoth et al., 2007). The hazard of death at age x in a Kannisto model is given by

$$\mu_K(x) = \frac{ae^{b(x-x_0)}}{1+ae^{b(x-x_0)}}\,, \tag{8}$$

where x_0 is the starting age of analysis, while $\ln a$ and b represent the intercept and the slope, respectively, of the (assumed) $\text{logit}(\mu_K(x))$ linear increase. The Kannisto hazard has an S-shaped (logistic) pattern. Fitting a Kannisto or a gamma-Gompertz-Makeham

[3]If the death rate at the censoring age lies on or below the ΓGM curve, the area under the resulting hazard $\mu(x)$ will be less than the area under the ΓGM hazard (after the age at censoring, the latter increases while the former stays constant). As the survival function is defined in terms of the hazard as

$$s(x) = \exp\left\{-\int_0^x \mu(t)dt\right\},$$

$s(x)$ will be overestimated (in comparison to the ΓGM hazard). Consequently, all mortality measures that are calculated by integrating $s(x)$ or $s^2(x)$ (life expectancy, life disparity, entropy, and the Gini coefficient) will be overestimated.

If the death rate at the age at censoring lies above the ΓGM hazard then the sign of the bias depends on the difference between areas A and B: if A (the area we "gain") is larger than B (the area we "lose" as a result of censoring), $\mu(x)$ will be overestimated, whereas $s(x)$ and the four mortality measures will be underestimated, and vice versa.

The modal age at death M is not a function of $s(x)$. The bias in M is always directed downwards, i.e. M can only be underestimated, and this occurs if censoring takes place at an age that precedes the true M. In this case, in the absence of a model, we just choose (roughly) the age at censoring as the modal age at death.

model to adult human mortality (until age 110) is equivalent as the two models differ only asymptotically – $\mu_K(x)$ tends to 1, while the ΓGM allows more flexibility about the plateau: $\mu(x) \xrightarrow[x\to\infty]{} b/\gamma + c$. Human mortality measures calculated from the Kannisto-adjusted HMD life tables are almost identical to the ΓGM measures even if the two models are fitted over different age ranges (as in Figure 5). On the other hand, mortality measures calculated from life tables based on raw mortality data, e.g., for countries that are not present in the HMD and rely on standard life-table methodology without applying any mortality model, can be substantially distorted. This can also be the case for human mortality data by cause of death, for hunter-gatherer populations or non-human species, where the proportion of censored individuals can be high.

4.1 Mortality Measures for Countries with Lower Data Quality

The Human Life-Table Database (HLTD, 2015) contains life tables for countries with lower mortality-data quality (for detailed selection criteria to HMD and HLTD see Shkolnikov et al., 2007; Wilmoth et al., 2007). Reported official mortality measures for HLTD countries are based on these datasets. However, apart from other problems HLTD data may contain (Shkolnikov et al., 2007), the last age group in many life tables, for historical and contemporary populations, contains a substantial proportion of the population (see Table 1). This questions the adequacy of life-table algebra to calculate mortality measures for these countries.

Country	Last Year(s)	Cens. Age	% Censored (Male)	% Censored (Female)
Bangladesh	2007	85	16.18	16.59
Brazil	2008	80	34.16	51.47
Botswana	2006	80	*3.05*	17.28
Colombia	2005	80	46.74	61.83
Dominican Rep.	2002	80	34.76	46.64
India	1995-1999	70	45.83	53.19
Iran	2004	85	14.93	13.47
Korea Rep.	1995	85	14.14	33.69
Sri Lanka	2000-2002	92	*5.57*	11.70
Malta	2007	85	30.32	48.18
Mongolia	1996-2000	70	40.94	53.22
Panama	2000-2005	80	45.19	57.41

Table 1: A list of HLTD countries whose last available life table (after 1990) has at least 10% censored individuals for at least one of the genders (Data source: HLTD, 2015).

