



UDC 314.422:574.34

PACS 07.05.Tp, 89.65.Cd,

DOI: 10.22363/2658-4670-2023-31-4-359-374

EDN: FZWSUR

## Demographic indicators, models, and testing

Gregory A. Shilovsky<sup>1,2</sup>,  
Alexandr V. Seliverstov<sup>1</sup>, Oleg A. Zverkov<sup>1</sup>

<sup>1</sup> *Institute for Information Transmission Problems of the Russian Academy of Sciences  
(Kharkevich Institute),*

*19 Bolshoy Karetny per., bldg. 1, Moscow, 127051, Russian Federation*

<sup>2</sup> *Lomonosov Moscow State University,*

*1 Leninskie Gory, bldg. 12, Moscow, 119991, Russian Federation*

(received: October 17, 2023; revised: December 1, 2023; accepted: December 29, 2023)

**Abstract.** The use of simple demographic indicators to describe mortality dynamics can obscure important features of the survival curve, particularly during periods of rapid change, such as those caused by internal or external factors, and especially at the oldest or youngest ages. Therefore, instead of the generally accepted Gompertz method, other methods based on demographic indicators are often used. In human populations, chronic phenoptosis, in contrast to age-independent acute phenoptosis, is characterized by rectangularization of the survival curve and an accompanying increase in average life expectancy at birth, which can be attributed to advances in society and technology. Despite the simple geometric interpretation of the phenomenon of rectangularization of the survival curve, it is difficult to notice one, detecting changes in the optimal coefficients in the Gompertz–Makeham law due to high computational complexity and increased calculation errors. This is avoided by calculating demographic indicators such as the Keyfitz entropy, the Gini coefficient, and the coefficient of variation in lifespan. Our analysis of both theoretical models and real demographic data shows that with the same value of the Gini coefficient in the compared cohorts, a larger value of the Keyfitz entropy indicates a greater proportion of centenarians relative to average life expectancy. On the contrary, at the same value of the Keyfitz entropy, a larger value of the Gini coefficient corresponds to a relatively large mortality at a young age. We hypothesize that decreases in the Keyfitz entropy may be attributable to declines in background mortality, reflected in the Makeham term, or to reductions in mortality at lower ages, corresponding to modifications in another coefficient of the Gompertz law. By incorporating dynamic shifts in age into survival analyses, we can deepen our comprehension of mortality patterns and aging mechanisms, ultimately contributing to the development of more reliable methods for evaluating the efficacy of anti-aging and geroprotective interventions used in gerontology.

**Key words and phrases:** lifespan, demographic indicator, Keyfitz entropy, Gini coefficient, coefficient of variation, phenoptosis, aging, Gompertz law

© Shilovsky G. A., Seliverstov A. V., Zverkov O. A., 2023



This work is licensed under a Creative Commons Attribution 4.0 International License

<https://creativecommons.org/licenses/by-nc/4.0/legalcode>

## 1. Introduction

One of the fundamental tasks of biodemography is the creation and computational verification of probabilistic models to explain the difference in lifespan and response to geroprotectors in different species [1–6]. To optimize the model, parallel calculation of demographic indicators for many parameter values is possible. As a result of comparing the calculated values with known demographic indicators, one can find not only the optimal values of the model parameters, but also the sensitivity of the values of demographic indicators to changes in these parameters. Of practical importance may be the reconstruction of changes in life expectancy in evolution, as well as the prediction of the effectiveness of anti-aging drugs and geroprotectors [7, 8].

A study of mortality fluctuations allows one to assess both the quality and comparability of mortality statistics. Other related problems are comparison of changes in the life expectancy at the national level with the changes in individual regions, identification of regions with high or low values of the life expectancy, estimation of the contribution of individual age groups and causes of death to the regional differences in the life expectancy, and determination of characteristics of mortality and causes of death for individual groups or different regions [9, 10].

The COVID-19 pandemic has revealed significant gaps in the coverage and quality of existing international and national statistical monitoring systems. Ensuring prompt availability of accurate and comparable data in each country for an adequate response to unexpected epidemiological threats is a very challenging task. The interest in studying associations between mortality oscillations and fluctuation of economic conditions has been rekindled recently because mortality is characterized with periodic oscillations [11, 12].

In general, modeling can be used for simulation of future behavior of demographic processes based on the available data in order to reveal main and additional rhythms. In particular, construction of wavelet spectrograms provides a possibility to calculate the matrix for synchronization value and synchronicity (simultaneous occurrence), syn-phase behavior (phase coincidence), and coherence (interconnection) of the investigated parameters of studied biorhythms. Statistical significance of the rhythms is evaluated through multiple random permutations of levels of the initial temporal series [13]. Decomposition into seasons and trends using the Loess approach (STL) is used for analysis of seasonal fluctuations of mortality risk, medical care expenditure, and even hospitalization levels. The STL method expands longitudinal data into the long-term trend, seasonal variations, and remaining variations not associated with the long-term trend or seasonal variations [14]. The long-term trend in the STL method reflects a number of possible external factors that change gradually with time [15].

During the last 150 years, the decrease in the seasonal fluctuations of mortality has facilitated an increase in the life expectancy [16]. New methods based on the analysis of time-dependent variability, trends, and interactions of numerous physiological and laboratory parameters, for which machine learning and artificial intelligence could be applied, will help to establish whether the dynamic regularities observed in large epidemiological studies have significance for the risk profile of an individual patient [17]. From the gerontological point of view, studying mortality fluctuations allows to switch

from investigating the effects of biorhythms on the development of acute and chronic phenoptosis to the elucidating the patterns of determined mortality rhythms [10].

