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Trends in Human Species-Specific Lifespan and Actuarial Aging Rate*

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Abstract—The compensation effect of mortality (CEM) was tested and species-specific lifespan was estimated using data on one-year age-specific death rates from the Human Mortality Database (HMD). CEM was confirmed using this source of the data and human species-specific lifespan estimates were obtained, which were similar to the estimates published before. Three models (Gompertz–Makeham, Gompertz–Makeham with mean-centered age, and Gompertz) produced similar estimates of the species-specific lifespan. These estimates demonstrated some increase over time. Attempts to measure aging rates through the Gompertz slope parameter led to the conclusion that actuarial aging rates were stable during most of the 20th century, but recently demonstrated an increase over time in the majority (74%) of studied populations. This recent phenomenon is most likely caused by more rapid historical decline of mortality at the younger adult age groups compared to the older age groups, thus making the age gradient in mortality steeper over time. There is no biomedical reason to believe that human aging rates accelerated recently, so that the actuarial aging rate is probably not a good measure of true aging rate (rate of functional loss). Therefore, better measures of aging rate need to be developed.

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INTRODUCTION

In order to understand fundamental biology of aging and to develop genuine anti-aging interventions it is important to find out first what is the best estimate of the aging rate in humans and aging rate determinants. The aging rate is often estimated as a slope coefficient of the Gompertz law describing exponential increase of mortality rate with age (also known as the Gompertzian slope). This approach is intuitively appealing, because hypothetical non-aging populations have slope coefficient equal to zero, and because the slope parameter characterizes the rate of mortality increasing with age.

Our earlier preliminary studies demonstrated, however, that the slope coefficient is not an ideal measure of the aging rate both for practical and theoretical reasons. For example, using slope coefficient as a measure of the aging rate leads to the counter-intuitive conclusion that women are aging faster than men, despite their lower death rates and higher life expectancy ("male-female aging rate paradox") [1]. Also, mathematical reliability theory of aging indicates that the slope coefficient is determined not only by the rate of functional loss with age ("true aging rate"), but also by the initial redundancy levels (initial reserve capacity) [1, 2]. Aging rate based on the Gompertz slope parameter is often called an actuarial aging rate in order to discriminate it from the true aging rate related to the loss of function [3]. Thus, there is a need for developing more accurate and adequate estimates of the aging rate.

The purpose of this article is to study the most recent changes in actuarial aging rates in human populations. Actuarial aging rates are different across different populations and are organized in such a way that lower initial

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mortality is compensated by its more rapid growth with age. This means that high mortality rates in the disadvantaged populations (within a given species) are compensated for by low actuarial aging rate (longer mortality doubling period). As a result of this compensation, relative differences in the mortality rates tend to decrease with age within a given biological species [1]. In this study we analyze compensation effect of mortality (CEM) in more detail paying particular attention to quantitative measures of this phenomenon such as species-specific lifespan [1].

BACKGROUND INFORMATION

The Strehler–Mildvan correlation. In 1960, American researchers Bernard L. Strehler and Albert S. Mildvan published an article entitled "General Theory of Mortality and Aging" in the leading scientific journal Science [4]. In this article, they describe an inverse relationship between the parameters of the Gompertz law:

$$\mu_x = A + R_0 \exp(\alpha x), \quad (1)$$

where μ_x is a hazard rate at age x and A, R_0, and α are parameters.

Strehler and Mildvan found out by neglecting the Makeham parameter A of the equation (1) that in those countries where the values of the pre-exponential multiplier (designated as R_0) were high, the values of the exponential index (α) were reduced. Subsequently, this observation became known as the Strehler–Mildvan correlation, and it acquired the status of a fundamental law describing the survivorship of organisms.

It can easily be seen that this phenomenon, if it really exists, is of great importance for determination of the species-specific characteristics of lifespan. Indeed, parameters of the Strehler–Mildvan correlation, linking the quantities R_0 and α, will be species-specific invariants as a consequence of the very principle used to calculate them. Gavrilov and Gavrilova questioned the Strehler–Mildvan approach and demonstrated that varying the Makeham parameter (A) from 0 to 0.01 per year is sufficient to produce spurious Strehler–Mildvan correlation [1]. Comparison of this spurious correlation with the correlation published by Strehler and Mildvan (1960) showed very good agreement between these two correlations. And attempts to use the Strehler and Mildvan correlation in constructing mathematical models of aging may lead to absurd results. Indeed, from the data presented in the article by Strehler and Mildvan (1960), it follows that the slope coefficient of the linear regression of $\ln R_0$ with α is only 68.5 years. However, according to the "General theory of mortality and aging" by Strehler and Mildvan (1960), this quantity should correspond to the age at which so-called vitality, "the capacity of an individual organism to stay alive" (p. 15), becomes zero. Inconsistency of these results of the Strehler–Mildvan correlation has been pointed out on several occasions [5, 6].