There are alternative ways of "closing" the life table, apart from the one described in (Preston et al., 2001). Horiuchi and Coale (1982) suggest a constant hazard in the last age group, as well, but adjusted for the growth rate of the this group (see Horiuchi and Coale, 1982: eq.7, p.322). Another option (used in HLTD) is to calculate life expectancy at the censoring age ω by a "table of correspondence between e_ω and e_0" (Shkolnikov et al., 2007)[4] and then adjust the (constant) death rate in the open-end interval. No matter how the constant hazard in the last age group is determined, aggregate mortality measures will be distorted, unless the level of mortality is chosen in such a way that area A equals area B (Figure 5). Another alternative (that does not assume constant mortality) is to redistribute the deaths in the open-end interval uniformly up to a fixed maximal age. In this case mortality measures are stable with respect to the choice of the age at censoring, but their accuracy is not

[4]For the age at censoring, we use here the notation in Shkolnikov et al. (2007) instead of x_C.

satisfactory, especially when the chosen maximal age increases. In the following section we demonstrate for 2007 Brazilian mortality data that all these methods for "closing" the life table can lead to erroneous conclusions about the magnitude of life expectancy.

4.2 Example: 2007 Gender-Specific Life Tables for Brazil

Contemporary Brazilian life tables are characterized by a large proportion of censored individuals. The 80+ open-end age group in 2007 contains over one third (34.16%) of male and over one half (50.89%) of female deaths. To estimate a ΓGM model, age-specific population (or death) counts must be available[5]. However, such data are missing for a number of HLTD populations which restricts the application of ΓGM-smoothing to the corresponding life tables.

Tables 2 and 3 present remaining life expectancy for 2007 Brazilian females and males, respectively, assuming for the last age interval (i) a constant hazard (according to Preston et al. (2001) and Shkolnikov et al. (2007)), (ii) a uniform distribution of deaths with maximal ages 100, 115, and 120, or (iii) a ΓGM model. When the age at censoring decreases (and the respective share of censored observation increases), remaining life expectancy e_{25} is stable in cases (ii) and (iii), while e_{25} according to (i) becomes unrealistically high. The ΓGM e_{25} increases by about 3 years when more than 2/3 of the population is censored, and e_{25} for uniformly distributed deaths in the last interval tends to be most realistic if the maximal age is 100. This is to be observed for life expectancy at birth, too (Tables 4 and 5). Note that life expectancy at birth reported by the Brazilian Institute of Geography and Statistics (IBGE) exceeds the ΓGM one by almost 3 years for females (76.44 vs 73.71) as around half of the deaths are censored, while for males, where about 1/3 of the individuals are censored, the two values are very close (68.82 vs 68.45).

[5]Brazilian age-specific population counts are publicly available at http://www.ibge.gov.br/english/estatistica/populacao/contagem2007/default.shtm

age	% censored	IBGE	HLTD	U100	U115	U120	ΓGM
80	50.89	53.83	51.88	54.16	58.12	59.43	51.04
75	63.75	63.30	63.30	54.22	59.17	60.80	53.74
70	73.60	73.99	73.99	53.90	59.60	61.51	54.47
65	80.73	91.69	91.69	53.11	59.38	61.46	57.39

Table 2: Remaining life expectancy at age 25 for 2007 Brazilian females calculated by assuming a constant hazard in the last age group (column 3: according to Preston et al. (2001); column 4: using HLTD tables of correspondence by Shkolnikov et al. (2007), a uniform distribution of deaths after the censoring age (column 5: to a maximal age of 100, column 6: to a maximal age of 115, column 7: to a maximal age of 120), and a ΓGM model (column 8).

age	% censored	IBGE	HLTD	U100	U115	U120	ΓGM
80	34.16	47.57	46.33	48.14	50.88	51.77	47.25
75	46.77	51.42	51.42	48.64	52.36	53.62	47.34
70	58.01	56.25	56.25	48.94	53.58	55.12	48.58
65	67.02	64.86	64.86	48.89	54.24	55.99	51.48

Table 3: Remaining life expectancy at age 25 for 2007 Brazilian males calculated by assuming a constant hazard in the last age group (column 3: according to Preston et al. (2001); column 4: using HLTD tables of correspondence by Shkolnikov et al. (2007)), a uniform distribution of deaths after the censoring age (column 5: to a maximal age of 100, column 6: to a maximal age of 115, column 7: to a maximal age of 120), and a ΓGM model (column 8).