There are also developed formal demographic measures to examine the complex relationships between the total life expectancy of two peers at birth, the proportion of their life that they can expect to live, and longevity [18]. A modification of the Gini coefficient is the Drewnovsky index, which is a measure of equality [19]. So, Aburto *et al.* simulated scenarios for improving mortality in a Gompertz model and showed that the new index can serve as an indicator of the shape of the mortality structure. The proposed method allows us to identify trends in lifespan changes in both humans and other species.

Our aim is to compare measures of shape of the survival curve. These measures should be dimensionless. They depend on the shape of the survival curve only.

## 2. Preliminaries

### 2.1. The Gompertz law

The Gompertz law is a probabilistic model of mortality that describes well the mortality of people aged 20 to 65 or up to 80 years. This law was proposed in the pioneering work of B. Gompertz and was originally used to assess risks in life insurance [20]. Problems with assessing the aging process as an increase in the probability of death (the number of deaths in one age interval) have existed for a long time [20, 21].

If the probability of death of an organism depended entirely on the level of disruption that increases with age, then the mortality rate of multicellular organisms should increase with age, regardless of the position of the species on the evolutionary tree. However, large differences in mortality dynamics across species have been found (increasing, constant, decreasing, convex, and concave mortality trajectories in both long-lived and short-lived species) [4, 22–25]. Possible mechanisms for the emergence of such diversity in evolution are actively discussed [7, 26, 27]. Despite the discussion of amendments to this law [28–30], its main idea has remained unchanged for almost two hundred years: the law determines the dependence of the conditional probability density of death on age.

Let us denote by  $\mu(a)$  the conditional probability density for each individual to die at age  $a$ , provided that he survived. The value of  $\mu(a)$  is called the strength of mortality. We will assume that the function  $\mu(a)$  is piecewise smooth. According to the Gompertz–Makeham law, starting from some age  $a_{\min}$  and up to age  $a_{\max}$ , this function is of the type  $\mu(a) = \alpha + \beta \exp(\gamma a)$ , where  $\alpha$ ,  $\beta$ , and  $\gamma$  are some coefficients that do not depend on the age  $a$ , but may depend on external conditions. Here  $\alpha$  is the probability density of an accidental death regardless of age. Originally  $\alpha = 0$  was assumed in the Gompertz law. For people  $a_{\min} \approx 30$  years and  $a_{\max} \approx 80$  years.

In theoretical calculations, we pass to a continuous change in age. In practice, for humans, the unit of time is usually one year, less often five years, and for species with a short lifespan such as nematodes or fruit flies, it is one day. The product  $\mu(a)\Delta a$  is only approximately equal to the probability of dying between  $a$  and  $a + \Delta a$ .

The probability of surviving to age  $a$  is equal to the survival function

$$\ell(a) = \exp\left(-\int_0^a \mu(\tau)d\tau\right).$$

Of course,  $\ell(0) = 1$  because only those born are taken into account.

## 2.2. The Makeham term

Accounting for accidental death, the conditional probability density of which does not depend on age, leads to an additional term called the Makeham term. The new conditional death probability density is  $\mu(a) = \exp(-s)u + \exp(ra - s)$ . Such an amendment to the Gompertz law was proposed by W.M. Makeham [29]. Usually the value of  $u$  is nonnegative, but negative values  $u > -1$  can also be considered, corresponding to an accidental escape from death. Such an amendment corresponds to multiplying the original survival function  $\ell(a)$  by the factor  $\exp(-\exp(-s)ua)$ .

For some large mammals such as the lion *Panthera leo*, the European roe deer *Capreolus capreolus*, the red deer *Cervus elaphus*, the chamois *Rupicapra rupicapra*, the sheep *Ovis aries*, and the yellow-bellied marmot *Marmota flaviventris* as well as birds the Bali myna *Leucopsar rothschildi* and the sparrowhawk *Accipiter nisus*, the conditional probability density of death has a non-zero minimum [4], which suggests that the  $u$  correction is nonzero. It can be concluded that such dynamics of mortality is typical for large mammals and some birds either having no enemies in nature like a hawk or kept in zoos.

## 2.3. The Keyfitz entropy $H$

Let us consider a demographic indicator called the Keyfitz entropy. This concept was introduced by Canadian demographer Nathan Keyfitz [31]. The Keyfitz entropy characterizes the deviation of the survival curve from a non-increasing step function that is equal to either 0 or 1.

$$\ell_{\text{rect}}(a) = \begin{cases} 1, & a \leq e_o, \\ 0, & a > e_o. \end{cases}$$

Let us denote the life disparity by

$$e^\dagger = -\int_0^\infty \ell(\tau) \ln \ell(\tau) d\tau.$$

The life expectancy at birth is denoted by

$$e_o = \int_0^{\infty} \ell(\tau) d\tau.$$

The Keyfitz entropy is equal to

$$H = \frac{e^\dagger}{e_o}.$$

The Keyfitz entropy is close to zero when almost everyone dies at the same age, no matter what that age is. In other words, rectangularization of the survival curve leads to the value of  $e^\dagger$  vanishing. On the other hand, the Keyfitz entropy decreases even more as the life expectancy  $e_o$  increases.