Compensation effect of mortality. Later, Gavrilov and Gavrilova attempted to improve the approach applied by Strehler and Mildvan and to take into account Makeham parameter (A). In the course of this study, they found the so-called compensation effect of mortality [1]. They found that within a given biological species the values of the age-dependent or senescent component of mortality (Gompertz term) are correlated in such a way that, when extrapolated, they meet at a single point. Compensation effect of mortality (CEM) refers to mortality convergence, when higher values for the slope parameter (in the Gompertz function) are compensated by lower values of the intercept parameter R_0 in different populations of a given biological species [1, 7]. CEM can be quantified using inverse relationship of the Gompertz parameters of the Gompertz–Makeham equation:

$$\ln R_0 = \ln M - B\alpha. \quad (2)$$

The coordinate corresponding to the age at which all the mortality trajectories intersect (B) has been called the species-specific lifespan [1]. It was found that for humans its value is equal to 95 $\pm$ 2 years [1]. It should be noted that the compensation effect of mortality can be observed by a simple visual inspection of mortality trajectories without calculation of Gompertz parameters (see Fig. 1). Also, CEM can be observed not only for humans, but for some other biological species [1, 7].

Summarizing this earlier research on the topic, we need to note that these studies have been made long ago and not many new empirical studies of CEM or Strehler–Mildvan correlation have been conducted since that time. Most studies on the topic are focused on theoretical developments of Strehler–Mildvan theory of aging [8-11]. A few empirical studies on the topic did not take into account the Makeham parameter producing spurious correlation between the Gompertz parameter estimates [3, 12]. This approach has been already criticized by some researchers [13]. Two publications present graphs of the inverse correlation between the Gomperz parameters using the data from the Human Mortality Database [14, 15], but do not make any attempt to provide quantitative measures of CEM.

One of the objectives of this study is to conduct large-scale empirical study of correlation between the Gompertz parameters taking into account the Makeham term (also known as background mortality [1, 16]) and test the compensation effect of mortality using contemporary data. Special emphasis has been made on quantitative measures of CEM and historical evolution of the species-specific lifespan. Possible factors, which may produce a spurious dependence between the Gompertz parameters [7] are also considered. More recent changes in the background mortality (Makeham term) were

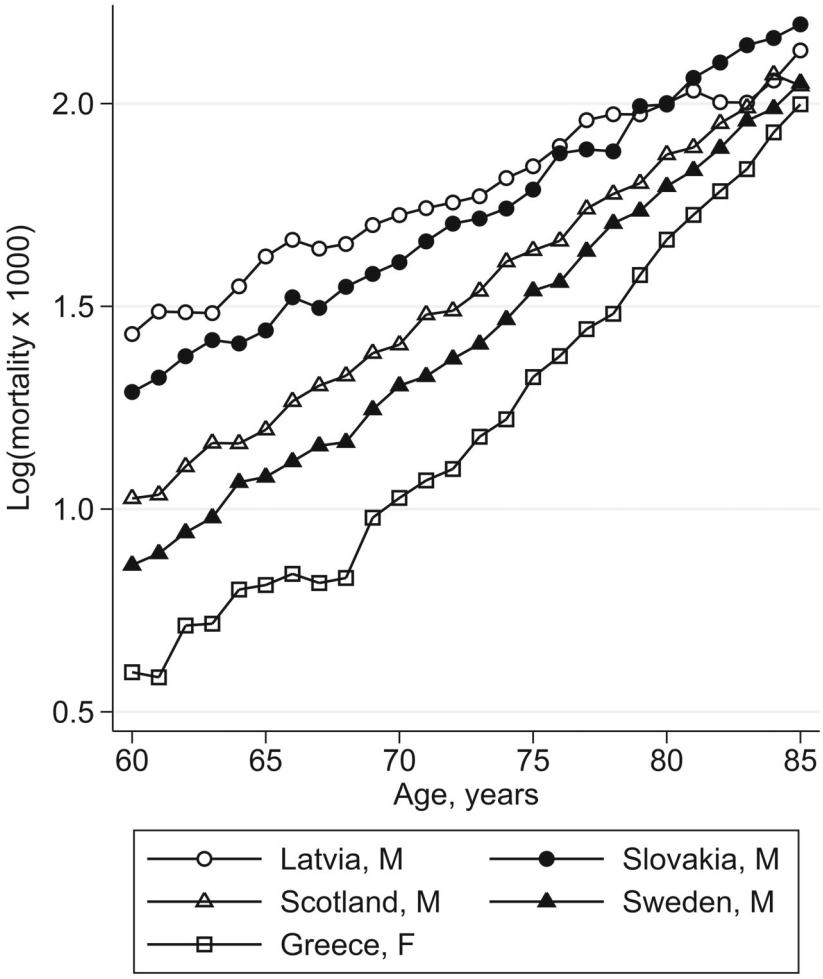

Fig. 1. Convergence of mortality trajectories at advanced ages for five European populations in 2010 illustrating compensation effect of mortality for males (M) and females (F).

analyzed by Bongaarts, who showed that in the contemporary populations background mortality indeed does not depend on age and is close to zero [16]. For this reason, we hypothesize that in the case of contemporary populations accounting for the Makeham term is not as important as it was in the past.