age	% censored	IBGE	HLTD	U100	U115	U120	ΓGM
80	50.89	76.44	74.55	76.75	80.59	81.85	73.71
75	63.75	74.03	85.60	76.82	81.61	83.18	76.32
70	73.60	-	95.95	76.50	82.02	83.87	77.03
65	80.73	-	113.07	75.74	81.80	83.82	79.86

Table 4: Life expectancy at birth for 2007 Brazilian females calculated by assuming a constant hazard in the last age group (column 3: according to Preston et al. (2001); column 4: using HLTD tables of correspondence by Shkolnikov et al. (2007)), a uniform distribution of deaths after the censoring age (column 5: to a maximal age of 100, column 6: to a maximal age of 115, column 7: to a maximal age of 120), and a ΓGM model (column 8).

age	% censored	IBGE	HLTD	U100	U115	U120	ΓGM
80	34.16	68.82	67.65	69.36	71.94	72.77	68.45
75	46.77	72.44	67.30	69.83	73.33	74.51	68.53
70	58.01	77.00	-t	70.11	74.48	75.93	69.70
65	67.02	85.10	-	70.06	75.10	76.75	72.43

Table 5: Life expectancy at birth for 2007 Brazilian males calculated by assuming a constant hazard in the last age group (column 3: according to Preston et al. (2001); column 4: using HLTD tables of correspondence by Shkolnikov et al. (2007)), a uniform distribution of deaths after the censoring age (column 5: to a maximal age of 100, column 6: to a maximal age of 115, column 7: to a maximal age of 120), and a ΓGM model (column 8).

The adjustment by Horiuchi and Coale (1982) affects just remaining life expectancy at the censoring age. Tables 6 and 7 compare the latter at censoring ages 65, 70, 75, and 80 for 2007 Brazilian females and females, respectively. For four different growth rates of the last age group (0.5%, 1%, 2%, and 5%) the Horiuchi and Coale adjustment does not remove the bias associated with the constant-hazard assumption in the open-end interval. As a result, it is only the ΓGM that provides coherent remaining life expectancy values no matter how low the censoring age is or, what is equivalent, how large the proportion of censored deaths is.

age	% censored	IBGE	HLTD	HC				ΓGM
				0.005	0.01	0.02	0.05	
80	50.89	9.87	6.16	9.76	9.64	9.42	8.78	2.60
75	63.75	26.76	26.38	25.53	24.35	22.14	16.66	8.08
70	73.60	41.92	41.92	38.36	35.10	29.39	17.24	12.36
65	80.73	64.22	64.22	54.65	46.51	33.67	12.78	19.27

Table 6: Remaining life expectancy at the censoring age (column 1) for 2007 Brazilian females calculated by assuming a constant hazard in the last age group (column 3: according to Preston et al. (2001); column 4: using HLTD tables of correspondence by Shkolnikov et al. (2007); columns 5-8: using the adjustment by Horiuchi and Coale (1982) for growth rates of 0.005, 0.01, 0.02, and 0.05), and a ΓGM model (column 9).

age	% censored	IBGE	HLTD	HC				ΓGM
				0.005	0.01	0.02	0.05	
80	34.16	8.91	5.49	8.82	8.73	8.55	8.05	2.91
75	46.77	18.59	18.59	18.07	17.57	16.59	13.99	5.16
70	58.01	27.36	27.36	26.06	24.82	22.51	16.78	9.19
65	67.02	40.45	40.45	37.18	34.16	28.85	17.38	15.42

Table 7: Remaining life expectancy at the censoring age (column 1) for 2007 Brazilian males calculated by assuming a constant hazard in the last age group (column 3: according to Preston et al. (2001); column 4: using HLTD tables of correspondence by Shkolnikov et al. (2007); columns 5-8: using the adjustment by Horiuchi and Coale (1982) for growth rates of 0.005, 0.01, 0.02, and 0.05), and a ΓGM model (column 9).

5 Conclusion

The life-table distribution of deaths is characterized by a constant hazard for the last open-end age interval. This is not a typical approach for treating censoring in survival analysis.