Surprisingly, there is such an age threshold that preventing death before reaching this threshold leads to a decrease in the Keyfitz entropy, and after reaching the age threshold, to its increase [32]. The development of society and scientific and technological progress leads to an increase in life expectancy over time [33]. But the lifespan of people with accurately confirmed age rarely exceeds 116 years [28]. Unfortunately, reports of centenarians who lived for more than 120 years are not confirmed or were refuted upon further verification.

Another observation is the relationship between life expectancy and the Keyfitz entropy [34, 35]. An increase in the standard of living of the population leads to a simultaneous decrease in  $e^\dagger$  and an increase in  $e_o$ , which leads to a decrease in the Keyfitz entropy. At the same time, an increase in  $e_o$  looks quite natural, while a decrease in  $e^\dagger$  *a priori* is less obvious, but is in good agreement with the phenoptosis hypothesis [10, 36].

Continuing the former research [35, 37–39], we compare some demographic indicators, including the Keyfitz entropy, calculated for different aging models.

#### 2.4. The Gini coefficient $G$

Another demographic indicator is the Gini coefficient

$$G = 1 - \frac{1}{e_o} \int_0^{\infty} \ell^2(\tau) d\tau.$$

It also vanishes on a non-increasing step function that takes only two values 0 or 1. This indicator was proposed in 1912 by the demographer Corrado Gini [40]. It is used in demography by other authors too [41–43].

#### 2.5. The coefficient of variation $CV_{LS}$

The coefficient of lifespan variation  $CV_{LS}$  is also used in demography [10, 37–39, 44]. The formula for calculating the coefficient of variation explicitly includes the first derivative of the survival function, which is equal to the

product of the survival function and the conditional death probability density  $\mu(a)$ . This derivative is usually called the distribution of deaths.

## 2.6. Integrals

We use the SymPy library to calculate the integrals in the considered examples. In fact, only some integrals are expressed in terms of elementary functions. Therefore, not only symbolic computing, but also numerical methods are used.

## 3. Results

### 3.1. The Keyfitz entropy and Gini coefficient in comparison

Both demographic indicators  $H$  and  $G$  are expressed in terms of the survival function by similar formulas. Both Keyfitz entropy and Gini coefficient measure the difference between the survival function and a non-increasing step function. However, these indicators differ significantly in their stability under changes in the survival function [39].

Let us make a transformation, taking into account the expansion of the natural logarithm in a series

$$\ln x = x - 1 - \frac{(x - 1)^2}{2} + \frac{(x - 1)^3}{3} - \frac{(x - 1)^4}{4} + \dots$$

So,

$$e^\dagger = e_o - e_n + \int_0^\infty \ell(\tau)(\ell(\tau) - 1)^2 \left( \frac{1}{2} - \frac{1}{3}(\ell(\tau) - 1) + \dots \right) d\tau.$$

The difference is equal to

$$H - G = \frac{1}{e_o} \int_0^\infty \ell(\tau)(\ell(\tau) - 1)^2 \left( \frac{1}{2} - \frac{1}{3}(\ell(\tau) - 1) + \frac{1}{4}(\ell(\tau) - 1)^2 - \dots \right) d\tau.$$

For small ages, the integrand is small, since the difference  $\ell - 1$  is close to zero. For sufficiently large ages, it is also small, since  $\ell$  does not increase and must tend to zero for the integral  $e_o$  to converge. However, here the integrand tends to zero only at about the same rate as the function  $\ell$  itself.

When the survival function  $\ell(a)$  is close enough to a non-increasing step function  $\ell_{rect}(a)$  the difference  $H - G$  is mainly determined by the behavior of the survival function near  $e_o$  and at large values of age. However, it depends little on the properties of this function at small ages.

For  $\ell_{rect}(a)$ , both Keyfitz entropy and Gini coefficient vanish. But unlike the Gini coefficient, the Keyfitz entropy can be arbitrarily large on survival functions close to  $\ell_{rect}(a)$ . In fact, the graph of the function  $x \ln(x)$  has a vertical tangent at  $x = 0$ . So, the first derivative of this function  $(x \ln(x))' = \ln(x) + 1$  tends to negative infinity  $-\infty$  in the limit  $x \rightarrow +0$ .

There is a sequence of monotonically nonincreasing functions  $\ell_k$  such that the sequence  $\ell_k$  converges to the limit function  $\ell$ , but the sequence of values of the Keyfitz entropy  $H[\ell_k]$  does not converge to the value  $H[\ell]$ . By convergence we mean pointwise convergence almost everywhere, i. e., except for the set of points of measure zero, the value of the function  $\ell$  at a point is equal to the limit of the values of the functions  $\ell_k$  at the same point. Informally, such survival functions  $\ell_k$  correspond to a situation when almost everyone dies early at the same age (hence,  $e_o$  is small), but a tiny part of long-lived individuals, tending to zero with increasing index  $k$ , live extremely long. By choosing the ratio between the proportion of centenarians and the maximum life expectancy, one can achieve an increase in the Keyfitz entropy.