MATERIALS AND METHODS

One of the goals of this study was to test the compensation effect of mortality found in the earlier studies [1, 17] using new contemporary data. To ensure comparability of results, compensation effect of mortality was tested using cross-sectional mortality data as it was done in other studies on the same topic [1, 4, 14, 17]. Cross-sectional data on mortality allow researchers to apply information on longer historical periods and are widely used by demographers in research on parametric models of mortality including the Gompertz model [16, 18-20].

Methods. In the first step of the analysis, we calculated parameters R_0 and α of the Gompertz–Makeham equation (equation 1). Parameters of the Gompertz–Makeham model were estimated using method of nonlinear regression in the age interval 30-80 years (*nlin* procedure in Stata package, version 14) as it was suggested before [18]. Threshold of 30 years was selected in order to avoid hump of external deaths at younger ages. Some researchers use logistic-Makeham model to study historical changes in period mortality in order to capture mortality deceleration after the age 85 years [16, 18]. In our study we analyze mortality below age 85 years, so that applying Gompertz or Gompertz–Makeham models is reasonable.

We have estimated parameters for the following three models:

1. Traditional Gompertz–Makeham model;
2. Gompertz–Makeham model with mean-centered age $(x - 60)$;
3. Gompertz model used by Strehler–Mildvan.

Model no. 2 is based on traditional statistical approaches of using mean-centered variables in order to reduce collinearity [21, 22]. This approach ameliorates collinearity, but does not completely eliminate it [22].

It was shown that the least squares fit often leads to an ill-defined non-linear optimization problem, which is extremely sensitive to sampling errors and smallest systematic demographic variations [23]. The best fit for the Gompertz parameters in this case turn out to be realized by the form of Strehler–Mildvan correlation [23]. The property of regression model is that it always goes through the mean values of x and y. As a result, statistical variation is minimal at the centered values of x. Thus, to lessen the problem of spurious correlation between the Gompertz parameters, we estimate parameters of the Gompertz–Makeham equation using age centered at 60 years (approximately middle of the age interval for parameter estimation).

Finally, model no. 3 is the Gompertz model used by Strehler and Mildvan (1960) [4]. This model is used to demonstrate magnitude of the bias caused by neglecting the Makeham parameter. We hypothesize that parameter estimates for this model would be similar to those for the models with Makeham parameter given almost zero background mortality in the contemporary populations [16, 18].

For each model, we run linear regressions between the Gompertz parameters ($\ln R_0$ and α) of the form presented in equation 2. Thus, the species-specific lifespan (slope parameter, B) and the intercept parameter (lnM) have been estimated.

In the study of recent historical changes of actuarial aging rates parameters of the Gompertz–Makeham model were estimated for each population (life table) using method of non-linear regression in the age interval 60-85 years. Mortality estimates obtained for the single-year age intervals demonstrate significant statistical variation and are more stable at older ages (see Fig. 1). So, the age interval of 60-85 years was selected to produce more stable estimates of the Gompertz–Makeham parameters and avoid mortality fluctuations at younger ages, common in the contemporary low-mortality populations. For the study of historical changes in actuarial aging rates we have estimated parameters for traditional Gompertz–Makeham model.

Data. Human Mortality Database (HMD) was used as a source of mortality data for this study [24]. This database contains mortality data for 45 countries with reasonably good quality of demographic statistics. Totally we used age-specific death rates for 3662 populations available in HMD covering periods from 1900 to 2014. Study of historical changes in the aging rates was focused on the most recent trends after 2000. The age-specific period death rates of males and females are available in the database from ages 0 to 110 and older. Data are available in one-year age and time increments denoted as M_x, where x indicates single year of age. Deaths at age 110 and over are combined together.

Table 1. Characteristics of the compensation law of mortality based on three models

Population	Regression coefficients*		Correlation coefficient between $\ln R_0$ and α	Number of populations
	lnM ± σ	B ± σ, years		
Standard Gompertz–Makeham model				
Men	−1.76 ± 0.03	84.62 ± 0.37	−0.9665	3662
Women	−1.50 ± 0.06	89.01 ± 0.50	−0.9473	3662
Total	−2.04 ± 0.06	83.44 ± 0.56	−0.9264	3662
Gompertz–Makeham model with age centered at 60 years				
Men	−1.76 ± 0.03	24.62 ± 0.37	−0.7383	3662
Women	−1.50 ± 0.06	29.01 ± 0.50	−0.6941	3662
Total	−2.04 ± 0.06	23.44 ± 0.56	−0.5685	3662
Gompertz model				
Men	−1.31 ± 0.04	89.80 ± 0.45	−0.9571	3662
Women	−1.18 ± 0.06	92.60 ± 0.56	−0.9400	3662
Total	−1.49 ± 0.06	89.42 ± 0.63	−0.9209	3662

Note. * Parameters of Gompertz–Makeham model were estimated in age interval 30-80 years.