Instead we propose fitting a parametric model (when a parametric model provides a satisfactory fit) by accounting for the censoring mechanism and using model-based mortality measures instead of their widely used life-table equivalents because the former are less sensitive to the age at censoring. Current life tables for many countries contain a large proportion of censored individuals, and we suggest calculating the corresponding mortality measures, especially life expectancy, by fitting a gamma-Gompertz-Makeham model because it captures well adult mortality, as well as addresses censoring accurately.

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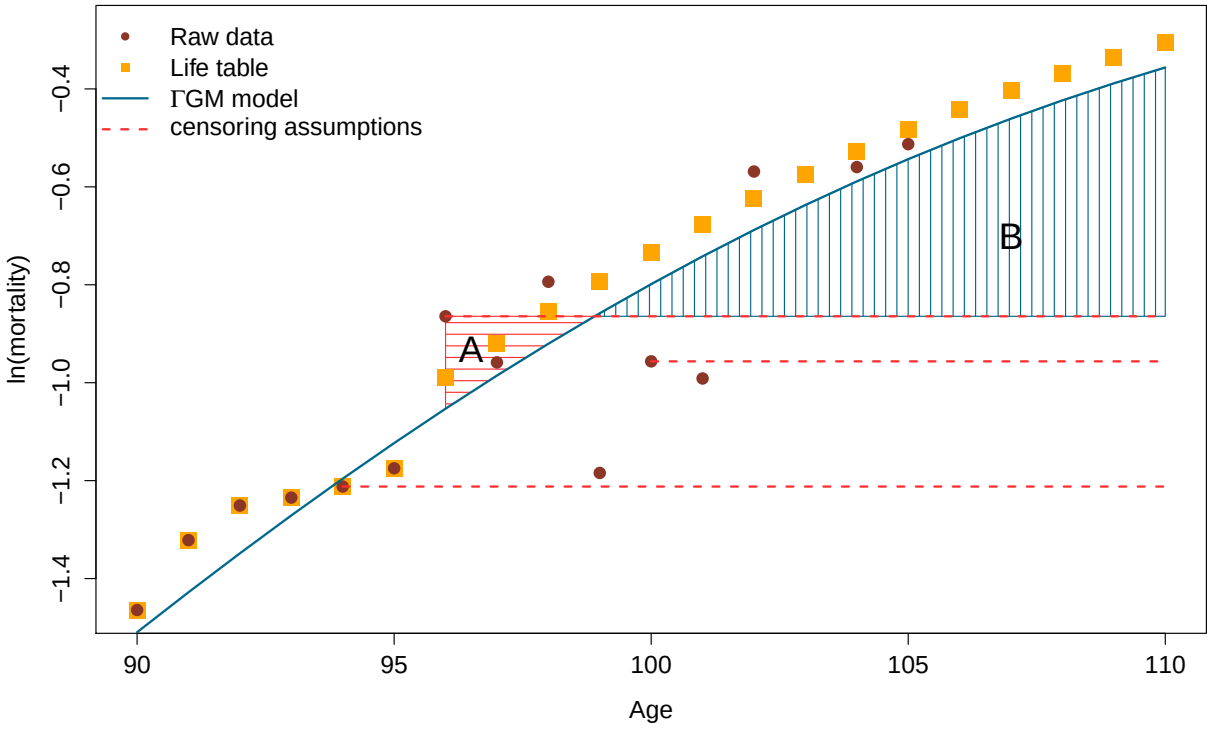

Figure 5: Log-mortality of Swedish males in 1970: raw data (circles), HMD data – raw data until age 95 and Kannisto-smoothed data from age 96 onwards (squares), and ΓGM fit (solid line). The three dashed horizontal lines (corresponding to censoring ages 94, 96, and 100) reflect the assumption that the hazard in the last open-age group in a life table is constant. If the observed death rate at the age at censoring overestimates the true force of mortality, remaining life expectancy will be overestimated/underestimated if the differences between areas A and B is negative/positive. If the observed death rate at the age at censoring underestimates the true force of mortality, remaining life expectancy will be overestimated.