**Example 1.** For sufficiently large indices  $k > \ln(e_o)$ , let the value of the survival function be

$$\ell_k(a) = \begin{cases} 1, & 0 \leq a \leq e_o - 1, \\ \exp(-k), & e_o - 1 < a < e_o - 1 + \exp(k), \\ 0, & a \geq e_o - 1 + \exp(k). \end{cases}$$

Then for large indices  $k$  the life expectancy is equal to the previously chosen number  $e_o$  and  $e^\dagger = k$ . Therefore, the Keyfitz entropy  $H[\ell_k]k/e_o$  tends to infinity as  $k \rightarrow \infty$ . However, in the limit at  $k \rightarrow \infty$ , the survival function  $\ell_k$  approaches  $\ell_{rect}(a)$ , i. e., there is rectangularization of the survival curve. The limit survival function  $\ell$  is equal to one for ages up to  $e_o$  and zero for larger ages. Obviously, at every point except  $e_o$  the function  $\ell \ln \ell$  vanishes. Hence, the Keyfitz entropy  $H[\ell]$  vanishes.

In the considered example, when passing to the limit, the expected lifespan changes abruptly. However, by increasing the absolute value, its relative change can be made arbitrarily small. On the contrary, the sequence of Gini coefficients  $G[\ell_k]$  tends to zero, i. e., it converges to the Gini coefficient of the limit function.

### 3.2. Generalized Gini coefficients

There is an obvious generalization of the Gini coefficient. For a number  $p > 1$ , let us define the generalized Gini coefficient of order  $p$

$$G_p = 1 - \frac{1}{e_o} \int_0^\infty \ell^p(\tau) d\tau.$$

Of course,  $G_2$  is the same as the Gini coefficient  $G$ .

For any value of  $p > 1$ ,  $G_p$  vanishes on a nonincreasing step function of the type  $\ell_{rect}(a)$  that takes only two values 0 or 1. To study the properties of the tail of the distribution, i. e., the presence of centenarians,  $1 < p < 2$  are of interest, for example,  $p = 3/2$ . For the aging model  $\mu(a) = \exp(a - 1 - s)$ , where  $s$  is a parameter, both indices  $G_{3/2}$  and  $G$  correlate with each other. On the other hand, the ratio of these two indicators differs from a constant.

### 3.3. Demographic indicators calculated by summation

In practice, integrals are replaced by finite sums since real lifespan is bounded and age is measured discretely. With a sufficiently large sample, the Gini coefficient is resistant to small errors, in particular, associated with the inevitable difficulties in determining the age. Refining the step  $w$  of age change leads to sharp changes in the first derivative of the survival function, which is included in the formula for calculating the coefficient of variation of life expectancy. However, both Keyfitz entropy  $H$  and Gini coefficient  $G$  depend only on the survival function itself. Therefore, step refinement does not spoil, but only refines the calculation of  $H$  and  $G$ . We consider the calculation of both Keyfitz entropy and Gini coefficient for the conditional death probability density  $\mu(a) = \exp(a)$ , which corresponds to the Gompertz law, for different values of the step  $w$ . For small step values, the result differs little from the result based on integration. The exact values are equal to  $H = 0.68$  and  $G = 0.39$ , respectively. The summation was carried out up to the age of 100 with an average life expectancy  $e_o = 0.60$  (refer to table 1).

Table 1

Both Keyfitz entropy  $H$  and Gini coefficient  $G$  depend on the step length  $w$

Step $w$	The Keyfitz entropy $H$	The Gini coefficient $G$
0.001	0.68	0.39
0.01	0.67	0.39
0.1	0.62	0.36
1	0.27	0.13

As the step increases, the values of demographic indicators calculated by summation decrease. Such a decrease can be confused with the approximation of the survival curve to a rectangularized one, but this is only the result of a calculation error.

### 3.4. Example: regime-change aging

Let us consider a one-parameter family of functions  $\mu$  that do not explicitly depend on time, where the parameter  $p$  is positive

$$\mu(a) = \begin{cases} \exp(a - 1 - p), & a \leq 1, \\ \exp(-p), & a \geq 1. \end{cases}$$

At small ages, aging occurs according to Gompertz; starting from age 1 (some threshold), aging does not depend on age. Here, the unit of age is conditional, and the model itself is not based on real demographic data. The survival function is

$$\ell(a) = \begin{cases} \exp(\exp(-1 - p) - \exp(a - 1 - p)), & a \leq 1, \\ \exp(\exp(-1 - p) - a \exp(-p)), & a \geq 1. \end{cases}$$

Calculations show that as the parameter  $p$  increases, the Keyfitz entropy  $H$ , the coefficient of variation  $CV_{LS}$ , and the Gini coefficient  $G$  increase as well (refer to table 2).

Table 2

Regime-change aging

$p$	$e_o$	$H$	$CV_{LS}$	$G$
0.1	1.421518092	0.8094109048	0.8113722122	0.4171815872
0.2	1.542122578	0.8193004266	0.8208801324	0.4205427489
0.3	1.674623382	0.8292751898	0.8305383031	0.4240996359
0.4	1.820323302	0.8392299859	0.8402332055	0.4277956999
0.5	1.980660797	0.8490722655	0.8498641749	0.4315779574
0.6	2.157223890	0.8587224607	0.8593440475	0.4353979373
0.7	2.351765625	0.8681138602	0.8685992389	0.4392123237
0.8	2.566221236	0.8771921308	0.8775693578	0.4429832962
0.9	2.802727165	0.8859145778	0.8862064852	0.4466786320
1.0	3.063642166	0.8942492046	0.8944741982	0.4502716046
1.1	3.351570657	0.9021736551	0.9023464481	0.4537407212
1.2	3.669388575	0.9096740879	0.9098063557	0.4570693552
1.3	4.020271956	0.9167440522	0.9168449959	0.4602452907
1.4	4.407728588	0.9233833701	0.9234601972	0.4632602424
1.5	4.835632967	0.9295970938	0.9296554211	0.4661093436
1.6	5.308264979	0.9353945249	0.9354387066	0.4687906562
1.7	5.830352620	0.9407883361	0.9408217340	0.4713046910
1.8	6.407119251	0.9457937809	0.9458189796	0.4736539663
1.9	7.044335810	0.9504280005	0.9504469809	0.4758426085
2.0	7.748378494	0.9547094368	0.9547237110	0.4778759915