RESULTS

Compensation effect of mortality. Table 1 shows quantitative characteristics of CEM obtained using data from the Human Mortality Database. Estimates of the species-specific lifespan based on the traditional Gompertz–Makeham model and HMD data (83.4 ± 0.6) are somewhat lower compared to the earlier published estimates (95 ± 2 years) [1]. Estimates of the species-specific lifespan based on the Gompertz model produce higher values: 89.4 ± 0.6 years. These values are closer to the estimates published earlier [1].

The model with mean-centered age variable shows significant decrease in the value of correlation coefficient between the Gompertz parameter estimates (see Table 1). Strength of the relationship between the Gompertz parameters can be expressed by squaring the correlation coefficient and multiplying by 100. After mean-centering of age, percent of variance explained reduced from 86.5% (r = –0.93) to 32.5% (r = –0.57), when statistical correlation between the parameter estimates is accounted for. Thus, 54% of variation can be explained by statistical spurious correlation between the Gompertz parameters. Still correlation between the Gompertz parameter estimates does not entirely disappear after this age mean-centering procedure and the species-specific lifespan estimates remain the same as in the case of the Gompertz–Makeham model (see Table 1).

As we expected, the Gompertz model produces the species-specific lifespan estimates, which are close to estimates obtained using the Gompertz–Makeham model, when contemporary data with low background mortality are used (see Table 1).

Estimates of R_0 and α (particularly α) obtained within one country at different points of time are often very stable in history [1, 18, 25]. As a result, the inverse correlation between $\ln R_0$ and α is strongly affected by the accuracy of the Gompertz parameter estimation for each single year of time. For this reason, we conducted additional data analyses (see Table 2) and estimated parameters of the inverse dependence between the Gompertz parameters for the following single calendar years: 1925, 1955, 1985, and 2010. This way we could analyze changes in the parameter estimates of the inverse dependence

Table 2. Characteristics of compensation effect of mortality based on three models, by time period

Year	Regression coefficients*		Correlation coefficient between $\ln R_0$ and α	Number of populations
	lnM ± σ	B ± σ, years		
Gompertz–Makeham model				
1925	–2.73 ± 0.72	73.08 ± 7.22	–0.9222	20
1955	–2.09 ± 0.25	81.02 ± 2.47	–0.9867	31
1985	–2.30 ± 0.28	81.31 ± 2.82	–0.9751	45
2010	–1.87 ± 0.26	90.40 ± 2.53	–0.9840	44
Gompertz–Makeham model with age centered at 60 years				
1925	–2.73 ± 0.72	13.08 ± 7.22	–0.3927	20
1955	–2.09 ± 0.25	21.02 ± 2.47	–0.8446	31
1985	–2.30 ± 0.28	21.31 ± 2.82	–0.7553	45
2010	–1.87 ± 0.26	30.40 ± 2.53	–0.8802	44
Gompertz model				
1925	–3.46 ± 0.63	64.15 ± 6.97	–0.9082	20
1955	–2.10 ± 0.27	80.89 ± 2.78	–0.9833	31
1985	–2.02 ± 0.34	84.22 ± 3.51	–0.9647	45
2010	–1.84 ± 0.27	91.37 ± 2.73	–0.9818	44

Note. * Model parameters were estimated in age interval 30-80 years.

Table 3. Characteristics of compensation effect of mortality based on standard Gompertz–Makeham model*

Population	Regression coefficients		Correlation coefficient between $\ln R_0$ and α	Number of populations
	$\ln M \pm \sigma$	$B \pm \sigma$, years		
age interval for parameters estimation: 30-80 years				
Men	-1.76 ± 0.03	84.62 ± 0.37	–0.9665	3662
Women	-1.50 ± 0.06	89.01 ± 0.50	–0.9473	3662
age interval for parameters estimation: 60-85 years				
Men	-1.06 ± 0.02	92.13 ± 0.21	–0.9909	3601
Women	-0.62 ± 0.06	97.17 ± 0.17	–0.9944	3601

Note. * Parameters of Gompertz–Makeham model were estimated in age interval 30-80 years (upper panel) and 60-85 years (lower panel).

occurring in time. These analyses show increase of the estimates for the species-specific lifespan over time: from 73 ± 7 years in 1925 to 90 ± 3 years in 2010 (Gompertz–Makeham model). Similar results were obtained for other studied models. The only difference was observed for the Gompertz–Makeham model and the Gompertz model in 1925. The Makeham term in 1925 was relatively high for all countries, so ignoring it in the case of the Gompertz model should result in the spurious Strehler–Mildvan correlation. Indeed, the species-specific lifespan obtained using the Gompertz model (64 ± 7 years) was lower compared to the estimate obtained with the Gompertz–Makehem model (73 ± 7 years). Estimate of the species-specific lifespan for the Gompertz model is close to the estimate obtained by Strehler and Mildvan (68.5 years) [4]. Estimates of the species-specific lifespan obtained for more recent time period (2010) are close to the estimates published earlier [1]. Estimates of the species-specific lifespan may depend not only on historical period, but also on the age interval of the parameter estimation. Mortality rates at younger age are subject to significant fluctuations and distortions of exponential mortality increase. Mortality estimates in the age interval 60-85 years look smoother and follow standard exponential growth with age better than at younger ages when mortality is extremely low. Table 3 presents estimates of the species-specific lifespan when the Gompertz–Makeham parameters are estimated using different age intervals. Note that parameter estimation in the age interval 60-85 years produces higher estimates of the species-specific lifespan.

Overall, we can conclude that estimates of the species-specific lifespan based on the contemporary data demonstrate good agreement with the earlier publication [1]. That means that quantitative measures of CEM for humans are rather stable. In the contemporary populations background mortality is very low [1, 16, 18], so that the Gompertz model without the Makeham parameter can be used to quantify CEM.

Historical changes of actuarial aging rates. We analyzed age-specific death rates for 1900-2014 using cross-sectional data. Working with the data, it became clear that mortality estimates below the age 60 years have very high variation and are not stable. For that reason, we estimated parameters of the Gompertz–Makeham model in the age interval 60-85 years. With these estimates, we recalculated parameters of the compensation effect of mortality (see Table 3). Note that the estimates of the species-specific lifespan using newly calculated parameters of the Gompertz–Makeham model are closer to the value of species-specific lifespan reported by Gavrilov and Gavrilova (1991) in their earlier study (95 ± 3 years) [1]. These results reconfirm again the existence of the compensation effect of mortality.

Availability of the historical data for actuarial aging rate provided us with opportunity to study time trends of actuarial aging rate in different countries. Our earlier studies demonstrated that the actuarial aging rates are relatively stable over time at least until the 1960s [1, 25]. Studies by Bongaarts confirmed this initial finding [16, 18]. With longer time series it became clear that the actuarial aging rates have more complex trajectories after the 1960s. Figures 2 and 3 show time trends of actuarial aging rates in Finland and Norway. These results confirm stability of actuarial aging rates before the 1970s while after that time actuarial aging rates started to grow with more complex trajectories.

Taking into account these complex time trajectories of actuarial aging rates, we analyzed the most recent trends past 2000. For each country we run linear regression of the Gompertz slope parameter on time in order to estimate general trend for actuarial aging rate. Table 4 presents slope coefficients of this linear regression together with corresponding *p*-values. Note that the actuarial