### 3.5. Slow aging models

Let us consider models of asymptotically slower aging than the Gompertz law provides. It is natural to call such models of aging sub-Gompertzian. The simplest model corresponds to the age-independent positive constant  $\mu(a) = m$ . Such a model of aging is realized, for example, in the hydra *Hydra magnipapillata*, the abalone mollusc *Haliotis rufescens*, and the hermit

crab *Pagurus longicarpus* [4]. In this case  $\ell(a) = \exp(-ma)$ . Life expectancy  $e_o = 1/m$ . The Keyfitz entropy is equal to  $H = 1$  for any value of the constant  $m > 0$ . The Gini coefficient is also a constant  $G = 0.5$ . The coefficient of variation equals  $CV_{LS} = 1$ .

The linear model  $\mu(a) = a$  is approximately realized in both nematode *Caenorhabditis elegans* and human louse *Pediculus humanus* [4]. The survival function is  $\ell(a) = \exp(-a^2/2)$ . The Keyfitz entropy equals  $H = 0.5$ . The Gini coefficient equals  $G = 0.29$ . The coefficient of variation equals  $CV_{LS} = 0.53$ .

For  $\mu(a) = a^d$ , the survival function is  $\ell(a) = \exp(-a^d/d)$ . The Keyfitz entropy equals  $H = 1/(d + 1)$ . It tends to zero as the degree  $d$  increases.

### 3.6. Models with delayed mortality

Let us consider models with the function  $\mu(a)$  equal to zero at the age up to some value  $b$ , starting from which this function grows. Such a model with delayed mortality is known as the Teissier model [45]. It corresponds to the guppy *Poecilia reticulata* [4]. On the other hand, such a model with  $b = \exp(-s)$  and  $m(a) = \exp(ra)$  can serve as a rough approximation to the Gompertz law, therefore, it allows making estimates of demographic indicators for a typical case using simplified calculation methods.

The survival function  $\ell(a)$  generates a family of functions  $\ell_b(a)$  equal to one for  $a < b$  and equal to  $\ell(a - b)$  for  $a > b$ . Depending on the magnitude of the shift  $b$ , the life expectancy increases  $e_o(b) = b + e_o$ . The Keyfitz entropy decreases and is equal to

$$H(b) = H \frac{e_o}{b + e_o}.$$

Similarly, the Gini coefficient decreases by the same factor

$$G(b) = G \frac{e_o}{b + e_o}.$$

In this case, the indicators depend not only on  $b$ , but also on  $e_o$ .

## 4. Conclusion

We have no reason to refuse the application of the Gompertz–Makeham law in vertebrates in a wide range of ages, excluding periods of high infant mortality and very advanced ages. On the other hand, for some invertebrates as well as for plants, the applicability of this model does not seem to be substantiated [30]. We conclude that, despite the fundamental applicability of the Gompertz–Makeham law under the indicated restrictions, the use of the demographic indicators considered in the article makes it possible to observe new patterns, and also opens up wide opportunities for their visualization. We considered several sub-Gompertzian models describing the aging of nematodes and insects. Within the framework of the sub-Gompertzian model of aging, age-dependent phenoptosis in the nematode *Caenorhabditis elegans* [36] is quantified as a rectangularization of the survival curve compared to this curve in the hydra *Hydra magnipapillata*, the abalone mollusk *Haliotis rufescens*, and the hermit crab *Pagurus longicarpus*. In turn, rectangularization of the

survival curve is assessed by demographic indicators ( $H$ ,  $G$ , and  $CV_{LS}$ ), each of which is significantly lower for the nematode than for hydra, abalone, and hermit crab. On the other hand, rectangularization of the survival curve, which increases with the development of scientific and technological progress, demonstrated through a decrease in the Keyfitz entropy [34], with a simultaneous increase in the average life expectancy in humans, is also in good agreement with the hypothesis of age-dependent chronic phenoptosis in humans.

In general, calculations on aging models demonstrate the effectiveness of using the Keyfitz entropy as well as the Gini coefficient as important demographic indicators, the change in which in the course of evolution is consistent with known data, in particular, for nematodes, for which the sub-Gompertzian aging model is applicable, compared with vertebrates, for which the Gompertz–Makeham law applies.

## Acknowledgments

Computations were performed at the Joint Supercomputer Center of the Russian Academy of Sciences (JSCC RAS).