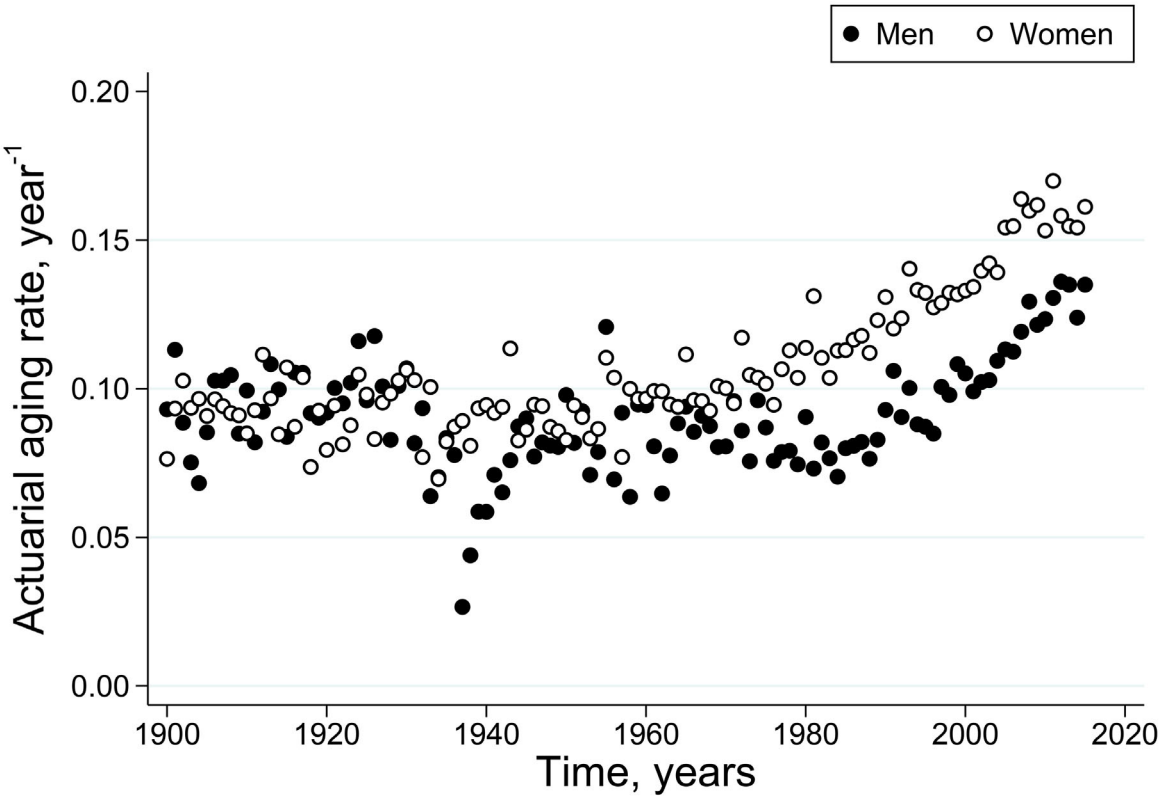

Fig. 2. Time trends of actuarial aging rate (Gompertz slope parameter) in Finland. Actuarial aging rate is estimated in the age interval 60-85 years.

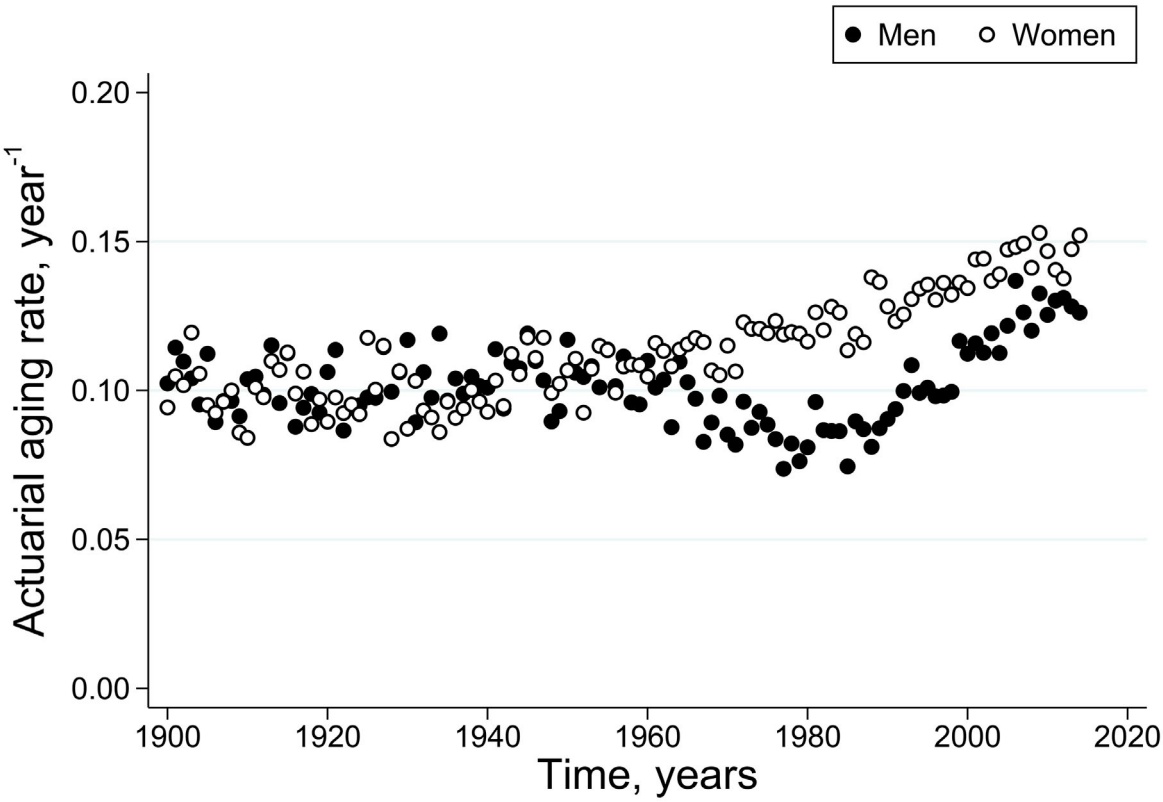

Fig. 3. Time trends of actuarial aging rate (Gompertz slope parameter) in Norway. Actuarial aging rate is estimated in the age interval 60-85 years.

Table 4. Historical changes of actuarial aging rates* after 2000

Country	Men		Women		Number of populations
	Slope coefficient of linear regression, $\times 10^3$	p-value	Slope coefficient of linear regression, $\times 10^3$	p-value	
Australia	1.5114	**0.001**	1.2951	**<0.001**	12
Austria	2.7407	**<0.001**	1.9276	**<0.001**	15
Belgium	1.8230	**<0.001**	1.4875	**0.001**	16
Bulgaria	2.4305	0.054	1.8606	**0.010**	11
Belarus	0.3843	0.386	1.2828	**0.001**	15
Canada	1.0965	**0.026**	–0.0790	0.769	12
Switzerland	2.1124	**<0.001**	0.8233	**0.026**	15
Czech Republic	2.1886	**<0.001**	2.3290	**<0.001**	15
Chile	2.7909	0.220	3.4081	**0.002**	6
East Germany	1.8330	**0.017**	2.4485	**<0.001**	14
Germany	2.1047	**<0.001**	2.4264	**<0.001**	14
West Germany	2.1980	**<0.001**	2.4446	**<0.001**	14
Denmark	2.6976	**<0.001**	1.2245	**<0.001**	15
Spain	1.6905	**<0.001**	1.4617	**<0.001**	15
Estonia	0.8753	0.296	0.5735	0.225	14
Finland	2.4491	**<0.001**	1.7940	**0.001**	16
France, Civilian Population	2.2854	**<0.001**	1.5790	**0.001**	15
France, Total Population	2.2854	**<0.001**	1.5790	**0.001**	15
England and Wales, Civilian National Population	2.0366	**<0.001**	1.8550	**<0.001**	14
England and Wales, Total Population	2.0366	**<0.001**	1.8550	**<0.001**	14
Northern Ireland	1.1501	0.136	2.1498	**0.002**	14
United Kingdom	1.9767	**<0.001**	1.7956	**<0.001**	14
Scotland	1.9311	**<0.001**	1.5401	**0.001**	14
Greece	2.1401	**0.005**	3.2622	**<0.001**	14
Hungary	1.7999	**0.004**	1.9305	**<0.001**	15

Table 4 *(cont.)*

Country	Men		Women		Number of populations
	Slope coefficient of linear regression, $\times 10^3$	*p*-value	Slope coefficient of linear regression, $\times 10^3$	*p*-value	
Ireland	2.4650	**<0.001**	2.4127	**<0.001**	15
Iceland	2.3997	0.130	3.8444	0.075	14
Israel	2.2684	**<0.001**	1.4485	**0.003**	15
Italy	3.2138	**<0.001**	2.2241	**<0.001**	13
Japan	1.5498	**<0.001**	1.0220	**<0.001**	15
Lithuania	2.3446	**0.001**	2.2812	**<0.001**	14
Luxemburg	–0.3850	0.731	1.8877	0.183	15
Latvia	–1.3286	0.194	0.6781	0.242	14
Netherlands	1.7530	**<0.001**	0.9418	**0.006**	13
Norway	1.2504	**0.003**	0.5473	0.108	15
New Zealand, non-maori	2.0541	0.063	–0.0909	0.937	9
New Zealand	1.5989	**0.005**	0.7302	0.185	14
Poland	1.4253	**<0.001**	2.3798	**<0.001**	15
Portugal	2.9999	**<0.001**	3.5276	**<0.001**	13
Russia	–0.6028	**0.019**	1.3931	**<0.001**	15
Slovakia	1.7801	**0.003**	2.5763	**<0.001**	15
Slovenia	2.7497	**0.020**	2.1366	**0.011**	15
Sweden	1.2518	**<0.001**	0.4516	0.146	15
Taiwan	–0.8695	**0.015**	0.3212	0.114	15
Ukraine	–0.8367	**0.003**	0.4007	0.091	14
USA	0.2857	**0.026**	–0.1836	0.214	15

Note. Slope coefficients of linear regression for dependencies of actuarial aging rate on time.

* Gompertz–Makeham parameters were estimated in age interval 60-85 years. Statistically significant dependencies are highlighted in bold.

aging rates increased over time in the majority of countries. Actuarial aging rates increased in 34 cases for men, in 34 cases for women, and in 30 cases it increased for both sexes. In 9 cases for men and 12 cases for women actuarial aging rates showed no statistically significant change. Thus, 74% of the studied populations show increase of actuarial aging rates after 2000.