## References

- [1] D. Avraam, J. P. de Magalhaes, and B. Vasiev, “A mathematical model of mortality dynamics across the lifespan combining heterogeneity and stochastic effects,” *Experimental Gerontology*, vol. 48, no. 8, pp. 801–811, 2013. DOI: 10.1016/j.exger.2013.05.054.
- [2] F. Colchero and B. Y. Kiyakoglu, “Beyond the proportional frailty model: Bayesian estimation of individual heterogeneity on mortality parameters,” *Biometrical Journal*, vol. 62, no. 1, pp. 124–135, 2020. DOI: 10.1002/bimj.201800280.
- [3] N. Hartemink, T. I. Missov, and H. Caswell, “Stochasticity, heterogeneity, and variance in longevity in human populations,” *Theoretical Population Biology*, vol. 114, pp. 107–116, 2017. DOI: 10.1016/j.tpb.2017.01.001.
- [4] O. R. Jones, A. Scheuerlein, R. Salguero-Gómez, et al., “Diversity of ageing across the tree of life,” *Nature*, vol. 505, no. 7482, pp. 169–173, 2014. DOI: 10.1038/nature12789.
- [5] A. Moskalev, Z. Guvatova, et al., “Targeting aging mechanisms: pharmacological perspectives,” *Trends in Endocrinology & Metabolism*, vol. 33, no. 4, pp. 266–280, 2022. DOI: 10.1016/j.tem.2022.01.007.
- [6] L. I. Rubanov, A. G. Zaraisky, G. A. Shilovsky, A. V. Seliverstov, O. A. Zverkov, and V. A. Lyubetsky, “Screening for mouse genes lost in mammals with long lifespans,” *BioData Mining*, vol. 12, p. 20, 2019. DOI: 10.1186/s13040-019-0208-x.
- [7] M. I. Mosevitsky, “Progerin and its role in accelerated and natural aging,” *Molecular Biology*, vol. 56, no. 2, pp. 125–146, 2022. DOI: 10.1134/S0026893322020091.

- [8] G. A. Shilovsky, “Variability of mortality: Additional information on mortality and morbidity curves under normal and pathological conditions,” *Biochemistry (Moscow)*, vol. 87, no. 3, pp. 294–299, 2022. DOI: 10.1134/S0006297922030087.
- [9] D. A. Jdanov, A. A. Galarza, V. M. Shkolnikov, *et al.*, “The short-term mortality fluctuation data series, monitoring mortality shocks across time and space,” *Scientific Data*, vol. 8, no. 1, p. 235, 2021. DOI: 10.1038/s41597-021-01019-1.
- [10] V. P. Skulachev, G. A. Shilovsky, *et al.*, “Perspectives of Homo sapiens lifespan extension: focus on external or internal resources?” *Aging*, vol. 12, no. 6, pp. 5566–5584, 2020. DOI: 10.18632/aging.102981.
- [11] R. Edwards, “Who is hurt by procyclical mortality?” *Social Science & Medicine*, vol. 67, no. 12, pp. 2051–2058, 2008. DOI: 10.1016/j.socscimed.2008.09.032.
- [12] I. I. Vasilyeva, A. V. Demidova, O. V. Druzhinina, and O. N. Masina, “Construction, stochastization and computer study of dynamic population models “two competitors — two migration areas,”” *Discrete and Continuous Models and Applied Computational Science*, vol. 31, no. 1, pp. 27–45, 2023. DOI: 10.22363/2658-4670-2023-31-1-27-45.
- [13] D. Hainaut and M. Denuit, “Wavelet-based feature extraction for mortality projection,” *ASTIN Bulletin*, vol. 50, no. 3, pp. 675–707, 2020. DOI: 10.1017/asb.2020.18.
- [14] S. Ebmeier, D. Thayabaran, I. Braithwaite, C. Bénamara, M. Weatherall, and R. Beasley, “Trends in international asthma mortality: analysis of data from the WHO Mortality Database from 46 countries (1993–2012),” *The Lancet*, vol. 390, no. 10098, pp. 935–945, 2017. DOI: 10.1016/S0140-6736(17)31448-4.
- [15] H. J. A. Rolden, J. H. T. Rohling, *et al.*, “Seasonal variation in mortality, medical care expenditure and institutionalization in older people: Evidence from a Dutch cohort of older health insurance clients,” *PLoS ONE*, vol. 10, no. 11, e0143154, 2015. DOI: 10.1371/journal.pone.0143154.
- [16] A. Ledberg, “A large decrease in the magnitude of seasonal fluctuations in mortality among elderly explains part of the increase in longevity in Sweden during 20th century,” *BMC Public Health*, vol. 20, p. 1674, 2020. DOI: 10.1186/s12889-020-09749-4.
- [17] J. P. Kooman, L. A. Usvyat, M. J. E. Dekker, *et al.*, “Cycles, arrows and turbulence: Time patterns in renal disease, a path from epidemiology to personalized medicine?” *Blood Purification*, vol. 47, no. 1-3, pp. 171–184, 2019. DOI: 10.1159/000494827.
- [18] J. A. Barthold Jones, A. Lenart, and A. Baudisch, “Complexity of the relationship between life expectancy and overlap of lifespans,” *PLoS One*, vol. 13, no. 7, e0197985, 2018. DOI: 10.1371/journal.pone.0197985.