We may conclude that in the cross-sectional data the actuarial aging rate is increasing over time in the majority of populations during the most recent time period. Possible explanation for the increasing trend of actuarial aging rate in the period data may be unequal rate of historical decline of mortality among older and younger age groups. It was found that mortality of centenari-

ans does not decline over time for at least the last 20-30 years. This phenomenon was first noticed by Drefahl for Swedish centenarians [26]. Later it was reported for the USA [27] and four European countries [28]. At the same time, mortality at younger ages continues to decline [29]. In this case the age-specific mortality trajectories would inevitably become steeper. Before that time, there was a parallel downward shift of mortality (in semi-log coordinates) and actuarial aging rates remained stable [30]. Based on this phenomenon, Bongaarts suggested his method of mortality projection (shifting mortality method) [18].

DISCUSSION AND CONCLUSION

The study of quantitative measures of the compensation effect of mortality using contemporary data confirmed that the inverse correlation between the Gompertz intercept parameter, $\ln(R_0)$, and the Gompertz slope parameter (α) of the Gompertz–Makeham equation does exist and is highly statistically significant when we compare different human populations. The estimates of the species-specific lifespan (parameter B) for the contemporary populations are close to the estimates obtained in the earlier publication [1]. It was also found that the estimates of the species-specific lifespan are lower in the historically earlier periods even after accounting for background mortality. These estimates increase over time.

Use of the mean-centered values of age to account for collinearity results in the decreasing estimates of correlation coefficients between the Gompertz parameters, but does not change estimates of the species-specific lifespan. It was claimed recently that the Strehler–Mildvan correlation may be caused by the statistical artifact due to spurious correlation between the Gompertz parameter estimates [23]. Reducing collinearity with the mean-centered age variable shows that spurious correlation between the Gompertz parameter estimates may be responsible for 54% of the explained variation, but does not entirely eliminate correlation between the Gompertz parameter estimates. Golubev [7] considered two sources of possible artifacts producing spurious Strehler–Mildvan correlation: statistical correlation between the Gompertz parameters [23] and ignoring the Makeham term [1]. He suggested that the generalized Gompertz–Makeham law still has a clear biological interpretation of its parameters despite existing problems (which could be alleviated) [7].

It was also found that the background mortality is very low in the contemporary populations [1, 16] and, hence, has no noticeable effect on the estimates of the species-specific lifespan (see Tables 1 and 2).

In this study we also studied the most recent trends of actuarial aging rates (Gompertz slope parameter). It was found that during the most of the 20th century actuarial aging rates showed remarkable stability when mortality decline was caused predominantly by the decrease of the Makeham term (background mortality) [1, 16, 18]. This observation is confirmed by the data for Finland and Norway presented in Figs. 2 and 3. However, during the last 20-30 years actuarial aging rates started to increase in many countries. We need to mention here a paper by Tai and Noymer who conducted a large-scale study of actuarial aging rates for all populations available in HMD on a historical scale starting from 19th century [14]. The authors measured actuarial aging rates using both Gompertz and Gompertz–Makeham models, although it is obvious that unbiased trends of the actuarial aging rates could be obtained only with the latter model. Still the authors put in their abstract the following misleading conclusion: "Over time in human populations, the Gompertz slope parameter has increased, indicating a more severe increase in mortality rates as age goes up." However, their own results based on the Gompertz–Makeham model clearly show that the aging rates are stable for very long time and then (around the 1990s) experience an increase (Figs. 6 and 7 in [14]). Stability of the actuarial aging rates was more pronounced for men [14].

Tai and Noymer showed their data for a mixture of many populations available in HMD, so it is not possible to conclude whether the recent increase in actuarial aging rates is a general phenomenon or is driven by a small number of populations. In our study we analyzed time trends in actuarial aging rates for each population separately. It turns out that indeed most populations (74%) demonstrate increase of actuarial aging rates after 2000. Still in 23% of the populations actuarial aging rates remain stable.

The observed increase of actuarial aging rates shows that attempts to measure aging rates through the Gompertz slope parameter are problematic. The recent increase of actuarial aging rates is counter-intuitive because there is no biomedical reason to believe that human aging rates accelerated recently. In fact, the opposite hypothesis seems more plausible, given the decrease in age-specific mortality rates and increasing life expectancy.

While studying this paradox of increasing actuarial aging rate we found that it is most likely caused by more rapid historical decline of mortality at the younger age groups compared to the older ones, thus making the age gradient in mortality steeper over time. This phenomenon challenges the existing method of mortality projections based on stability of aging rate over time [16, 18]. This recent increase of actuarial aging rates will make mortality trajectories steeper and thus contribute to the compression of mortality and morbidity when frail organisms would die quickly. Compression of mortality can be compared with the slow phenopto-

sis when the old organism is quickly killed by its own mechanism of acute phenoptosis to eliminate individual that cannot be involved in natural selection [31]. Recent growth of actuarial aging rates demonstrates that actuarial aging rate is not a good measure of biological aging rate, so that better measures of aging rate need to be developed.

Contributions. L.G. designed the study, analyzed and interpreted results, edited the manuscript. N.G. conducted statistical analyses and prepared the manuscript.

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