- [19] J. M. Aburto, U. Basellini, A. Baudisch, and F. Villavicencio, “Drewnowski’s index to measure lifespan variation: Revisiting the Gini coefficient of the life table,” *Theoretical Population Biology*, vol. 148, pp. 1–10, 2022. DOI: 10.1016/j.tpb.2022.08.003.
- [20] B. Gompertz, “XXIV. On the nature of the function expressive of the law of human mortality, and on a new mode of determining the value of life contingencies,” *Philosophical Transactions of the Royal Society of London*, vol. 115, pp. 513–583, 1825. DOI: 10.1098/rstl.1825.0026.
- [21] E. S. Deevey, “Life tables for natural populations of animals,” *The Quarterly Review of Biology*, vol. 22, no. 4, pp. 283–314, 1947. DOI: 10.1086/395888.
- [22] A. V. Khalyavkin, “Influence of environment on the mortality pattern of potentially non-senescent organisms. General approach and comparison with real populations,” *Advances in Gerontology*, vol. 7, pp. 46–49, 2001.
- [23] S. V. Myl’nikov, “Towards the estimation of survival curves parameters and geroprotectors classification,” *Advances in gerontology*, vol. 24, no. 4, pp. 563–569, 2011.
- [24] R. E. Ricklefs, “Life-history connections to rates of aging in terrestrial vertebrates,” *Proceedings of the National Academy of Sciences*, vol. 107, no. 22, pp. 10 314–10 319, 2010. DOI: 10.1073/pnas.1005862107.
- [25] J. W. Vaupel, J. R. Carey, K. Christensen, et al., “Biodemographic trajectories of longevity,” *Science*, vol. 280, no. 5365, pp. 855–860, 1998. DOI: 10.1126/science.280.5365.855.
- [26] A. V. Markov, “Can kin selection facilitate the evolution of the genetic program of senescence?” *Biochemistry (Moscow)*, vol. 77, no. 7, pp. 733–741, 2012. DOI: 10.1134/S0006297912070061.
- [27] M. Skulachev, F. Severin, and V. Skulachev, “Aging as an evolvability-increasing program which can be switched off by organism to mobilize additional resources for survival,” *Current Aging Science*, vol. 8, no. 1, pp. 95–109, 2015. DOI: 10.2174/1874609808666150422122401.
- [28] N. S. Gavrilova and L. A. Gavrilov, “Are we approaching a biological limit to human longevity?” *The Journals of Gerontology: Series A*, vol. 75, no. 6, pp. 1061–1067, 2020. DOI: 10.1093/gerona/glz164.
- [29] W. M. Makeham, “On the law of mortality and the construction of annuity tables,” *The Assurance Magazine, and Journal of the Institute of Actuaries*, vol. 8, no. 6, pp. 301–310, 1860.
- [30] G. A. Shilovsky, T. S. Putyatina, A. V. Markov, and V. P. Skulachev, “Contribution of quantitative methods of estimating mortality dynamics to explaining mechanisms of aging,” *Biochemistry (Moscow)*, vol. 80, no. 12, pp. 1547–1559, 2015. DOI: 10.1134/S0006297915120020.
- [31] N. Keyfitz, “What difference would it make if cancer were eradicated? An examination of the Taeuber paradox,” *Demography*, vol. 14, no. 4, pp. 411–418, 1977. DOI: 10.2307/2060587.

- [32] Z. Zhang and J. Vaupel, “The age separating early deaths from late deaths,” *Demographic Research*, vol. 20, pp. 721–730, 2009. DOI: 10.4054/DemRes.2009.20.29.
- [33] J. Oeppen and J. W. Vaupel, “Broken limits to life expectancy,” *Science*, vol. 296, no. 5570, pp. 1029–1031, 2002. DOI: 10.1126/science.1069675.
- [34] F. Colchero, R. Rau, O. R. Jones, *et al.*, “The emergence of longevous populations,” *Proceedings of the National Academy of Sciences*, vol. 113, no. 48, pp. 7681–7690, 2016. DOI: 10.1073/pnas.1612191113.
- [35] L. Németh, “Life expectancy versus lifespan inequality: A smudge or a clear relationship?” *PLOS ONE*, vol. 12, no. 9, e0185702, 2017. DOI: 10.1371/journal.pone.0185702.
- [36] E. R. Galimov, J. N. Lohr, and D. Gems, “When and how can death be an adaptation?” *Biochemistry (Moscow)*, vol. 84, no. 12–13, pp. 1433–1437, 2019. DOI: 10.1134/S0006297919120010.
- [37] G. A. Shilovsky, T. S. Putyatina, S. N. Lysenkov, V. V. Ashapkin, O. S. Luchkina, A. V. Markov, and V. P. Skulachev, “Is it possible to prove the existence of an aging program by quantitative analysis of mortality dynamics?” *Biochemistry (Moscow)*, vol. 81, no. 12, pp. 1461–1476, 2016. DOI: 10.1134/S0006297916120075.
- [38] G. A. Shilovsky, T. S. Putyatina, V. V. Ashapkin, O. S. Luchkina, and A. V. Markov, “Coefficient of variation of lifespan across the tree of life: Is it a signature of programmed aging?” *Biochemistry (Moscow)*, vol. 82, no. 12, pp. 1480–1492, 2017. DOI: 10.1134/S0006297917120070.
- [39] T. F. Wrycza, T. I. Missov, and A. Baudisch, “Quantifying the shape of aging,” *PLOS ONE*, vol. 10, no. 3, e0119163, 2015. DOI: 10.1371/journal.pone.0119163.
- [40] M. Boldrini, “Corrado Gini,” *Journal of the Royal Statistical Society. Series A (General)*, vol. 129, no. 1, pp. 148–150, 1966.
- [41] K. Hanada, “A formula of Gini’s concentration ratio and its application to life tables,” *Journal of the Japan Statistical Society, Japanese Issue*, vol. 13, no. 2, pp. 95–98, 1983. DOI: 10.11329/jjss1970.13.95.
- [42] V. Shkolnikov, E. Andreev, and A. Z. Begun, “Gini coefficient as a life table function,” *Demographic Research*, vol. 8, pp. 305–358, 2003. DOI: 10.4054/DemRes.2003.8.11.
- [43] J. Smits and C. Monden, “Length of life inequality around the globe,” *Social Science & Medicine*, vol. 68, no. 6, pp. 1114–1123, 2009. DOI: 10.1016/j.socscimed.2008.12.034.
- [44] N. S. Gavrilova, L. A. Gavrilov, F. F. Severin, and V. P. Skulachev, “Testing predictions of the programmed and stochastic theories of aging: Comparison of variation in age at death, menopause, and sexual maturation,” *Biochemistry (Moscow)*, vol. 77, no. 7, pp. 754–760, 2012. DOI: 10.1134/S0006297912070085.
- [45] A. Comfort, *The biology of senescence*. Elsevier, 1979.

**For citation:**

G. A. Shilovsky, A. V. Seliverstov, O. A. Zverkov, Demographic indicators, models, and testing, *Discrete and Continuous Models and Applied Computational Science* 31 (4) (2023) 359–374. DOI: 10.22363/2658-4670-2023-31-4-359-374.

**Information about the authors:**

**Shilovsky, Gregory A.** — Candidate of Biological Sciences, Senior Researcher in Laboratory 6 at Institute for Information Transmission Problems of the Russian Academy of Sciences (Kharkevich Institute); Researcher in Faculty of Biology at Lomonosov Moscow State University (e-mail: `gregory_sh@list.ru`, phone: +7(495)6502501, ORCID: <https://orcid.org/0000-0001-5017-8331>)

**Seliverstov, Alexandr V.** — Candidate of Physical and Mathematical Sciences, Leading Researcher in Laboratory 6 at Institute for Information Transmission Problems of the Russian Academy of Sciences (Kharkevich Institute) (e-mail: `slvstv@iitp.ru`, phone: +7(495)6502501, ORCID: <https://orcid.org/0000-0003-4746-6396>)

**Zverkov, Oleg A.** — Candidate of Physical and Mathematical Sciences, Researcher in Laboratory 6 at Institute for Information Transmission Problems of the Russian Academy of Sciences (Kharkevich Institute) (e-mail: `zverkov@iitp.ru`, phone: +7(495)6502501, ORCID: <https://orcid.org/0000-0002-8546-364X>)

УДК 314.422:574.34

PACS 07.05.Tr, 89.65.Cd,

DOI: 10.22363/2658-4670-2023-31-4-359-374

EDN: FZWSUR

## Демографические показатели, модели и проверка

Г. А. Шиловский<sup>1,2</sup>, А. В. Селиверстов<sup>1</sup>, О. А. Зверков<sup>1</sup>

<sup>1</sup> *Институт проблем передачи информации имени А. А. Харкевича РАН, Большой каретный пер., д. 19, стр. 1, Москва, 127051, Российская Федерация*

<sup>2</sup> *Московский государственный университет имени М. В. Ломоносова, Ленинские горы, д. 1, стр. 12, Москва, 119991, Российская Федерация*

**Аннотация.** Используя простые демографические показатели для описания динамики смертности, можно скрыть важные особенности кривой выживания, особенно в периоды быстрых изменений, вызванных, например, внутренними или внешними факторами, и особенно в самом старшем или самом молодом возрасте. Поэтому вместо общепринятого метода Гомпертца часто используются другие методы, основанные на демографических показателях. У человека хронический фенотип, в отличие от возрастного-независимого острого фенотипа, проявляется ректангуляризацией кривой выживания с одновременным увеличением средней продолжительности жизни при рождении в результате развития общества и научно-технического прогресса. Несмотря на простую геометрическую интерпретацию явления ректангуляризации кривой выживания, его трудно заметить, прослеживая лишь изменения оптимальных коэффициентов в законе Гомпертца–Мейкхама из-за высокой вычислительной сложности, а также увеличения погрешности расчёта. Этого можно избежать путём расчёта демографических показателей, таких как энтропия Кейфитца, коэффициент Джини и коэффициент вариации продолжительности жизни. Как теоретические примеры, так и расчёты, основанные на реальных демографических данных, показывают, что при одинаковом значении коэффициента Джини в сравниваемых когортах большее значение энтропии Кейфитца указывает на большую долю долгожителей относительно средней продолжительности жизни. Напротив, при том же значении энтропии Кейфитца большее значение коэффициента Джини соответствует относительно большой смертности в молодом возрасте. Мы предполагаем, что уменьшение энтропии Кейфитца может быть связано со снижением фоновой смертности, отражённой в модели Мейкхама, или со снижением смертности в более раннем возрасте, что соответствует изменениям в другом коэффициенте закона Гомпертца. Другой причиной может быть снижение смертности в малых возрастах, что соответствует уменьшению другого коэффициента в законе Гомпертца. Включив динамические возрастные изменения в анализ выживаемости, мы можем углубить наше понимание моделей смертности и механизмов старения, что в конечном итоге внесёт вклад в разработку более надёжных методов оценки эффективности мер против старения и геропротекторов, используемых в геронтологии.

**Ключевые слова:** продолжительность жизни, демографический показатель, энтропия Кейфитца, коэффициент Джини, коэффициент вариации, фенотип, старение, закон Гомпертца